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THE EFFECT OF DYNAMIC EXERCISE ON THE BLOOD PRESSURE RESPONSE TO ISOMETRIC EXERCISE IN NORMOTENSIVE MALES

by

Jacqueline L. Vanderluit

In partial fulfillment of
Master of Science in Kinanthropology.

Presented to Graduate Studies.
University of Ottawa

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to Mom and Dad

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ABSTRACT

The purpose of this study was to determine whether the pressor response to a static handgrip exercise would be blunted if performed during or immediately following dynamic exercise. Twenty male subjects (mean age 27 yrs) performed one minute handgrip (HG) contractions under three conditions 1) standing at rest, 2) during the 4th to 5th min of treadmill walking exercise and 3) during recovery one min following the combined exercise. The handgrip exercises were performed at 30% and 40% of maximum voluntary contraction (MVC). MVC was defined as the highest of three maximum contractions. Walking speed was set to elicit individual heart rate's of 100±10 beats per minute. Systolic (SBP), diastolic (DBP) and mean arterial pressure (MAP), as well as heart rate (HR), were recorded every two seconds with a Finapres BP monitor. The changes (Δ) in BP and HR responses from (1) rest to HG exercise (restHG), (2) the 3-4 min walking at 100 bpm to walking+handgripping (exHG) during the 4-5 min of exercise and (3) 0-1 min of recovery to 1-2 min post exercise while handgripping (postexHG) were statistically analyzed by a 2X3 ANOVA with repeated measures design. The results indicated that there was a main effect of intensity, such that HG exercises performed at 40% MVC produced a significantly greater blood pressure response (SBP, DBP and MAP) than HG at 30% MVC for all three conditions. The ΔSBP response to exHG and postexHG at 30% MVC were significantly lower than the response at restHG, while at 40% MVC, postexHG was significantly lower from restHG, but not exHG. The ΔDBPs at postexHG were significantly higher than the response observed at restHG and exHG for both intensities. There were no significant differences in ΔMAP across the three HG conditions at 30% MVC; however, ΔMAP for exHG at 30% MVC was significantly lower than the three conditions at 40% MVC, and ΔMAP for postexHG at 40% MVC was significantly greater than all the other conditions. Although there was no significant difference between 30% and 40% MVC for ΔHR, significant differences were noted across the three HG conditions within each level of intensity. A comparison of regression coefficients (slopes) of SBP and DBP (measured during the one min HG) against time, indicated that for the postexHG, the increase in SBP and DBP with time during one min HG was significantly greater than for the restHG or exHG conditions with the exception of SBP at 40% MVC where no significant differences were noted. It is concluded that the SBP response to static exercise is blunted when a

HG at 30% MVC is performed during moderate dynamic exercise but not with HG contractions at 40% MVC. In addition, the SBP response to static exercise is also blunted when HG contractions at both 30% and 40% MVC are performed following dynamic exercise. Also, dynamic exercise augments the DBP response to static contractions (30% & 40% MVC) performed following dynamic effort. Responses measured post dynamic exercise are confounded by time, since the static HG of recovery was compared to measurements taken one minute earlier. The blood pressure and heart rate response to static exercise may be masked by the falling values of recovery.

INTRODUCTION

The hemodynamic response to exercise depends on whether it is performed in the static or dynamic exercise mode. Static exercise is the maintenance of a muscle contraction where no change in muscle length occurs. Conversely, dynamic exercise involves changes in muscle length as a consequence of the contraction and relaxation phases (Temkin, 1983). During static exercise an increase in intramuscular pressure and mechanical pressure compresses the blood vessels, thereby hindering blood flow to the active muscle (Humphreys and Lind, 1963; Kilbom and Persson, 1981; Lewis et al., 1985). Thus, the cardiovascular response is directed towards "maintaining perfusion pressure" to the contracted muscle to ensure an adequate blood supply (Humphreys and Lind, 1963). This is accomplished by vasodilation of the blood vessels within the active muscle and strong vasoconstriction of blood vessels throughout the rest of the body (Lind and McNicol, 1967). The strong vasoconstrictory response, along with the mechanical compression of the blood vessels, results in a marked rise in both systolic and diastolic blood pressures while the overall systemic vascular resistance is maintained (Lind and McNicol, 1967; Lewis et al., 1985). The large increase in both the systolic and diastolic blood pressure is termed the pressor response (Humphreys and Lind, 1963).

In contrast to static exercise, dynamic exercise consists of rhythmic contraction and relaxation phases of the muscle which enhances blood perfusion of the active muscle as well as increasing the venous return to the heart. Thus, the cardiovascular response is directed primarily towards "meeting the oxygen demands" of the exercising muscle. This is accomplished by increasing blood flow through vasodilation of the blood vessels to the active muscle and vasoconstriction of vessels in nonactive tissues (Lind and McNicol, 1967; Colombo et al., 1989). The vasodilation of the arterioles reduces the total peripheral resistance which along with an increase in venous return and cardiac output results in an increase in the circulating blood volume. Therefore, the rise in systolic blood pressure attributed to the increase in cardiac output is limited by a fall in systemic vascular resistance, while the diastolic blood pressure increases slightly or is maintained (Perez-Gonzalez, 1981; Lewis et al., 1985).

The blood pressure responses to isometric and dynamic exercise therefore, are distinctly different. In dynamic exercise, total peripheral resistance decreases and there is a much greater circulating blood volume resulting in an increase in SBP and little or no change in DBP whereas with static exercise, the circulating blood volume remains the same but there is a much greater increase in both systolic and diastolic blood pressure due to more extensive vasoconstriction.

The heart rate responses to static and dynamic exercise, however, are quite similar (Lewis et al., 1985). The initiation of either exercise results in a rapid rise in heart rate followed by a more gradual increase during the activity and a decrease with exercise cessation (Asmussen, 1981). The immediate heart rate response with the onset of either static and dynamic exercise has been attributed to parasympathetic (vagal) withdrawal, while the later rise has been associated with sympathetic stimulation (Martin et al., 1985; Clements et al., 1984). This similarity in heart rate response patterns for static and dynamic exercise indicates that heart rate is controlled by the same mechanisms for both modes of exercise: the parasympathetic and sympathetic systems.

Few studies, have examined the effect of combined isometric and dynamic exercise on the cardiovascular response. An early study by Lind and McNicol (1967) examined the blood pressure response to a handgrip contraction superimposed on dynamic walking exercise on four healthy male subjects. Handgrip contractions at 30% and 50% maximum voluntary contraction (MVC) were combined individually with three different walking intensities of 2.5, 3 and 3.5 miles/hour. The results indicated that the increment in systolic blood pressure with static HG exercise was the same regardless of whether the handgrip contraction was performed alone or when combined with the different walking intensities. However, the increase was greater at HGs of 50% than 30% MVC. Thus, they indicated that the pressor response occurred despite the decrease in peripheral resistance associated with dynamic exercise. The maintenance of the pressor response to static effort during dynamic exercise was attributed to a more intense vasoconstrictor response at the higher intensities of dynamic exercise (Lind and McNicol, 1967). A more recent study by Jetté, Kottmann, Landry and Blümchen (1990) superimposed a static handgrip contraction of 33% MVC on three different workloads (40, 80 and 120 watts) of supine cycle ergometry in coronary heart disease patients. Their results supported the findings of Lind and

McNicol (1967), in showing that a similar increase in SBP occurred at rest and at each of the three levels of dynamic exercise. Jetté et al. (1990) therefore, suggested that the increase in blood pressure with static exercise during dynamic effort was due to greater sympathetic stimulation causing an increased vasoconstriction in the periphery. However, both studies provide limited evidence that the pressor response is maintained during dynamic exercise since blood pressure was measured with a standard sphygmomanometer and stethoscope restricting the number of measurements that can be taken per minute. In addition, the study by Lind and McNicol (1967) only tested four healthy male subjects while Jetté et al.(1990) tested coronary heart disease patients in the supine position. The purpose of this study, therefore, was to determine whether the pressor response associated with static exercise is blunted when performed during and following dynamic exercise in normotensive healthy male subjects. This study was unique from the previous studies by Lind and McNicol (1967) and Jetté et al.(1990) in that all subjects were screened prior to testing to ensure a normtensive blood pressure response at rest and to a moderate level of exercise and in addition, all blood pressure and heart rate measurements were recorded at two second intervals using the Finapres BP monitor.

METHODOLOGY

Subjects

Twenty moderately active, male volunteers between 24 to 32 yrs old (mean age 27 ± 2 yrs) participated in this study. A modified PAR-Q was utilized to determine the health status of the participants (Jetté et al., 1991b). All subjects selected for this study met the following criteria: answered "no" to all questions on the modified PAR-Q, were non-smokers, had a resting blood pressure less than 140/90 mmHg, were not taking prescribed medication which could alter the cardiovascular response or physical performance, and had a normotensive blood pressure response to a moderate level (stage A of CAFT) of exercise (Jetté, Landry and Sidney, 1991). Informed written consent was obtained from each subject prior to participation in the study. Ethics approval was granted by the University of Ottawa's Ethics Committee. The physical characteristics of the subjects are listed in Table 1. The mean height of the subjects was 178±6.9 cm, mean weight 77±11.4 kg, mean BMI 24±3.2 kg/m^2 and predicted maxVO_2 51±5.8 ml/kg/min.

Materials

Resting blood pressure and heart rate were measured using a standard sphygmomanometer and stethoscope. Each subject was fitted with a Finapres BP monitor 2300 (Ohmeda, Louisville, Colorado) finger cuff to measure continuous exercising blood pressure and heart rate. The modified Canadian Aerobic Fitness Test (CAFT) (Jetté, Landry and Sidney, 1991) was administered to the subjects to determine their exercise normotensiveness. Predicted maximum aerobic capacity was calculated from the results of the last stage performed of the CAFT using the procedure of Jetté, Campbell, Mongeon and Routhier (1976). Dynamic walking exercise was performed on the Quinton Treadmill Model 643 (Quinton Instruments, Seattle, Washington). The speed of the treadmill was set to elicit an exercising heart rate of 100±10 beats per minute (100 bpm) for each subject. All handgrip contractions were performed on a handgrip dynamometer utilizing an air pressure gauge (H.O. Trerice Co., Detroit, Michigan) connected by surgical tubing to a rubber squeeze bulb. Handgrip strength was measured according to air pressure in kiloPascals.

Procedure

The testing protocol was conducted on two separate days. On day one, the volunteers were briefed as to the nature of the testing and their involvement in the study. The subjects completed the modified PAR-Q (Jetté, Landry and Sidney, 1991), an informed written consent and a health questionnaire (Jetté, Quenneville and Sidney, 1991). They were then seated for a five minute period. The resting heart rate and blood pressure measurements were recorded using a standard stethoscope and sphygmomanometer at the end of the rest period, to ensure that the subjects were normotensive.

The subjects then performed the modified Canadian Aerobic Fitness Test (CAFT)(Jetté, Landry and Sidney, 1991). Subjects between the ages of 24 and 29 yrs started at stage 5 while those between 30 and 32 yrs started at stage 4. The CAFT protocol was followed with the inclusion of a one minute rest between stages during which heart rate was measured in the first 15 seconds followed by blood pressure between the 15th and 45th second. The blood pressure response to the first stage was utilized to screen subjects for an exaggerated blood pressure response (SBP measurement >150 mmHg at stage A) to dynamic exercise (Jetté, Landry and Sidney, 1991). If the heart rate did not exceed a predetermined limit following the first two stages and no detrimental symptoms were observed, the participant was permitted to continue to the second and then to a third stage of the CAFT for the prediction of max$V0_2$. The procedure of Jetté et al. (1976) was used to calculate the predicted max$V0_2$ for each subject.

Each subject's age, height, and weight were measured from which the body mass index (BMI) was later calculated. These anthropometric measurements along with the predicted max$V0_2$ were utilized to determine whether subjects represented a normal sample of the population.

Proper handgrip technique using the dynamometer was demonstrated to the subjects so as to prevent them from performing the Valsalva manoeuvre. All handgrip contractions were performed in the upright, standing position. Subjects were given several trials to familiarize themselves with the dynamometer. Three maximum voluntary handgrip contractions (MVC) of the left hand were performed with a two-minute recovery between each trial. The maximum of the

three consecutive contractions was used to determine the MVC. All handgrip contractions were performed with the left hand while the right hand was used for blood pressure measurements.

The Finapres BP monitor finger cuff was fitted on 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. Subjects then practised maintaining handgrip contractions at 30% and 40% MVC for one minute durations while heart rate and blood pressures were recorded continuously using the Finapres BP monitor (Hartmann and Bassenge, 1989). This was done to allow subjects to become familiar with the technique of maintaining the handgrip contraction and to become accustomed to the Finapres BP monitor.

Next, subjects were given a demonstration as to the proper walking technique on the Quinton Treadmill. Subjects walked at 0% slope with progressive increases in treadmill speed until their exercising heart rate stabilized at 100 bpm, at which time the speed of the treadmill was recorded. Subjects then, practised walking while squeezing the dynamometer at 30% and 40% MVC.

On day two, the one minute resting heart rate and blood pressure measurements were recorded in the standing position with the Finapres BP monitor after a five minute rest period. These standing heart rate and blood pressure measurements were utilized for comparison with the responses to the resting 30% and 40% MVC handgrip tests (Prewalk HG) which were performed also in the upright position.

The subjects then performed a handgrip contraction at 30% MVC for one minute while in the standing position (Prewalk HG). After a five minute rest, the handgrip contraction was repeated at 40% MVC for one minute. Heart rate and blood pressures were recorded every two seconds from 5 seconds before each one minute handgrip contraction and for 15 seconds into the recovery. There was a 5 min rest following the second handgrip contraction.

Next, subjects performed dynamic exercise on the treadmill at the predetermined

speed (corresponding to 100 bpm). At the start of the fourth and last minute of walking exercise, a handgrip contraction (Walk+HG) of 30% MVC was performed and maintained for a one minute duration. Subjects stopped both exercises at the end of this minute and stood immobile on the treadmill. One minute after the cessation of exercise, the subjects again performed the static handgrip contraction (Postwalk HG) at 30% MVC for an additional minute. Heart rate and blood pressures were recorded continuously at two second intervals from the start of the third minute of walking exercise until 15 seconds into the recovery of the Postwalk HG contraction. After a ten minute rest, subjects repeated the walking exercise using handgrip contractions of 40% MVC.

To control for an order effect, subjects were randomly divided into two equal groups such that group A performed all static exercises initially utilizing a handgrip contraction of 30% MVC followed by 40% MVC and group B performed handgrip contractions initially at 40% MVC followed by 30% MVC.

Definitions

Prewalk HG: the one minute handgrip contractions performed at rest.

Walk+HG: the one minute handgrip contractions performed during dynamic walking exercise.

Postwalk HG: the one minute handgrip contractions performed following dynamic exercise.

RestHG: the change in bp and HR from the one min rest to the one min Prewalk HG.

ExHG: the change in bp and HR from the 3-4 min walking exercise immediately preceding HG contraction to the 4-5 min Walk+HG.

PostexHG: the change in bp and HR from the 0-1 min recovery to the 1-2 min Postwalk HG.

Data Analysis

All data was entered and analyzed on a Macintosh SE/30 computer using the Excel 2.2a Spreadsheet, StatsView SE + Graphics and SuperANOVA packages. A descriptive analysis of the group included the means and standard deviations for age, height, weight, BMI, resting heart rate, resting blood pressure, predicted maxVO_2 and maximum HG strength.

The cardiovascular response to handgrip contraction was compared according to the three measurements taken: alone (at rest) vs. during the fourth minute of dynamic exercise vs. one minute following dynamic exercise. A 3x2 factorial design analysis of variance (ANOVA) with repeated measures on two factors was used to determine significant differences between the means. The dependent variables (DV) included heart rate, systolic and diastolic blood pressure and mean arterial pressure. The two seconds recordings of heart rate, systolic, diastolic and mean arterial blood pressure were averaged across the minute for each rest and exercise intervals. The independent variables (IV) included two levels of intensity (30% and 40% MVC) and the three time conditions involving the change in bp and HR from rest to HG exercise [1. change from standing rest to the Prewalk HG contraction (restHG), 2.the change from walking (3-4 min) to the combined Walk+HG contraction (4-5 min) (exHG) and 3. the change from recovery (0-1 min) to the Postwalk HG contraction (1-2 min) (postexHG)]. Differences were assessed at an α level of < 0.05 for significance. When a significant difference was found, a Least Means square analysis was utilized to determine between which means the difference existed.

The SBP and DBP measurements of the 20 subjects were averaged across each two second interval for each of the one minute HG contractions (prewalk HG, walk+HG and postwalk HG) at both 30% and 40% intensities. Lines of best fit were calculated for SBP and DBP measurements versus time. A comparison of the regression coefficients (slopes) for the three HGs within each level of intensity were analyzed for SBP and DBP (Edwards, 1984). Differences were assessed at an α level of < 0.05 for significance.

RESULTS

Figures 1 to 4 depict the subjects' average blood pressure and heart rate responses at two second intervals from 5 seconds pre HG contraction to 15 seconds post HG for the three HGs 1) standing rest (prewalk HG), 2) during dynamic exercise (walk+HG) and 3) during recovery (postwalk HG). The mean SBP responses for the three HGs at 30% MVC and 40% MVC are displayed in Figure 1. At both intensities, the walk+HG and postwalk HG have higher SBP values than the prewalk HG. The mean SBP response to walk+HG at 30% MVC shows more variation than the SBP response to the other HGs including HGs at 40% MVC. Figure 2 depicts the DBP responses to the HGs at 30% and 40% MVC. In contrast to the SBP, the DBP response to static exercise during recovery (postwalk HG) is greater than the DBP response to HG at prewalk HG and walk+HG, this response is evident at both 30% and 40% MVC. The three HG conditions at both the 30% and 40% MVC intensities show a similar linear rise in MAP. Although linear, the MAP values across the three HG conditions are distinct from prewalk HG with the lowest values, to walk+HG and to the highest values with postwalk HG (Figure 3). Within both 30% and 40% MVC, the heart rate response to walk+HG was much greater than observed at prewalk HG and postwalk HG (Figure 4).

The mean of the one minute heart rate, systolic, diastolic and mean arterial blood pressure response at rest, exercise and during recovery with and without HG exercises at 30% and 40% MVC are shown in Table 2. The changes (Δ) in blood pressures and heart rate (ΔSBP, ΔDBP, ΔMAP and ΔHR) from rest, walking exercise and recovery to HG exercise were statistically analyzed across the three HGs and between the 30% and 40% MVC intensities (Table 3). A significant difference was found between 30% and 40% MVC intensities of HG exercise for ΔSBP, ΔDBP and ΔMAP. At 30% MVC, ΔSBP at restHG (16.2$\pm$11.2 mmHg) was significantly greater than ΔSBP at exHG (9.1$\pm$10.5 mmHg) and postexHG (5.9$\pm$9.1 mmHg). However, at 40% MVC, ΔSBP at restHG (17.8$\pm$7.5 mmHg) was significantly greater than only postexHG (13.9$\pm$8.9 mmHg). The increase in ΔSBP with HG exercise was greater for exHG and postexHG at 40% MVC than at 30% MVC. For both 30% MVC and 40% MVC the increases in ΔDBP at postexHG was significantly greater than the increase seen at restHG and exHG. No significant

differences were found for ΔMAP among the three HG conditions at 30% MVC; however, the exHG at 30% MVC was found to be significantly lower than all three HG conditions at 40% MVC. In addition, the postexHG ΔMAP at 40% MVC was significantly greater than the ΔMAP for all the other HG conditions including 30% MVC. In contrast to the blood pressure responses, ΔHR for each of the HG conditions (restHG, exHG and postexHG) were significantly different from each other within both 30% and 40% MVC intensities. The ΔHR at exHG displayed the largest increase, while the postexHG showed a decrease in ΔHR at both 30% and 40% MVC. Apparently, the increase in HR during static exercise measured 1-2 min post dynamic exercise was not sufficient to overcome the larger decrease in HR; the recovery HR (no contraction) was measured 0-1 min post dynamic exercise.

A line of 'best fit' was calculated separately for the mean systolic and diastolic blood pressures recorded at 2 second intervals for the one minute of HG exercise (prewalk HG, walk+HG and postwalk HG) for both 30% and 40% MVC conditions as depicted in Figures 5 and 6. A comparison of regression coefficients for the slopes of the lines was performed within each level of intensity (Table 4). A strong linear trend was observed in the SBP (r^2=0.87 to 0.97) and DBP (r^2=0.92 to 0.98) responses for all three HG conditions at both intensities, with the exception of the SBP response to walk+HG at 30% MVC which showed a poor linear trend (r^2=0.34). At 30% MVC, the average slope of the SBP response to postwalk HG was significantly greater than the average slopes of the prewalk HG and walk+HG; however, no significant differences were found among the three HG contractions at the 40% MVC intensity. Similarly, the average slope of the DBP response for the postwalk HG was significantly greater than the average slopes of the prewalk HG and walk+HG at both 30% and 40% MVC.

DISCUSSION

The pressor response associated with a static contraction is in proportion to the amount of pressure required by the circulatory system to overcome the resistance to blood flow in the contracted muscle (Lind & McNicol, 1967). The main purpose of this study was to examine whether the pressor response to a handgrip contraction would be blunted when performed during dynamic exercise. Studies on normal subjects by Lind and McNicol (1967) and with cardiac patients by Jetté et al. (1990a), separately found that the SBP response to HG exercises at rest was similar to the SBP response when HG exercise was combined with dynamic exercise. Our results, however, differ from these earlier findings. The ΔSBP to the HG contractions indicate that the mean increase seen at restHG was significantly greater than the increases at both exHG and postexHG at the 30% MVC intensity, but significantly greater than only the postexHG at 40% MVC. Thus, it appears that the SBP response to HG contractions at both 30% and 40% MVC may be blunted when performed immediately following walking exercise. In addition, the SBP response to a HG contraction performed during walking exercise also appeared to be blunted at 30% MVC but not at 40% MVC. Immediately following dynamic exercise, reactive hyperemia occurs within the active muscle as it resumes its relaxed state and the blood vessels vasodilate thereby lowering the peripheral resistance (McArdle et al., 1986). Therefore it appears that the reduced peripheral resistance associated with dynamic exercise may blunt the SBP response to an isometric contraction performed immediately following dynamic exercise. However, the SBP response to postexHG also had the greatest rate of increase (slope) but was only significantly different from restHG and exHG at 30% MVC. The SBP slopes appeared greater with HG contractions at 40% than at 30% MVC. In addition, greater vasoconstriction drive as a consequence of the short recovery (only 1 min) between the exHG and postexHG may account for greater rate of increase with the postexHG.

As stated earlier, the pressor response to an isometric contraction has been shown to be proportional to the intensity of the contraction (Lind & McNicol, 1967; Riendl et al., 1977). Our results, however show a greater ΔSBP at 40% MVC than at 30% MVC but not ΔDBP. Saltin et al. (1981) have shown that with isometric contractions of greater than 15% MVC, there is a

progressive occlusion of blood flow to the active muscles. In addition, Asmussen (1981) indicated that contractions less than 10-25% MVC could be maintained for long durations. However, at greater intensities of static contraction the onset of fatigue occurs sooner as blood flow occlusion is increased. Thus, in our study, the greater blood flow occlusion during the 40% MVC exHG contraction likely resulted in a more intense vasoconstriction and pressor response and therefore may have been more resistant to the blunting effect of dynamic exercise. In addition, the ΔSBP response to 30% MVC exHG in Figure 1 displays a jagged line with a low linear trend (r^2=0.34) (Table 4) which may indicate that the ΔSBP may be blunted to a varible degree across the minute interval. Thus the 30% MVC may represent the upper limit of intensity at which dynamic exercise can blunt the pressor response to an isometric contraction. Further studies utilizing HG contractions greater and less than 30% MVC would be needed to demonstrate this phenomenon.

Peripheral resistance during static exercise does not change whereas with dynamic exercise, a decrease in peripheral resistance is reflected by a maintenance or decrease in diastolic blood pressure (Temkin, 1983). Our results indicate no significant difference in ΔDBP between the restHG and exHG at both 30% and 40% MVC. However the postexHG, had a significantly larger increase in ΔDBP in comparison with the restHG and exHG. This large increase in ΔDBP during the recovery HG may be attributed to a number of factors such as: a compensatory adjustment for the blunted SBP response; a result of hyperemia associated with dynamic exercise; or to an inadequate recovery time between the combined exHG and postexHG. The DBP may be greater at postexHG to maintain the perfusion pressure and blood flow to the active muscles in the presence of a decreased SBP. In addition, the majority of individuals subjectively indicated that the postwalk HG was the most difficult to maintain. The short one-minute interval between the combined handgrip and postwalk handgrip may not allow for full recovery and removal of waste products from the active muscles. An inadequate recovery would likely alter the normal muscle fibre recruitment pattern to the HG contraction. More fast twitch fibres would probably be recruited during the recovery HG as a result of fatigued muscle fibres. Recruiting a greater proportion of fast twitch muscle fibres has been shown to produce a greater blood pressure response (Petrofsky et al., 1981). A difference in recruitment pattern with fatigue may also explain why subjects felt that the postwalk HG was the most difficult to maintain since fast twitch muscle

fibres fatigue rapidly, thus requiring a greater central drive in order to maintain the HG contraction at a constant intensity. The combination of inadequate recovery time and a greater number of fast twitch muscle fibres recruited, results in an increase in anaerobiosis which can amplify the sympathetic vasoconstrictory response by releasing a greater amount of waste products into the bloodstream (Asmussen, 1981).

The changes in mean arterial blood pressure (ΔMAP) showed few significant differences among the three HG contractions, with the exception of the response to postexHG at 40% MVC which was significantly larger than all other conditions. During exHG, it appears that the increase in SBP is blunted and DBP increased in such a way as to compensate one another resulting in a similar MAP response as at restHG. The significantly higher MAP at the 40% MVC postexHG may be attributed to the greater intensity at which the exercise is performed in addition to a greater rise in vascular resistance demonstrated in the diastolic blood pressure response.

Contrary to the findings of studies by Lind and McNicol (1967) and Kilbom and Persson (1981) the change in heart rate response (ΔHR) to the three HG exercises were significantly different from each other within 30% and 40% MVC intensities but not between intensities. The largest increase was seen with exHG, while at postexHG the heart rate decreased. The magnitude of the heart rate response is proportional to the degree of sympathetic stimulation (Asmussen, 1981). Thus, the enhanced heart rate response to combined walk and handgrip exercise may be attributed to a greater sympathetic stimulation as observed in the blood pressure responses. Since postwalk HG one minute following the walk+HG, the heart rate was elevated prior to the postwalk HG contraction. As the recovery value is an average of the measurements across the entire minute between the two HG contractions, the HR recovery value (92.4±9.2 bpm) is greater than the postwalk HG (86.8±10.9 bpm).

In conclusion, the ΔSBP to an isometric contraction appears to be blunted when performed immediately following dynamic exercise. However, when the isometric contraction is combined with dynamic exercise, the ΔSBP is only blunted if the contraction is at the lower intensity (30% MVC). In contrast to the ΔSBP response, dynamic exercise immediately preceding a HG

contraction does not blunt the ΔDBP response. The larger muscle mass activated in dynamic exercise causes widespread vasodilation within the muscle mass which has been shown to maintain the DBP (Tuttle and Horvath, 1957). However, when the HG contractions were performed one minute following dynamic exercise, the ΔDBP was significantly greater than at rest or when combined with dynamic exercise. This large increase in DBP may be required to maintain the perfusion pressure to the active muscle as a result of hyperemia as well as a change in the muscle fiber recruitment pattern. The MAP is not significantly affected by whether a HG contraction is performed at rest, during dynamic exercise or immediately following dynamic exercise as a result of a compensation between the SBP and DBP responses. Similarly the HR response to an isometric contraction performed during dynamic exercise is not blunted but rather appears to be indicative of a greater sympathetic stimulation to combined isometric and dynamic exercise.

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TABLE 1: Physical Characteristics of Subjects (n=20)

Variable	Units	Mean	Range	Standard Deviation
Age	yrs	27	24-32	+/- 2.4
Height	cm	178	166-194	+/- 6.9
Weight	kg	77	60-104	+/- 11.4
BMI	kg/m^2	24	21-35	+/- 3.2
Rest HR	bpm	67	54-92	+/- 10.4
Rest Sbp	mmHg	117	105-128	+/- 6.6
Rest Dbp	mmHg	73	58-88	+/- 9.2
Predicted MaxVO2	ml/kg/min	51	41-60	+/- 5.8
Maximum HG	kPa	96	68-111	+/- 12.8

TABLE 2: Group means For SBP, DBP, MAP and HR measured at rest, during dynamic exercise and recovery and with and without Handgrip Exercise at 30% and 40% MVC

Variables		SBP	DBP	MAP	HR
Rest	x	116.6	76.2	87.1	80.4
	SD	11.8	10.6	11.5	8.7
30%					
Prewalk HG	x	132.8	86.7	98.7	81.9
	SD	9.5	10.2	10.1	10.2
Walk	x	140.6	76.2	94.4	99.6
(3-4 min)	SD	14.5	11.3	11.3	9.1
Walk+HG	x	149.7	84.1	103.4	106.2
(4-5 min)	SD	14.8	11.3	11.2	10.2
Recovery	x	138.7	82.1	97.2	92.4
(0-1 min)	SD	16.0	11.1	11.9	9.2
Postwalk HG	x	144.5	96.4	108.7	86.8
(1-2 min)	SD	16.0	12.6	13.0	10.9
40%					
Prewalk HG	x	134.4	89.5	99.9	84.0
	SD	9.7	11.1	12.4	9.7
Walk	x	138.0	74.2	93.0	102.8
(3-4 min)	SD	16.0	10.1	11.3	13.9
Walk+HG	x	153.4	86.0	105.3	107.7
(4-5 min)	SD	15.6	11.6	12.3	9.9
Recovery	x	139.2	81.4	96.5	92.9
(0-1 min)	SD	13.7	8.8	10.5	11.2
Postwalk HG	x	153.1	99.0	115.9	90.9
(1-2 min)	SD	16.8	12.5	14.1	12.7

TABLE 3: The Change in Hemodynamic Response From Rest, Walking and Recovery to Handgrip Exercise

Variables		ΔSBP	ΔDBP	ΔMAP	ΔHR
30%					
1. RestHG	x	16.2 (2,3)	10.5 (3)	11.6 (6)	1.5 (2,3)
	SD	11.7	5.4	7.3	4.8
2. ExHG	x	9.1 (1,4,5,6)	7.9 (3)	9.0 (4,5,6)	6.5 (1,3)
	SD	10.5	8.4	9.1	7.9
3. PostexHG	x	5.9 (1,4,5,6)	14.1 (1,2)	11.5 (6)	-5.6 (1,2)
	SD	9.1	7.4	7.0	6.3
40%					
4. RestHG	x	17.8 (2,3,6)	13.3 (6)	12.8 (2,6)	3.6 (5,6)
	SD	7.5	6.1	9.7	5.3
5. ExHG	x	15.4 (2,3)	11.8 (6)	12.4 (2,6)	4.9 (4,6)
	SD	8.7	6.2	7.0	9.3
6. PostexHG	x	13.9 (2,3,4)	17.7 (4,5)	19.4 (1,2,3,4,5)	-2.0 (4,5)
	SD	8.9	5.9	6.5	9.1

Note: SBP, DBP, MAP and HR had a main effect of HG exercise.
SBP, DBP and MAP had a main effect of intensity.
SBP and MAP had a significant interaction effect of HG exercise and intensity.
Significant differences found at the $p<0.05$ level of significance.

TABLE 4: Regression Slopes, Point of Origin and Linear Trend of the SBP and DBP Response to the Prewalk HG, Combined Walk+HG and the Postwalk HG at 30% and 40% MVC

Handgrips	Slope	Point of Origin	Linear Trend (r^2)
SBP 30% MVC †			
a) Prewalk HG	0.18 (c)	127.4	0.88
b) Walk+HG	0.12 (c)	146.4	0.34
c) Postwalk HG	0.23 (a,b)	137.5	0.87
SBP 40% MVC ††			
a) Prewalk HG	0.33	122.8	0.97
b) Walk+HG	0.37	143.0	0.97
c) Postwalk HG	0.38	141.8	0.93
DBP 30% MVC †			
a) Prewalk HG	0.23 (c)	79.8	0.97
b) Walk+HG	0.20 (c)	78.1	0.92
c) Postwalk HG	0.32 (a,b)	86.9	0.95
DBP 40% MVC †			
a) Prewalk HG	0.32 (c)	79.2	0.95
b) Walk+HG	0.31 (c)	77.0	0.97
c) Postwalk HG	0.40 (a,b)	86.9	0.98

Note:

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

†† No significant differences at the $p<0.05$ level of significance found between the systolic blood pressure slopes for handgrip exercises at 40% MVC.

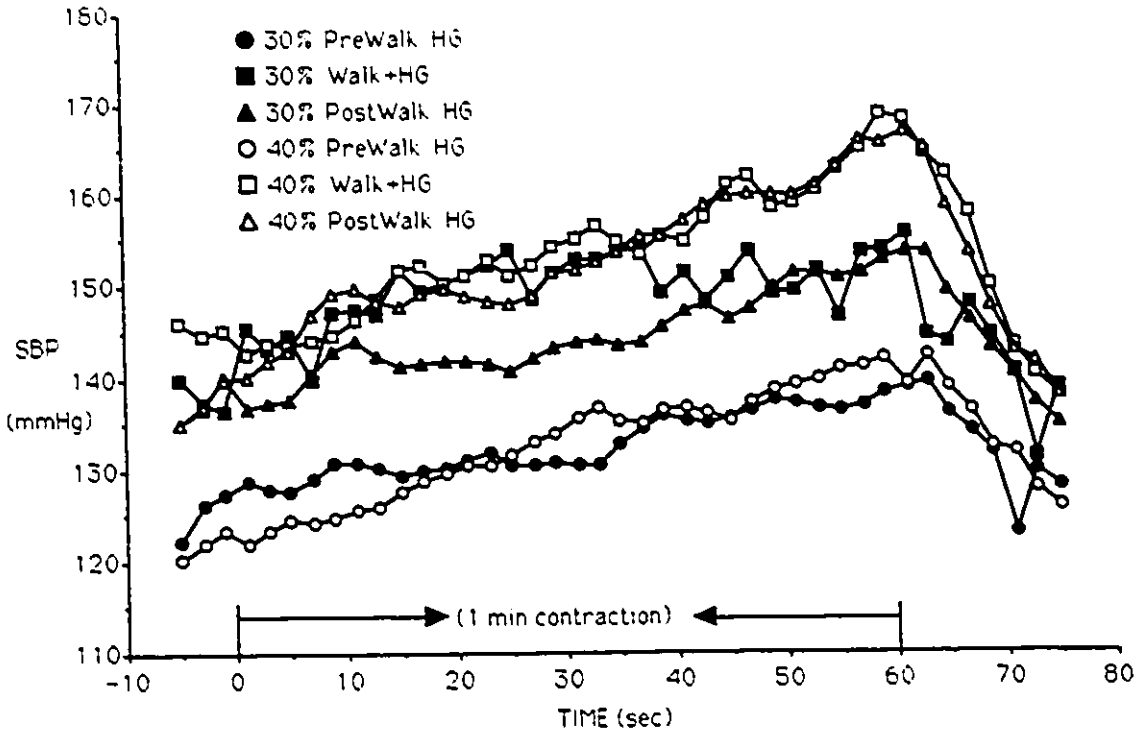

Figure 1: Continuous SBP Responses (2 sec intervals) to the PreWalk HG, Walk+HG and PostWalk HG conditions at 30% and 40% MVC from 5 sec before to 15 seconds following contraction.

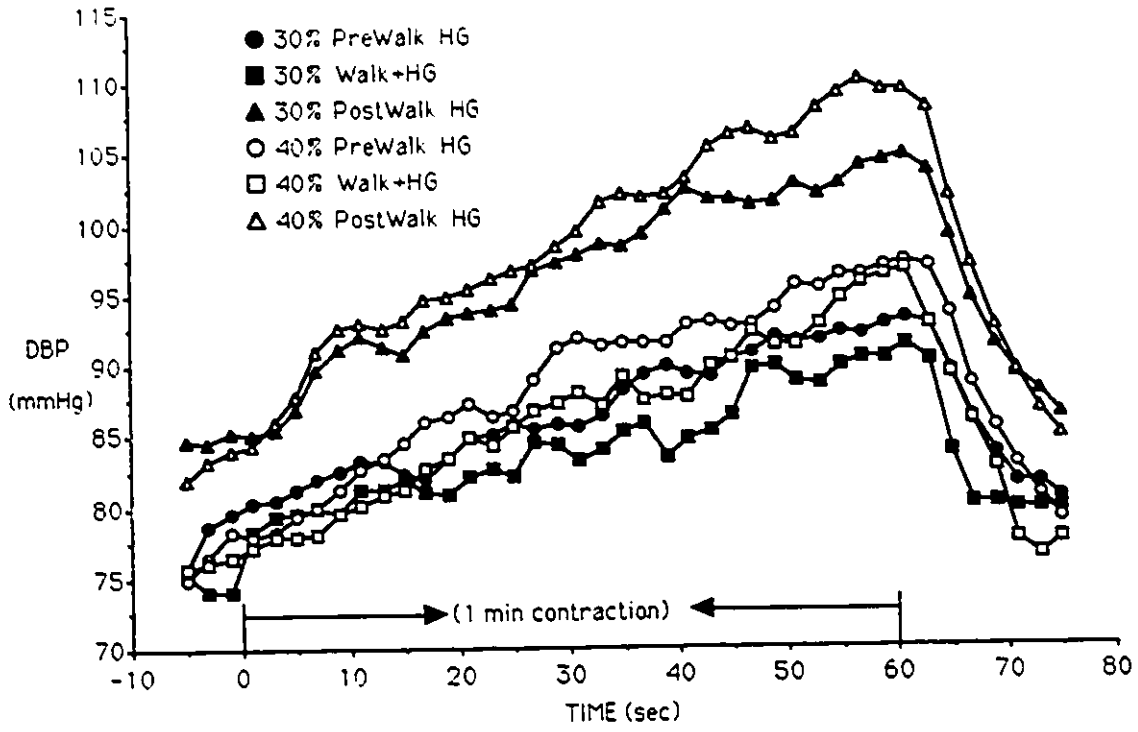

Figure 2: Continuous DBP Responses (2 sec intervals) to the PreWalk HG, Walk+HG and PostWalk HG conditions at 30% and 40% MVC from 5 sec before to 15 seconds following contraction.

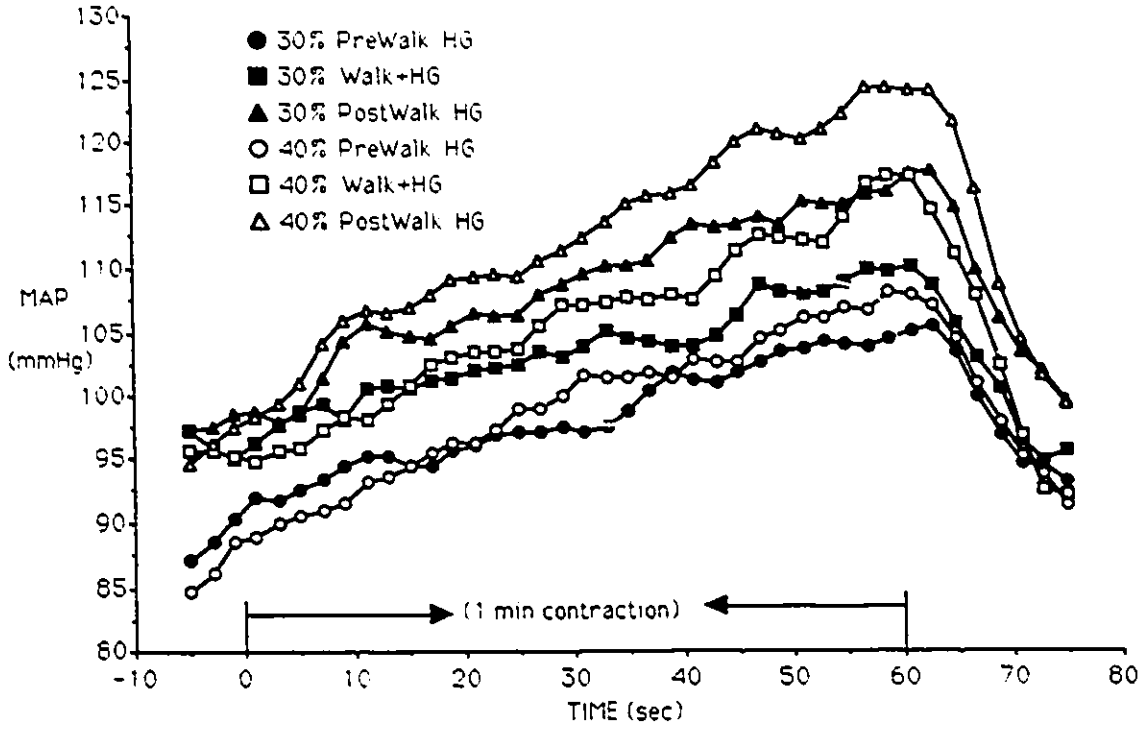

Figure 3: Continuous MAP Responses (2 sec intervals) to the PreWalk HG. Walk+HG and PostWalk HG conditions at 30% and 40% MVC from 5 sec before to 15 seconds following contraction.

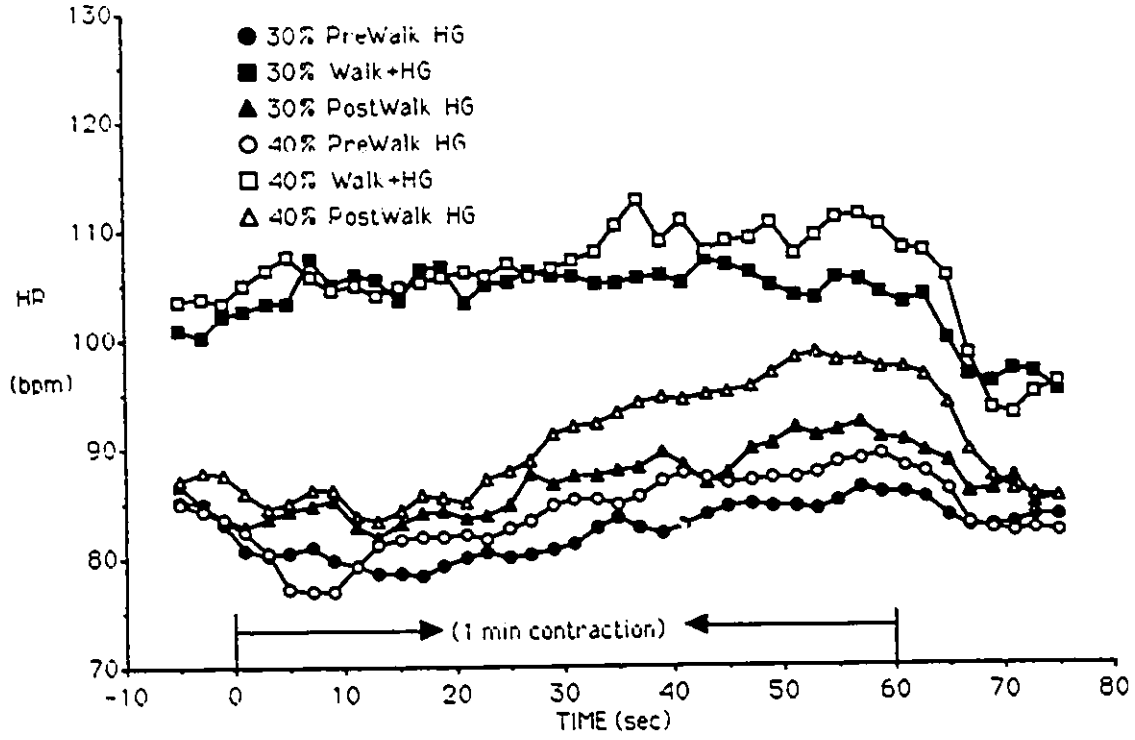

Figure 4: Continuous HR Responses (2 sec intervals) to the PreWalk HG, Walk+HG and PostWalk HG conditions at 30% and 40% MVC from 5 sec before to 15 seconds following contraction.

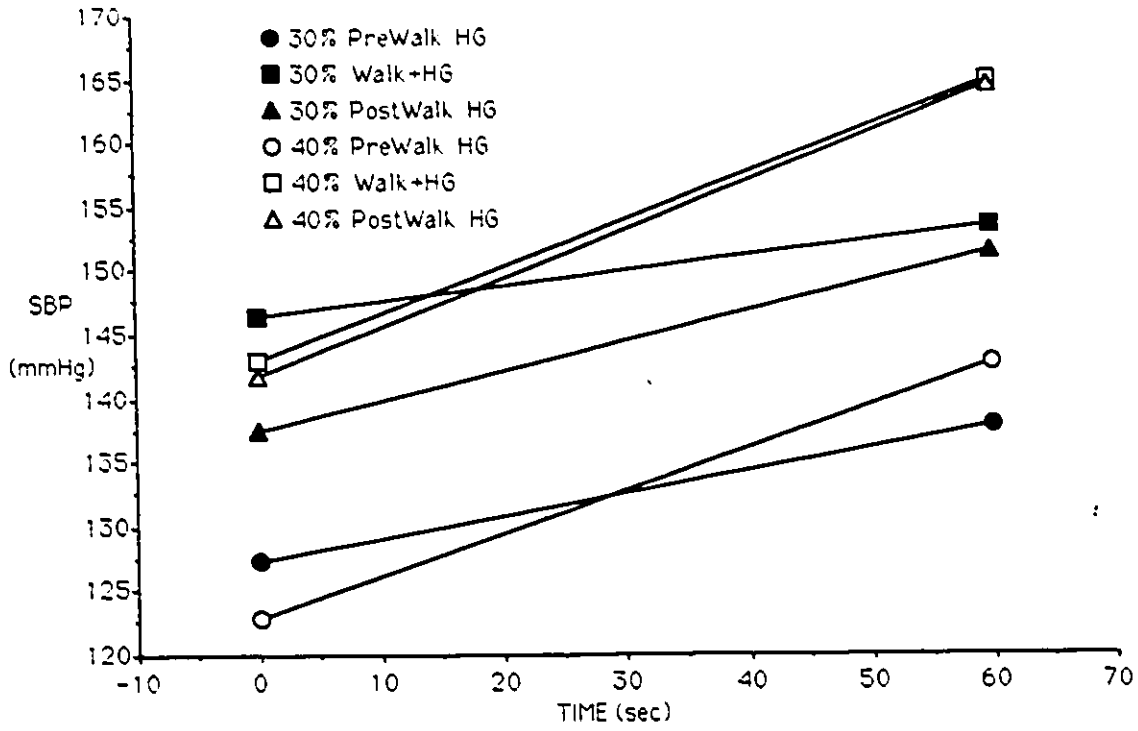

Figure 5: Regression slopes of SBP for the PreWalk HG, Walk+HG and PostWalk HG conditions at 30% and 40% MVC.

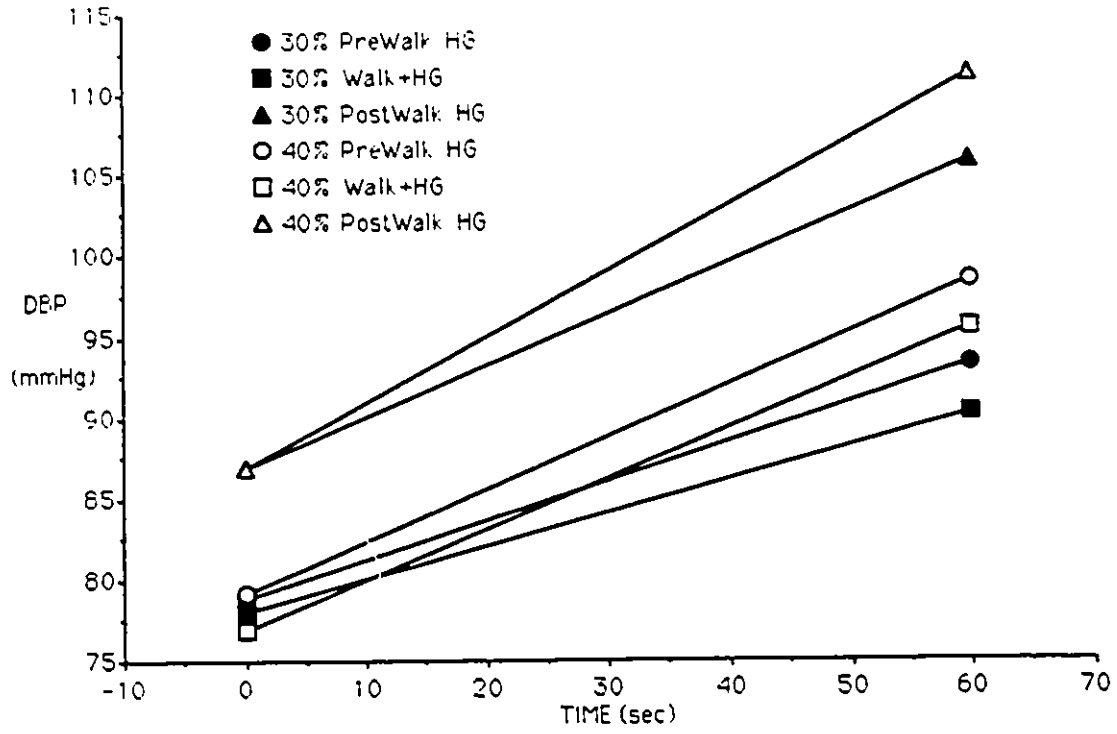

Figure 6: Regressions slopes of DBP for the PreWalk HG, Walk+HG and PostWalk HG conditions at 30% and 40% MVC.

I

INTRODUCTION

Exercise is classified as either dynamic or static. Dynamic exercise includes those activities in which there is movement, having contraction and relaxation phases, whereby external work is performed (Temkin, 1983; McArdle et al., 1986). Conversely, static exercise is the maintenance of a muscle contraction; since no movement occurs no external work is performed (Temkin, 1983). Due to the nature of the two forms of exercise each places a different stress on the cardiovascular system.

The cardiovascular response to dynamic exercise is primarily directed towards "meeting the oxygen demands" of the exercising muscles. This is accomplished by increasing blood flow through vasodilation of the blood vessels to the active muscle and vasoconstriction of vessels in nonactive tissues (Lind and McNicol, 1967; Colombo et al., 1989). The response results in an increase in the circulating blood volume which is accommodated by the heart increasing its cardiac output. The vasodilation of the arterioles reduces the total peripheral resistance. Therefore, the rise in systolic blood pressure attributed to the increase in cardiac output is limited by a fall in systemic vascular resistance, while diastolic blood pressure remains the same (Perez-Gonzalez, 1981; Lewis et al., 1985)

The initial rapid rise in heart rate and systolic blood pressure with static exercise is similar to the initial response to dynamic exercise. However, with static exercise the cardiovascular response is directed towards 'maintaining perfusion pressure' (Humphreys and Lind, 1963). Since the muscle contraction is held during static exercise there is no relaxation phase and the tension within the muscle is not released, thus resulting in an increase in intramuscular pressure. The rise in intramuscular pressure, along with mechanical pressure, can compress the blood vessels within the exercising muscles, thereby limiting the blood flow (Humphreys and Lind, 1963; Kilbom and Persson, 1981; Lewis et al., 1985). The body counteracts by increasing the perfusion pressure to maintain an adequate blood supply to the working muscle (Humphreys and

Lind, 1963). This is accomplished by vasodilation of the blood vessels within the active muscle and strong vasoconstriction of blood vessels throughout the rest of the body (Lind and McNicol, 1967). The strong vasoconstrictory response, along with the mechanical compression of the blood vessels, results in a rise in both systolic and diastolic blood pressures without a decrease in systemic vascular resistance (Lind and McNicol, 1967; Lewis et al., 1985). The large increase in blood pressure is termed the pressor response.

The blood pressure response to both isometric and dynamic exercise therefore, are distinctly different. In dynamic exercise, total peripheral resistance decreases and a much greater volume of blood is pumped by the heart whereas with static exercise, the circulating blood volume remains the same but there is a much greater increase in blood pressure.

Few studies however, have examined the effect of combined isometric and dynamic exercise on the cardiovascular response. An early study by Lind and McNicol (1967) examined the blood pressure response to a handgrip contraction superimposed on dynamic walking exercise on four healthy male subjects. Handgrip contractions of 30 and 50% maximum voluntary contraction (MVC) were combined with three different walking intensities of 2.5, 3 and 3.5 m/hr. The results indicated that the increment in systolic blood pressure was the same regardless of whether the handgrip contraction was performed alone or when combined with the different walking intensities. Thus, they indicated that the pressor response occurred despite the decrease in peripheral resistance associated with dynamic exercise. The maintenance of the pressor response was attributed to a greater vasoconstrictor response (Lind and McNicol, 1967). A more recent study by Jetté, Kottmann, Landry and Blümchen (1990) superimposed static handgrip contractions of 33% MVC on three different workloads (40, 80 and 120 watts) of supine cycle ergometry using coronary heart disease patients. Their results supported the findings of Lind and McNicol (1967), with a similar pressor response occurring at rest and at all three levels of dynamic exercise. Jetté et al. (1990) therefore, suggested that the increase in blood pressure was due to greater sympathetic stimulation causing an increased vasoconstriction in the periphery. However, both studies provide limited evidence that the pressor response is maintained during dynamic exercise since blood pressure is measured with a standard sphygmomanometer and stethoscope and subjects were not

screened prior to testing for an exaggerated blood pressure response to exercise. In addition, the study by Lind and McNicol (1967) only tested four male subjects while Jetté et al.(1990) tested coronary heart disease patients. The purpose of this study, therefore, is to determine whether the exaggerated increase in systolic blood pressure associated with static exercise is blunted when performed during dynamic exercise in normotensive healthy middle-aged male subjects. The heart rate and blood pressure will be monitored continuously with measurements being recorded every 2 seconds utilizing a Finapress BP monitor.

1.2 Statement of The Problem

To determine whether dynamic exercise has a blunting effect on the blood pressure response to an isometric stressor.

1.3 Hypothesis

The pressor response will occur with a static handgrip contraction regardless of whether it is performed at rest or during dynamic walking exercise. The static handgrip performed at 40% MVC will produce a significantly greater pressor response than the handgrip at 30% MVC.

1.4 Scope of The Study

Twenty (24 - 32 yrs) male subjects were selected for the study. Subjects were requested to come on two days for testing. On the first day, subjects were requested to complete the modified PAR-Q (Appendix A), informed consent (Appendix B) and health questionnaire (Appendix C) prior to testing. Next, subjects were seated for five minutes with the Finapress monitor on the subject's right hand. At the conclusion of five minutes, the resting blood pressure and heart rate measurements were recorded. The resting measurements were utilized to verify that subjects were normotensive. The subjects were then administered the modified Canadian Aerobic Fitness Test (CAFT) to screen for subjects who demonstrated an exaggerated blood pressure response to dynamic exercise and also to calculate each individual's predicted maxVO_2. Basic

anthropometric measurements including age, height, weight, girths (chest, waist and hip), were evaluated along with the results of the CAFT to determine whether the subjects represented a normal sample of the population. Each subject performed three consecutive maximum handgrip contractions (with their left hand) from which the maximum was used to establish the participant's maximum voluntary contraction.

On the second day, subjects' resting blood pressure and heart rate measurements were recorded after five minutes seated and again following one minute standing. Subjects were randomly divided into two equal groups. Group A performed all exercises initially with handgrip contractions of 30% MVC followed by 40% MVC and group B performed handgrip contractions initially with 40% MVC, followed by 30% MVC. Next, subjects were requested to maintain a handgrip contraction at 30% MVC for one minute, and after a five minute recovery period, at 40% MVC for one minute. Heart rate and blood pressure measurements were recorded every two seconds during each handgrip contraction until 15s postexercise with the Finapres monitor. Following a five minute recovery period, subjects repeated the 30% MVC handgrip contraction during the fourth minute of dynamic exercise (walking on a treadmill at a heart rate of 100 beats per minute) and again one minute immediately following exercise. The contraction was maintained for one minute each time. Subjects were given ten minutes recovery time and then asked to repeat the protocol using a handgrip contraction of 40% MVC. The continuous blood pressure measurement enabled one to note immediate changes in blood pressure and determine whether dynamic exercise blunted the pressor response to static exercise. The results were analyzed using a two-way factorial ANOVA design with repeated measures. When a significant difference was found the Least Means Square post hoc analysis was utilized to determine the difference in means.

1.5 Definition of Terms

Cardiac output (Q):

This is the amount of blood pumped by the heart in one minute; the product of the heart rate and stroke volume.

Maximal oxygen consumption (max $V0_2$):

The maximal oxygen consumption as predicted from the results of the last stage of CAFT using the equation of Jetté (Appendix D).

Maximum voluntary contraction (MVC):

The maximum value of three repeated isometric handgrip contractions.

Pressor response:

The rapid increase in blood pressure during an isometric contraction caused by an increase in cardiac output with little change in peripheral resistance.

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 and is termed peripheral resistance.

II

LITERATURE REVIEW

2.1 Introduction

Dynamic and static exercise are commonly classified according to the cardiovascular response elicited. The study by Alam and Smirk (1937) established that the large increase in the blood pressure associated with static exercise was the main hemodynamic difference between the two forms of exercise. Further research on the hemodynamic differences between static and dynamic exercise has examined the mechanisms which control these factors. However, only a few studies have explored the hemodynamic and pressor response to the imposition of static exercise on dynamic exercise. One such study by Hung, McKillip, Savin, Magder, Kraus, Houston, Goris, Haskell and DeBusk (1982) proposed that the vasodilatory effect of dynamic exercise may blunt the vasoconstrictive response of isometric exercise when the two modes of exercise are combined. The purpose of my paper was to determine whether dynamic exercise could, in fact, blunt the pressor response associated with static exercise.

2.2 Mode of Contraction

The mode of contraction is primarily responsible for the difference in the hemodynamic responses to static and dynamic exercise. In static exercise, the muscle fibre maintains a contraction over a constant fibre length whereas in dynamic exercise, the muscle fibre length is continually changing (shortening and lengthening) with the contraction and relaxation phases (Asmussen, 1981; Temkin, 1983). Thus, due to the change in fibre length, external work is performed with dynamic exercise but not during static exercise (Asmussen, 1981; Temkin, 1983; Lewis et al. 1985).

2.2.1 Energy Cost

The energy cost of static and dynamic exercise has been compared using oxygen consumption values. When both static and dynamic exercises were performed at similar tensions, the oxygen consumption values were found to be lower with static exercise (Asmussen, 1981; Lewis et al. 1985). The oxygen consumption and thus the energy expenditure associated with dynamic exercise was due to the external work performed. Total energy is equal to the activation energy plus the product of a constant and the work performed [E(tot.) = E(act.) + kWork]. Activation energy is the same for dynamic and static exercise at equal tensions (Asmussen, 1981). Thus, for static exercise, the total energy is equivalent to the activation energy, whereas with dynamic exercise, the total energy is equal to the energy for a similar static contraction plus the kWork factor (Asmussen, 1981). However, for dynamic exercise to approximate the same tension value as static exercise, a greater recruitment of muscle fibres as well as more energy are required for the repetitive development of tension (Asmussen, 1981, Lewis et al. 1985).

Static contractions of maximal intensities have a greater anaerobic contribution to the total energy cost as a result of partial or total blood flow occlusion which limits the oxygen delivery (Asmussen, 1981). Thus, oxygen consumption during exercise may not accurately indicate the total energy requirements during the exercise unless recovery values are also measured.

During combined static and dynamic exercise, the oxygen consumption is greater than dynamic exercise alone. However, the rise in oxygen uptake with the addition of static (handgrip) contraction is the same regardless of the intensity of dynamic exercise (Kilbom and Persson, 1981). Therefore, the total oxygen consumption and energy cost are simply the addition of the static and dynamic exercise components.

Thus, the difference in mode of contraction between static and dynamic exercise affects their respective energy costs. The energy cost associated with static exercise is primarily related to the activation energy to recruit muscle fibres, whereas with dynamic exercise the energy cost is equal to the activation energy plus a factor related to the amount of work performed. When

comparing the oxygen demands between the two modes of exercise, the oxygen requirement rises with an increase in dynamic exercise intensity, whereas with static exercise at a similar intensity, the oxygen demand is less due to a greater anaerobic energy contribution. With combined static and dynamic exercise the energy cost is simply derived from the addition of the two modes of exercise.

2.3 Heart Rate

Irrespective of the mode of contraction, the onset of exercise produces an immediate tachycardia response which is followed by a more gradual increase in heart rate (Haskell et al. 1981).

2.3.1 Autonomic Control

Tachycardia is a reflex response to the onset of exercise. The rapid increase in heart rate with the initiation of exercise (either static or dynamic), as well as the abrupt drop in heart rate with exercise cessation, indicates a close association between the control of heart rate and the actual mechanical event (Asmussen, 1981). McCloskey and Streatfeild (1975) used blood flow occlusion cuffs on the active arms during and following exercise to determine whether the reflex heart rate response was related to an ischemic event or to muscular activity. The results showed that the heart rate response with respect to the onset and cessation of exercise was not affected by blood flow occlusion (McCloskey and Streatfeild, 1975). Thus, these results support Asmussen's theory that the change in heart rate is associated with muscle activity. These findings were supported in a similar study employing blood flow occlusion by Rusch et al. (1981), and thereby indicating that muscle ischemia, an elevation in muscle metabolites and hormonal factors has only minimal effect on the heart rate response to exercise (Asmussen, 1981).

The heart rate response to static and dynamic exercise has been divided into an initial rapid response followed by a more gradual elevation in heart rate (Asmussen, 1981; Haskell et al. 1981; Goldstraw and Warren, 1985). This immediate heart rate response with the onset of

exercise has been attributed to parasympathetic or vagal withdrawal, while the later rise in heart rate has been associated with sympathetic stimulation. Martin et al. (1985) performed a study on healthy, young males using autonomic drug blockade to assess the mechanisms which regulate the heart rate response to static exercise. The administration of atropine was used as a parasympathetic blockade and propranolol as a sympathetic blockade. The rapid immediate response was absent with the administration of atropine, although the later increase in heart rate still occurred. In contrast, the sympathetic blockade with propranolol demonstrated a typical heart rate response to static exercise although the magnitude attained was lower. When a combination of atropine and propranolol was administered, a complete autonomic blockade occurred which inhibited any increase in heart rate (Martin et al., 1985). From these results, Martin et al. (1985) concluded that the initial rapid response must be attributed to the withdrawal of vagal tone and parasympathetic activity and that the later increase in heart rate was due to sympathetic stimulation. Their findings confirm the combined results of earlier studies by Freychuss (1970) and Clement et al. (1984) who examined the role of the parasympathetic and sympathetic systems on the heart rate response to isometric contraction with the administration of atropine and propranolol respectively. Recent studies have supported these findings, when examining the heart rate response specifically to static exercise (Goldstraw and Warren, 1985) to dynamic exercise (Blomqvist et al., 1981) and combined exercise (Kilbom and Persson, 1981). A similar study to Martin et al. (1985) using drug blockade on cats, also produced comparable results (Gonyea et al. 1981). Lewis et al. (1985) concluded that there is a similar heart rate response to submaximal static and dynamic exercise when utilizing the same muscle groups. Thus, the same mechanisms regulate the heart rate response to exercise regardless of whether it is performed in the static, dynamic or combined mode. However, the maximum heart rate achievable is lower with static than dynamic exercise (Lewis et al., 1985). Asmussen (1981) has suggested that there is more feedback from the active muscles with dynamic exercise which may account for the higher absolute heart rate attained. The magnitude of the heart rate response to exercise is proportional to the degree of sympathetic stimulation. Martin et al. (1985) have indicated that sympathetic stimulation is primarily influenced by the intensity and duration of contraction although the intensity of the exercise determines the length of time a contraction can be maintained. Bannister and Griffiths (1972) examined the level of circulating catecholamines at various intensities of dynamic cycling exercise. Their results are in

agreement with the findings of Martin et al. (1985) on static exercise suggesting that the amount of norepinephrine and epinephrine secreted during exercise is proportional to the intensity of exercise.

A study by Lind and McNicol (1967) examined the cardiovascular response to a dual handgrip contraction at 20%, 30% and 50% MVC intensities superimposed on three dynamic treadmill exercise rates (at oxygen uptakes of 1.1, 1.7 and 2.8 l/min) with four healthy males. The results indicated that the rise in heart rate with a single handgrip contraction at rest was equivalent to the heart rate increase when a handgrip contraction of similar intensity was added to dynamic exercise. A study by Jetté et al. (1990), combined handgrip exercise with three different levels of supine cycle ergometry (40 W, 80 W and 120 W) and supported the findings of Lind and McNicol (1967) that the increase in heart rate with the superimposition of handgrip on dynamic exercise was equivalent to the handgrip contraction performed alone. Conversely, Kilbom and Persson (1981) comparing the heart rate and blood pressure response to a handgrip contraction of 20% MVC superimposed on dynamic exercise (100 W cycle ergometry) found that the heart rate elevation was less with the addition of handgrip than when the handgrip exercise was performed separately.

The heart rate response to either dynamic or static exercise has been shown to be directly related to the onset and cessation of muscle activity. The control mechanisms are the same for both modes of exercise. The withdrawal of parasympathetic stimulation is responsible for the initial rapid response and the subsequent gradual elevation in heart rate is due to the progressive increase in sympathetic stimulation. Greater elevations in heart rate occur with dynamic exercise and have been attributed to a more pronounced sympathetic activation observed during this mode of exercise.

2.4 Cardiac Output

The magnitude of the cardiac output is directly related to the product of the heart rate and stroke volume response to exercise. With dynamic exercise an increase in heart rate and stroke volume is associated with a rise in the oxygen demand, thus producing a linear relationship between cardiac output and oxygen uptake. Conversely during static exercise, the rise in cardiac

output is more closely related to the blood pressure elevation.

2.4.1 Heart Rate and Stroke Volume Contributions

Lewis et al. (1985) examined the role of muscle mass and mode of contraction (static versus dynamic) on the circulatory response to exercise. The static handgrip and leg extensions were performed at 24-25% MVC, while the dynamic exercise component consisted of handgrip and leg extensions at 33-40 and 20 contractions/min respectively. Lewis et al.(1985) observed a more pronounced increase in cardiac output with leg extension, than with handgrip, irrespective of the mode of contraction. They concluded therefore, that the magnitude of the response was related to the muscle mass regardless of mode of contraction. However, when they compared static and dynamic exercise performed with the same muscle mass and tension, the greater energy requirement associated with dynamic exercise resulted in a larger cardiac output. Blomqvist et al. (1981) also contrasted the hemodynamics of static and dynamic exercise and found cardiac output and stroke volume to be closely associated with oxygen uptake, thus supporting the theory that stroke volume contributes to the increase in cardiac output with dynamic exercise but has little effect during static contractions. Perez-Gonzales, Schiller and Parmley (1981) conducted a study to assess the cardiovascular response to isometric exercise using noninvasive and direct methods. A sustained handgrip contraction at 30% MVC caused an increase in heart rate and cardiac output with no significant change in stroke volume. From these observations, they concluded that the increase in cardiac output was due primarily to the significant rise in heart rate. When the handgrip contraction was repeated following the administration of propranolol, no significant increase in heart rate or cardiac output was observed, although stroke volume was preserved. Thus, the ability to maintain the stroke volume in the presence of an increased afterload was attributed to both a pronounced increase in beta-adrenergic stimulation causing a rise in heart rate and an increase in end-diastolic diameter (Frank-Starling mechanism) (Perez-Gonzales et al. 1981). These results are in agreement with the findings of Lind et al. (1964) and Martin et al. (1974). Seaman, Wiley, Zechman and Goldey (1973) studied venous tone in relation to the cardiovascular responses during static handgrip exercise. An increase in venoconstriction with static exercise was found comparable in magnitude to dynamic exercise.

Seaman et al. (1973) suggested that venoconstriction may play a role in maintaining and/or increasing the cardiac output during static exercise. Studying the effect of combining handgrip to various intensities of supine dynamic leg exercise, Kilbom and Persson (1981) found no significant change in stroke volume with handgrip exercise alone or combined. The increase in cardiac output with the addition of handgrip to dynamic exercise was less than handgrip exercise performed alone. Thus, the rise in cardiac output was attributed solely to the change in heart rate in both instances (Kilbom and Persson, 1981). Shepherd et al. (1981) summarized that the increase in cardiac output with dynamic exercise is closely related to the oxygen demand irrespective of muscle mass and intensity. Whereas in static exercise, the cardiac output is much greater than the oxygen demand and may be associated with muscle mass and the degree of blood flow occlusion (Lind et al., 1964; Shepherd et al., 1981).

2.4.2 Muscle Pump

In dynamic exercise, the rhythmic contraction and relaxation phases act as a pumping action in returning the blood to the heart (Temkin, 1983; McArdle et al., 1986). This muscle 'pump effect' increases the venous return to the heart (preload) and stroke volume which contributes to the rise in cardiac output (Asmussen, 1981; Shepherd et al., 1981). In contrast, isometric exercise (static contraction) does not elicit rhythmical contraction and relaxation phases, and therefore does not aid the return of blood to the heart. The increase in cardiac output with static exercise is therefore less than that of dynamic exercise (Temkin, 1983).

2.4.3 Distribution of Cardiac Output

The distribution of the cardiac output during exercise has been studied utilizing blood flow measurements and temperature changes. A portion of the increase in cardiac output with static, dynamic and combined exercise is directed to the heart to sustain the elevation in myocardial oxygen consumption (Kilbom and Persson, 1981). Static exercise is associated with widespread vasoconstriction in contrast to a vasodilatory response with dynamic exercise (Asmussen, 1981; Lind and McNicol, 1967; Kilbom and Persson, 1981). Kilbom and Persson

(1981) measured leg blood flow during static, dynamic and combined exercise. Blood flow to the active muscle during static contraction is restricted due to mechanical occlusion of the blood vessels, whereas with dynamic exercise, no hindrance to blood flow is noted. During static handgrip exercise alone and when superimposed on dynamic exercise no significant increase in leg blood flow was observed (Kilbom and Persson, 1981). However, a rise in the temperature of the subcutaneous tissue of the thorax along with a decrease in venous temperature suggested an increase in skin perfusion. These findings are in agreement with Asmussen (1981) who stated that partial blood flow occlusion to the active muscle results in a redistribution of blood to the non-exercising parts. Conversely, studies by Humphreys and Lind (1963) and Lind et al. (1964) did not find any increase in blood flow to the non-active muscles, splanchnic, renal or cutaneous vascular beds and only a slight elevation in blood flow to the active muscles. These findings were further supported in a later study by Lind, Dahms, Williams and Petrofsky (1981) which measured blood flow and EMG activity in statically exercising and resting arms. Their observations on the active arm indicated a direct linear relationship between blood flow and EMG activity. They also found that contractions of long duration often elicited an increase in EMG activity in both the exercising and resting arms when there was an increase in effort to maintain the contraction intensity. Thus, when they measured blood flow and EMG in the contralateral resting arm, it was not surprising that there was an increase in EMG activity and blood flow in some subjects and no increase in either factor in other subjects. Their results, therefore, indicate that the increase in cardiac output with static exercise is directed only to the active muscles. In conclusion, Lind et al. (1964) stated that the disproportionate increase in cardiac output with static effort is confined by general vasoconstriction and thus contributes to the exaggerated rise in blood pressure. In contrast, the increase in cardiac output with dynamic exercise is channelled directly to the active muscles with no hindrance to blood flow thus satisfying the oxygen demands of the muscle (Asmussen, 1981).

In conclusion, the rise in cardiac output with static exercise is solely a result of the increase in heart rate with little to no change in stroke volume, however the magnitude of the cardiac output increase is related to the degree of blood flow occlusion and size of the active muscle mass. The widespread systemic vasoconstriction increases the perfusion pressure, thereby

channelling the cardiac output primarily to the active muscle mass. During dynamic exercise, cardiac output increases in proportion to both heart rate and stroke volume changes and results in a larger volume of circulating blood. The magnitude of the rise in cardiac output is directly related to the oxygen demands of the active muscle and greater than an equivalent static workload due to vasodilatation within the active muscle.

2.5 Muscle Perfusion

The mode of contraction has a significant effect on the blood perfusion of an active muscle. With dynamic exercise there are rhythmic contraction and relaxation phases. During the relaxation phase the blood flow is restored through the muscle vasculature (Asmussen, 1981). In contrast to dynamic exercise, sustained contractions (>15% MVC) of static exercise can progressively occlude blood flow through the exercising muscle (Asmussen, 1981; Saltin et al., 1981). A compromise between intramuscular pressure, metabolic factors and perfusion pressure are the major factors influencing muscle perfusion (Shepherd et al., 1981).

2.5.1 Intramuscular Fluid Pressure

During a sustained static contraction, the active muscle fibres swell and stiffen within the fascia preventing the flow of fluid between the muscle compartment and interstitial tissue thus, resulting in a rise in intramuscular fluid pressure (Sejersted et al., 1984). Humphreys and Lind (1963) measured blood flow in the forearm during static exercise and observed that blood flow to the active muscle was not occluded until exercise intensity exceeded 70% MVC. However, Sejersted, Hargens, Kandel, Blom, Jensen and Hermansen (1984) suggested that the intensity of contraction at which blood flow is occluded, may differ depending on the muscle and its shape. They suggested that muscles which have short, curving fibres may develop greater intramuscular pressure since the contractile stress is directed towards the center of the muscle. In contrast, muscles which have long straight fibres, stress is transferred to the tendons and the intramuscular pressure is lower. Sejersted et al. (1984) examined the intramuscular fluid pressure at different locations within active muscle compartments and the effect of the intensity of

contraction on the magnitude of the intramuscular pressure. The results showed a progressive increase in pressure from the periphery to the centre of the muscle where the concentration of contracting muscle fibres was greatest. This is in agreement with similar findings by Saltin et al. (1981) stating that although, the intramuscular pressure is linearly related to the force, there is a large variation in the magnitude of the pressure at various locations within the muscle. In addition, contractions less than 15% MVC resulted in low pressures whereas contractions greater than 15% MVC obtained much larger pressures. In a progression from low to high intensity contractions, there was a progressive reduction in blood flow beginning with the microvascular circulation to an eventual occlusion of blood flow to the active muscle (Sejersted et al., 1984). The rise in intramuscular fluid pressure, therefore, compresses the blood vessels in the active muscle limiting blood perfusion and oxygen delivery thereby resulting in a greater anaerobic energy contribution (Asmussen, 1981; Kilbom and Persson, 1981; Lewis et al. 1984). Conversely, the active contraction and relaxation phases associated with dynamic exercise prevents a rise in intramuscular fluid pressure and restores blood flow through the active muscle (Asmussen, 1981).

2.5.2 Metabolic Factors

Saltin et al. (1981) examined the potassium ion and lactate concentrations within the interstitial tissue of a muscle group during contractions at intensities of 15, 25 and 50% MVC. At a contraction of 15% MVC there was a slight elevation in potassium and lactate concentrations within the interstitial fluid and muscle. However, at intensities of 25 and 50% MVC a much greater accumulation was noted. Saltin et al. (1981) associated this accumulation with the decrease in blood perfusion and mechanical compression with contractions of increasing intensities. One of the earlier studies examining the cardiovascular response to static exercise was performed by Alam and Smirk (1937). Their results also indicated that the accumulation of metabolites within the muscle was directly related to the intensity of the exercise and degree of blood flow occlusion. Alam and Smirk (1937) suggested that the increased concentration of metabolites had a local vasodilatory effect on the blood vessels within the contracting muscle, thus counteracting the general vasoconstriction and enhancing blood perfusion to the muscle. Edwards and Wiles' (1981) current review examined the energy requirements versus the onset of fatigue with varying

levels of intensity and blood flow occlusion. They concluded that at forces greater than 15% MVC there was a progressive occlusion of blood flow causing the energy supply to become increasingly anaerobic, in agreement with the findings of Saltin et al. (1981). Contractions less than 10-25% MVC could be maintained for long durations, however at greater intensities, the onset of fatigue progressively increased until contractions of 60-70% MVC where blood flow to the muscle was completely occluded and time to fatigue was greatly reduced (Asmussen, 1981). Although the time a contraction can be maintained was found to be inversely related to the rate of energy turnover, further studies revealed that failure of membrane excitation may be the limiting factor (Edwards and Wiles, 1981). Similarly, the greater energy requirement of dynamic exercise, produces an excess of metabolites causing vasodilation of the local blood vessels (Blomqvist et al., 1981). However, the metabolites do not accumulate within the muscle as in static contractions, as a result of the absence of blood flow occlusion. Kilbom and Persson (1981) examined the addition of static handgrip contraction to dynamic leg exercise and found no change in the blood flow to the legs. They concluded that the local metabolic vasodilation must be capable of counteracting the widespread vasoconstrictory response associated with static exercise (Kilbom and Persson, 1981).

2.5.3 Perfusion Pressure

There is a large discrepancy between the perfusion pressures of static and dynamic exercise as a result of the increased pressure load associated with the widespread systemic vasoconstriction during static exercise, in comparison to the combination of a rise in circulating blood volume and decrease in peripheral resistance during dynamic exercise (Quarry and Spodick, 1974). A comparison of the cardiovascular responses to static handgrip contractions of 15, 30, 50 and 100% MVC was performed by Quarry and Spodick (1974). A progressive rise in systolic and diastolic blood pressure was observed with an increase in the duration of contraction and with an increase of intensity. The rise in blood pressure was attributed to a corresponding elevation in cardiac output (Quarry and Spodick, 1974). Using blood flow occlusion during and following static contraction, Alam and Smirk (1937) provided evidence that the rise in blood pressure was directly associated with an ischemic response originating in the active muscle. Lind et al. (1964) suggested that the rise in blood pressure may be advantageous, by elevating the perfusion pressure

to overcome the increased resistance to blood flow in the active muscle and improve circulation. In contrast, during dynamic exercise only the systolic component of blood pressure increases (Quarry and Spodick, 1974). However, similar to static contractions, the intensity and frequency of the exercise is directly related to the magnitude of the hemodynamic response (Shepherd et al., 1981). In addition, Shepherd et al. (1981) suggested that similar mechanisms regulate the increase in arterial blood pressure that counteract the hindrance to blood flow.

Muscle perfusion during either static or dynamic exercise is a compromise between intramuscular pressure, the release of metabolites and perfusion pressure. There is a much greater resistance to muscle perfusion with static exercise due to the constant tension within the muscle causing mechanical occlusion of blood vessels, greater intramuscular pressure and systemic vasoconstriction. Thus, the local release of metabolites and the increase in perfusion pressure are the only factors counteracting this increased resistance to blood flow in the active muscle during static exercise. In dynamic exercise, the blood flow is restored during the relaxation phase of contraction and there is more extensive vasodilation within the active muscle; therefore, blood flow better equates with the oxygen demands of the muscle. During combined exercise, the local vasodilator mechanisms associated with dynamic exercise prevented a decrease in the muscle perfusion by counteracting the systemic vasoconstriction.

2.6 Blood Pressure Response

The large pressor response associated with static exercise has been identified as the major hemodynamic distinction between static and dynamic exercise. The pressor response occurs only during isometric exercise as a result of differences in arterial systolic, diastolic and mean blood pressure and systemic vascular resistance changes with static and dynamic exercise (Lind and McNicol, 1967). The hemodynamic differences between static and dynamic exercise are perhaps best summarized by Temkin (1983) in Table 1.

TABLE 5

Hemodynamic Responses to Exercise

	Isometric	Dynamic
Heart Rate	+	+++
Systolic Blood Pressure	+++	++
Diastolic Blood Pressure	+++	-/↓
Mean Blood Pressure	+++	-/↓
Cardiac Output	++	+++
Stroke Volume	-/+	+++
Systemic Vascular Resistance	-/+	↓

+	minimally increased
++	moderately increased
+++	markedly increased
-/+	no change
-/↓	minimally decreased
↓	decreased

(adapted from Temkin, 1983, p.381, Arizona Medicine.)

2.6.1 Systolic and Diastolic Changes

The typical blood pressure response to a static contraction is an elevation in both systolic and diastolic measures, whereas with dynamic exercise only the systolic blood pressure rises and diastolic remains stable (Tuttle and Horvath, 1957; Lind and McNicol, 1967; Asmussen, 1981). This exaggerated rise in systolic and diastolic blood pressure with static exercise has been attributed to a proportional increase in cardiac output, and widespread vasoconstriction (Lind and McNicol, 1967; Asmussen, 1981; Jandik et al., 1984). However, studies by Freychuss (1970) and Williams, Mudd and Lind (1981) examining the role of the sympathetic system, attributed the initiation and maintenance of the pressor response with isometric exercise, primarily to vasoconstriction. Both studies administered the alpha-adrenergic block, phentolamine, and found a greatly reduced blood pressure response to an isometric handgrip contraction. Williams et al. (1981) indicated alpha-adrenergically mediated vasoconstriction could effectively prevent 15-20

ml/min per 100 ml of blood flow from reaching the active muscle. This indicated that vasoconstriction occurred within active muscle as well as within inactive tissues. They suggested that the small arteries and large arterioles supplying the muscle may therefore be affected by vasoconstriction (Williams et al., 1981). It has been suggested that this widespread vasoconstriction may have a functional role in increasing the cardiac output and improving muscle perfusion pressure during static exercise (Martin et al., 1974; Nutter and Wickliffe, 1981). The rise in systolic blood pressure with dynamic exercise is also associated with an increase in the cardiac output and vasoconstriction. However, the vasoconstrictory response is not as intense as with static exercise, affecting only the splanchnic, renal and hepatic blood vessels (Lind and McNicol, 1967). However, studies have shown that diastolic blood pressure is maintained during dynamic exercise (Tuttle and Horvath, 1957). In comparing the hemodynamic responses to static handgrip and dynamic cycling exercise, Tuttle and Horvath (1957) measured blood pressure immediately following each exercise bout. They observed no significant change in diastolic blood pressure with dynamic exercise and attributed this to the local vasodilation.

In examining the response to combining a static handgrip contraction with dynamic treadmill exercise, Lind and McNicol (1967) observed an equivalent rise in blood pressure to a static handgrip contraction of the same intensity performed alone. However, the systemic vascular resistance was lower during combined exercise thus, indicating that a larger cardiac output and/or greater vasoconstriction was required to elicit the same pressor response (Lind and McNicol, 1967). These results are in accordance with a recent study by Jetté et al. (1990) which examined the effect of combining static handgrip with dynamic supine cycling exercise on left ventricular function in myocardial infarct patients. A comparable increase in blood pressure to handgrip exercise alone, was found when handgrip was superimposed on three different intensities of dynamic exercise. Jetté et al.(1990) suggested that activation of the sympathetic adrenergic system associated with static contraction may override the systemic vasodilatory effect of dynamic exercise.

2.6.2 Systemic Vascular Resistance

A number of studies have indicated that systemic vascular resistance does not change significantly during static exercise in normal subjects (Lewis et al., 1984; Lind and McNicol, 1967; Asmussen, 1981). In addition, Colombo et al. (1989) comparing the cardiovascular response to static and dynamic exercise between normals and hypertensive subjects found systemic vascular resistance to be greater in both normals and hypertensives with static contraction due to a decrease in peripheral resistance during dynamic exercise. The maintenance or slight rise in peripheral resistance with static exercise was attributed to an increase in sympathetic stimulation of alpha-adrenergic receptors in the blood vessels. However, Asmussen (1981) suggested that peripheral resistance may be greater with static contractions due to the following: 1) peripheral vasodilation is not very effective in increasing blood supply to the active muscle during heavy static contractions; 2) mechanical pressure on the blood vessels will increase intramuscular pressure and add to the peripheral resistance, and 3) with increasing intensity of exercise there is a corresponding increase in anaerobiosis which may amplify the sympathetic vasoconstrictory response by producing an excess of metabolites. In contrast, a decrease in vascular resistance is observed during dynamic exercise (Asmussen, 1981). Lewis et al. (1984) indicated that vasodilation was metabolically mediated and increases with a larger active muscle mass and may override the alpha-adrenergic vasoconstriction. In addition, Martin et al. (1974) indicated that systemic vascular resistance may decrease due to 1) passive dilatation and 2) reflex vasodilation due to baroreceptor stimulation. Hannum and Kasch (1981) also noted a reduction in peripheral resistance with dynamic exercise and attributed it to an increased vasodilatation. They suggested that the vasodilatation was a consequence of thermoregulation, as well as in accordance with Kjellmer's (1965) study, a potassium influx increasing vascular volume.

2.6.3 Mean Arterial Pressure

The mean arterial blood pressure (MAP) is directly proportional to the product of the cardiac output and systemic vascular resistance (Asmussen, 1981). The marked increase in MAP with static exercise is due primarily to an increase in cardiac output and vasoconstriction with

little change in vascular resistance (Lewis et al., 1985; Asmussen, 1981). In contrast, the MAP does not change significantly with dynamic exercise (Asmussen, 1981; Temkin, 1983). Temkin (1983) suggested that MAP is maintained with dynamic exercise as a result of the large increase in cardiac output along with peripheral vasodilatation which decreases the systemic vascular resistance.

The rise in the systolic and diastolic blood pressure with static exercise are mediated primarily by sympathetic alpha-adrenergic stimulation resulting in systemic vasoconstriction and a rise in cardiac output. The maintenance of systemic vascular resistance during isometric exercise, has been attributed to the following factors: mechanical compression and increased intramuscular pressure occluding the blood vessels, ineffective vasodilation within the muscle and increasing anaerobiosis which enhances sympathetic vasoconstriction. The MAP rises during static exercise, reflecting the increases in systolic and diastolic blood pressure. Only systolic blood pressure rises during dynamic exercise, while the diastolic does not change. The maintenance of the diastolic blood pressure and the drop in systemic vascular resistance in dynamic exercise have been attributed to a greater vasodilatation within the active muscle and a less extensive systemic vasoconstriction. During combined static and dynamic exercise, a greater sympathetic vasoconstrictor response is required to attain a similar static pressor response as a result of the larger cardiac output attributed to dynamic exercise.

The following factors influence the overall blood pressure response: central command, peripheral reflex, muscle mass and baroreceptor activity (Asmussen, 1981). These will be dealt with primarily in relation to static contractions with appropriate references to dynamic and combined exercise.

2.7 Central Control

The control of the hemodynamic response to exercise is a balance between central and peripheral influences (Perez-Gonzales, 1981; Shepherd et al., 1981). Cortical irradiation and muscle fibre recruitment both contribute to the central drive component.

2.7.1 Cortical Irradiation

One of the earliest studies, examining the cardiovascular and respiratory responses to exercise was performed by Krogh and Lindhard (1913). Observing the rapid rise in heart rate and ventilation with the onset of exercise, they dismissed any possible peripheral reflex pathway. They proposed instead, that the rapid response must be a result of cortical irradiation (Krogh and Lindhard, 1913). Mitchell et al.(1981) depicted this cortical irradiation hypothesis as a central command which originates in the higher motor centres and sends signals to the medullary cardiovascular centre and directly to the exercising muscle (Figure 1). Seals, Washburn, Hanson, Painter and Nagle (1983) have related central command to the individual's perception of effort and therefore state that the central drive is under voluntary control. Mitchell et al. (1980) suggested that

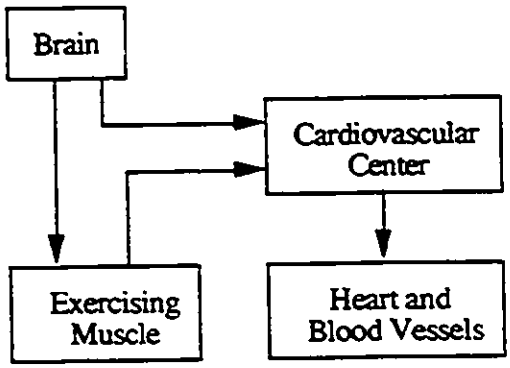

Figure I: Pressor Response To Static Exercise. (from Mitchell et al., 1981, p. I-75, by permission from the American Heart Association, Inc.)

the rapid elevation of heart rate and blood pressure at the onset of exercise indicates a neurogenic component and thus an influence of central drive. In addition, the later gradual rise of the hemodynamic response may also be attributed to the effect of central drive through an increase in fibre recruitment. Rowell, Freund and Hobbs (1981) suggest that the efferent signals from the central nervous system may descend down cortical and spinal tracts to form basic commands which may be regulated by the peripheral receptors. Studies using drugs to elicit selective autonomic blockade have shed light on the roles of the parasympathetic and sympathetic system in mediating central commands to the peripheral alpha and beta-adrenergic receptors (Goldstraw and Warren, 1985). Martin et al.(1974) gave evidence that the initial tachycardia response to exercise

was a result of vagal withdrawal due to a reduction of parasympathetic stimulation. The administration of propranolol, a beta-adrenergic receptor blockade indicated the importance of sympathetic stimulation as a secondary (delayed) mechanism to accelerate heart rate and initiate vasoconstriction during static exercise (Martin et al., 1974). Rusch et al. (1981) also examined the effect of the central system on the blood pressure response and vascular resistance during isometric exercise. Combining a mental stressor (mental arithmetic) with a static contraction resulted in a greater increase in heart rate and mean arterial blood pressure indicating the influence of the central nervous system (Rusch et al., 1981). Thus, there is evidence pointing to an influence arising from the central nervous system; however, Shepherd et al. (1981) indicated that the higher centres from which they originate remain unknown.

2.7.2 Muscle Fibre Recruitment

Mitchell et al. (1981) compared the blood pressure response to static contractions between a strong and weak leg in human subjects. A larger rise in systolic blood pressure and EMG activity was observed in the stronger leg when each leg contracted at the same percent of its respective MVC. However, the blood pressure response and EMG level were alike when each leg contracted at the same absolute force, thereby indicating that the activation of an equal number of motor units will result in the same blood pressure response (Mitchell et al., 1981). Petrofsky, Phillips and Lind (1981) examined muscle fibre type and order of recruitment in relation to the blood pressure response to exercise. Their observations indicated a lower blood pressure response when slow twitch fibres were predominantly activated, for example, at low exercise intensities (ie. 10% MVC) and in the early stages of a contraction before fast twitch fibres were recruited. Petrofsky et al. (1981) suggested that the lower blood pressure associated with slow twitch recruitment may be linked to differences in muscle metabolism or density of sensory receptors around the muscle fibres. In addition, Buck, Amundsen and Nielsen (1980) proposed that central drive may recruit accessory muscles to maintain a contraction after the onset of fatigue, thus contributing to the pressor response.

The command to exercise arises in the higher brain centers and is sent to the

periphery; however, evidence has indicated that a copy of the motor signal is also sent to the cardiovascular centre located in the brainstem. The rapid increase in heart rate with the onset of exercise has been attributed to vagal and parasympathetic withdrawal. Similarly increases in both heart rate and blood pressure throughout the duration of the exercise, is related to an increase in effort, sympathetic stimulation and muscle fibre recruitment to maintain the exercise intensity.

2.8 Peripheral Reflex

Petrofsky et al.(1981) have suggested that the overall circulatory response to exercise is related to an interaction between central irradiation and a peripheral reflex mechanism. Mitchell et al.(1981) indicated that afferent signals from the periphery act on the cardiovascular center to elicit the hemodynamic changes of the heart and blood vessels (Figure 1). The activation of peripheral receptors during muscle activity sends afferent signals to the central nervous system to elicit specific responses. Shepherd et al. (1981) have identified these peripheral factors as an increase in metabolites, the stimulation of muscle afferents and the release of potassium and lactate ions.

2.8.1 Metabolites

The study by Alam and Smirk (1937) illustrated that the rapid rise in blood pressure is reflexive in nature and is a result of the release of metabolites from contracting muscle into the bloodstream. In studying static contractions and high intensity dynamic exercise, Alam and Smirk (1937) found comparable increases in metabolites and sustained pressor response. The maintenance of the elevated blood pressure for both activities was attributed to the accumulation of metabolites within the muscle caused by blood flow occlusion (Alam and Smirk, 1937). Perez-Gonzales (1981) using blood flow occlusion before and after rhythmic and static contractions, ascertained that dynamic contractions elicited the same pressor response associated with static exercise. The decrease in blood flow was found to be the determining factor by causing an accumulation of metabolites which in turn evoked the pressor response (Perez-Gonzales, 1981). Thus, the accumulation of metabolites within muscle plays a role in maintaining the blood supply

through local vasodilation and stimulation of the central nervous system to increase perfusion pressure (Lind et al., 1964)

2.8.2 Muscle Afferents Type III And IV

Type III and IV(C) afferent fibres arising from receptors within the muscles have been studied to assess their involvement in eliciting the cardiopulmonary and respiratory responses. Type III are small, myelinated fibres and type IV are non-myelinated fibres. Type III afferent fibres arise predominantly from pressure/pain receptors; however, type III and IV have also been shown to react to chemical and mechanical stimuli (Kalia et al., 1981; Kniffki et al., 1981). Applying pressure to the tendon junction stimulated afferent fibres and resulted in a rise in blood pressure, heart rate and ventilation (Kalia et al., 1981). The response to exercise was compared to the response to squeezing the muscle while utilizing antidromic block (impeding signals from myelinated muscle afferents). Kalia et al. (1981) observed similar hemodynamic changes for both activities. However, when exercise was compared to muscle squeezing following procaine application, thereby, effectively blocking unmyelinated nerves, a greatly reduced ventilatory and cardiovascular response was reported. Kalia et al. (1981) therefore concluded that non-myelinated fibres play a role in evoking the cardiovascular response. Further research by Kniffki et al. (1981) has shown variation in sensitivity to stimuli within the type III and IV fibres. Thus, the fibres have been classified according to pressure as low threshold pressure units (LTP) and high threshold pressure units (HTP) with type III having approximately a 50:50 ratio, while type IV have a split of 70% HTP, 4% LTP and 25% inactivated by pressure (Kniffki et al., 1981). The HTP fibres are stimulated by firm pressure which causes pain and are believed to have a role in the physiological response to exercise. A number of group III and IV fibres are also differentially receptive to muscle contraction. The group IV fibres tend to be sensitive primarily to high intensity contractions while group III respond to a progressive increase in the force of contraction. However, there is another group of fibres containing a mix of both types which have been found to respond only to contraction during ischemic conditions. Kniffki et al. (1981) concluded, that although the research is not conclusive as to the specific afferents which initiate the cardiovascular reflex, a combination of type III and IV afferents are likely to play a

dominant role in the central drive component.

2.8.3 Potassium Ion And Lactate Release

Potassium and lactate are products of metabolism which are released into the interstitial space and diffuse into the bloodstream during static exercise. Saltin, Sjogaard, Gaffney and Rowell (1981) examined the relationship between the concentration of potassium (K+) and lactate (La) in the interstitial muscle space to heart rate and blood pressure changes with static exercise. Due to the impaired release of K+ and La, no significant changes in their concentrations within the interstitial space were observed during contraction. However, immediately following the contraction, a distinct increase was reported. A range from low to high concentrations of La and K+ were found when measured from different locations within the interstitial space of the muscle. This diversity in concentrations was attributed to variations in intramuscular pressure, blood flow and energy turnover within the muscle. Thus, Saltin et al. (1981) suggested that a measurement taken from the vein draining the muscle may be more indicative of the general muscle response. The concentration of La reached highest values only after heart rate and blood pressure had returned to resting values. Thus, indicating that no significant relationship between La and the hemodynamic response to static exercise was observed. In contrast, the rise in K+ tended to follow a similar pattern to the rise in blood pressure during and fall in blood pressure following exercise. Saltin et al. (1981) therefore, concluded that the potassium ion may be an important chemical factor in modifying the central control of the hemodynamic response to exercise. In contrast, to a possible effect on the central nervous system, Kjellmer (1965) found that the release of K+ into the bloodstream resulted in a vasodilatory effect on the local blood vessels.

Thus, muscular activity results in a reflex loop to the central nervous system, and specifically, the medullary cardiovascular centre to modulate the hemodynamic response. The release of metabolites from the active muscle into the blood stream causes local vasodilation as well as acts on the central nervous system to stimulate sympathetic vasoconstriction thereby improving blood perfusion pressure. Type III and IV afferent fibres arising from receptors within the muscle are activated during exercise and have been shown to affect the hemodynamic response to exercise.

In addition, the movement of potassium ions into the interstitial fluid may stimulate and activate small afferent fibres through the peripheral reflex loop as well as contribute to the local vasodilatory response.

2.9 Influence Of Muscle Mass And Intensity

Muscle mass and intensity of contraction have each been separately implicated as being the primary determinant of the magnitude of the pressor response (Mitchell et al., 1980; Quarry and Spodick, 1974).

2.9.1 Muscle Mass

Mitchell et al. (1980) examined the blood pressure response to static contractions of finger adduction, handgrip and knee extension in young, healthy male subjects. Their results indicated that with a larger muscle mass, a pronounced blood pressure response was elicited. This finding was attributed to a larger central drive as a result of a progressive recruitment of motor units. Also, the peripheral reflex drive was activated by the larger muscle mass and attributed to a greater quantity of metabolites acting upon peripheral receptors within the muscles (Mitchell et al., 1980; Mitchell et al. 1981). A similar study also comparing finger adduction and handgrip exercise at 40% MVC resulted in a greater pressor response with the larger muscle mass (Buck et al. 1980). Seals et al. (1983) and Nagle, Seals and Hanson (1988) performing similar studies compared the cardiovascular responses among handgrip, leg extension and dead lift each at 30% MVC. Both studies showed a progressive rise in blood pressure and heart rate with an increase in muscle mass, in agreement with the earlier findings of Mitchell et al. (1980 and 1981). Recently, Misner et al. (1990) performed a similar investigation of finger flexion, leg extension and double leg extension at MVC. Both the heart rate and blood pressure responses demonstrated a positive relationship with increasing muscle mass. However, Misner et al. (1990) noted that the relationship between muscle mass and heart rate decreased during prolonged static exercise at MVC. Thus, they suggested that heart rate and blood pressure during static exercise may have different control mechanisms.

2.9.2 Intensity

Lind and McNicol (1967) examined the blood pressure response to simultaneous contractions of more than one muscle group performed at the same intensity, and then repeated the measurements with muscle groups contracting simultaneously at different intensities. They observed that contractions performed at the same percent MVC did not result in an addition of the pressor responses of each individual contraction. When two muscle groups contracted at different intensities, the pressure response was similar to the response of the higher tension alone. Thus, Lind and McNicol (1967) concluded that muscle mass did not appear to play a role in the pressure response and that the relative intensity of the contraction was likely the determining factor. Quarry and Spodick (1974) compared the blood pressure response to handgrip exercise at four different intensities (15%, 30%, 50% and MVC). Increases in systolic and diastolic blood pressures were observed with a progressive rise in contraction intensity. Riendl et al. (1977) performed a similar study examining the hemodynamic responses to finger adduction versus ankle plantar flexion. The results are in agreement with the earlier findings of Quarry and Spodick (1974) which suggest that muscle mass has no effect on the systolic blood pressure response. However, Riendl et al.(1977) noted a larger diastolic blood pressure response with a smaller muscle mass. This difference may be due to a variance in the pressor response to arm and leg contraction (Astrand et al., 1965). A relationship between the intensity of contraction and the magnitude of the pressor response was found in a number of studies (Seals, 1989; Perez-Gonzalez, 1981; Goldberg et al. 1982). Asmussen (1981) indicated that the percent MVC was related to the magnitude of the heart rate response rather than muscle mass. In addition, Asmussen (1981) proposed that the pressor response was related to a combination of muscle mass affecting mechanical resistance and intramuscular pressure, while the intensity of the exercise corresponded to the reflex vasoconstriction and central drive in relation to the voluntary effort. Asmussen's (1981) results support the earlier findings of McCloskey and Streatfeild (1975), which suggested that the magnitude of the pressor response corresponded to the intensity of the exercise and that the active muscle mass affects the peripheral muscular reflex in relation to blood flow occlusion.

2.9.3 Arm Versus Leg Exercise

Studies by Astrand et al. (1965) and Mitchell et al. (1979) have alluded to a difference in the pressor response between arm and leg static contractions. Significantly higher blood pressure responses were noted with arm exercise when exercise of similar oxygen uptake and cardiac output was performed with combining both arms and legs (Astrand et al., 1965). Due to equivalent cardiac outputs, the difference in blood pressure was attributed to a difference in the peripheral vascular resistance. There is a greater peripheral vascular resistance during arm exercise due to vasoconstriction in the legs, whereas with leg exercise the peripheral resistance is not as significant due to vasoconstriction within a smaller muscle mass (Astrand et al., 1965). Conversely, Asmussen (1981) in comparing the cardiovascular response to the size of the contracting muscle suggested that since large muscles may have fewer motor nerves due to larger motor units there may also be fewer afferent nerve fibres. Thus, at a similar contraction intensity, Asmussen (1981) proposed that the afferent inflow to the central nervous system may be the same irrespective of the size of the muscle. In addition, Mitchell et al. (1980) found a higher blood pressure and heart rate response with leg exercise in comparison to arm exercise. In contrast, they attributed this difference to the size of the muscle mass.

In conclusion, the hemodynamic response to exercise is related to a combination of the active muscle mass and the intensity of the contraction. It has been suggested that heart rate and blood pressure have different control mechanisms, with heart rate tending to be influenced primarily by the intensity of the activity. The intensity of the exercise is closely related to the central nervous system influence on the pressor response, whereas muscle mass is linked with the peripheral reflex component. Differences observed in the pressor response between leg and arm activity have been related to a number of possible factors including: extent of vasoconstriction, density of peripheral receptors and amount of active muscle mass.

2.10 Baroreceptor Influence

Carotid and aortic baroreceptors are stretch receptors located within the respective

arterial walls. An increase in arterial pressure stretches the wall of the artery, thus stimulating afferent signals from the baroreceptor. Increased activity of the baroreceptor stimulates the parasympathetic system to decrease heart rate and the alpha-adrenergic receptors to increase peripheral vasodilation in order to reduce the blood pressure (McArdle et al., 1986; Smith et al., 1988). Studies assessing the role of the aortic and carotid baroreceptors have presented conflicting results indicating a normally activated versus a depressed response during static exercise.

2.10.1 Normal Activation Versus Depressed Response

Nutter and Wickliffe (1981) elicited a vasoconstrictive response in the blood vessels supplying the skin, inactive tissue, kidney, gut and coronary circulation in vagotomized dogs by direct stimulation of muscle afferent fibres. However, vasoconstriction in the active muscle was blunted and vasodilation in the skin surrounding the muscle was observed and attributed to the effect of the carotid baroreflex. Similar results were noted by Abboud, Mark and Thames (1981) who assessed the effect of baroreceptor activity on the peripheral reflex in humans. Forearm vascular resistance was compared during: lower body negative pressure (LBNP), isometric handgrip contractions of 10 and 20% MVC at rest and then repeated during LBNP. They observed small increases in forearm vascular resistance during LBNP alone and at 20% MVC handgrip contraction at rest. However, the largest increase in forearm vascular resistance was found when LBNP was combined with handgrip contraction. Abboud et al. (1981) concluded that during LBNP, the carotid baroreceptor activity was decreased, thus enhancing the vasoconstrictive response to isometric exercise. Conversely, Cunningham, Petersen, Peto, Pickering and Sleight (1972) suggested that baroreceptor sensitivity decreased during static exercise. They attributed this decrease in baroreceptor sensitivity, to the significant increase in sympathetic outflow which resulted in an uninhibited increase in heart rate and blood pressure. However, further research by Gonyea et al. (1981) indicate that a withdrawal of vagal tone is responsible for the elevated heart rate response and that baroreceptor activity is uninhibited during isometric exercise. In conclusion, Lewis et al. (1985) and Abboud et al. (1981) suggest that the baroreflexes modify the heart rate and blood pressure response to static exercise.

Studies utilizing lower body negative pressure have inhibited baroreceptor activity, thereby resulting in an enhanced sympathetic activation. Thus, these studies have provided evidence that baroreceptor activity is uninhibited and has a modifying effect on the hemodynamic response to isometric exercise.

2.11 Compensatory Measures

Studies have shown that although the mechanisms which elicit the normal pressor response to isometric exercise may be hindered, the pressor response is still evoked (Perez-Gonzales, 1981). Thus, in disease states such as coronary heart disease and hypertension, the normal aging process or as a result of the unique adaptation associated with training the pressure response is elicited by other compensatory mechanisms.

2.11.1 Cardiovascular Disease And Hypertension

Haskell et al. (1981) examined the rise in blood pressure and heart rate in cardiac transplant patients during isometric exercise. In patients without central innervation, no increase in heart rate or cardiac output were observed; however, a normal pressor response was elicited. Thus, the rise in blood pressure was a result of an increase in systemic vascular resistance and sympathetically mediated vasoconstriction (Haskell et al., 1981). A similar response to static exercise was seen in the study by Jetté et al. (1990) in patients with heart disease and left ventricular dysfunction. Jetté et al. (1990) noted that in normals, static exercise elicits an increase in heart rate and myocardial contractility through sympathetic stimulation. However, with heart disease, the impaired ventricular function prevents an increase in contractility or any significant contribution from the Frank-Starling mechanism. These results confirm the findings of Krayenbuehl et al. (1981) who examined the pressure load on the left ventricle during handgrip exercise in aortic valve disease patients. Krayenbuehl et al. (1981) noted that handgrip exercise produced a significant pressure load on the heart primarily as a result of an enhanced vasoconstrictive response. Perez-Gonzalez (1981) noted that despite various "obstructions" to the normal response to exercise, for example, sympathetic blockade and impaired contractile reserve,

the pressor response is still evoked by readjustments of the other mechanisms; ie. systemic vasoconstriction, muscle vasodilatation and an increase in peripheral vascular resistance.

Helfant, DeVilla and Meister (1971) and Stefadourous (1983) suggested that isometric exercise may be an effective method of assessing abnormal cardiac function as a result of the different mechanisms responsible for initiating the pressor response. The simplicity of the handgrip test along with its safety and speed with which the test can be administered to determine abnormal ventricular function was indicated in the study by Helfant et al. (1974). Stefadourous (1983) suggested that isometric exercise can be used for diagnostic purposes to detect left ventricular dysfunction, as a stress test for coronary heart disease patients who are unable to perform dynamic activity, and lastly to determine the source of certain heart murmurs.

Colombo et al. (1989) examined the hemodynamic response to three different stressors (static handgrip contraction, dynamic bicycle exercise and tyramine infusion) in normotensive and hypertensive subjects. The increases in systolic blood pressure and heart rate were similar in both normotensive and hypertensive subjects. However, the diastolic blood pressure increased during dynamic exercise in hypertensives, whereas in normals, there was a decrease. The rise in diastolic blood pressure was attributed to an increased sensitivity of the peripheral alpha-adrenergic receptors to sympathetic stimulation (Colombo et al., 1989). Tyramine infusion which causes the secretion of plasma norepinephrine, was utilized to assess the reactivity to sympathetic stimulation. The results of tyramine infusion illustrated a significantly greater systolic blood pressure increase in hypertensive subjects than normotensive thus indicating an increased sympathetic reactivity in the hypertensives (Colombo et al.,1989).

2.11.2 Effect Of Age

Kino, Lance, Shahamatpour and Spodick (1975) compared the hemodynamic response to MVC handgrip exercise in subjects ranging from 23-31 yrs. with subjects 54-78 yrs. They observed a significantly lower heart rate response in the older subjects in comparison to the younger subjects. The lower heart rate and depressed myocardial contractility was attributed to a

decrease in sympathetic tone with advancing age (Kino et al. 1975). Baldwa, Sharma, Bhansali and Gupta (1983) also noted a significantly lower heart rate response to isometric handgrip exercise in subjects older than 50 yrs versus those younger than 50 years. However, much greater increases in systolic and diastolic blood pressures and the rate pressure product were evident in the older subjects. Baldwa et al. (1983) suggested that a progressive increase in total peripheral resistance with age may account for the higher blood pressure response observed in the older participants. Petrofsky and Lind (1975) also examined the effect of age on isometric exercise and noted that systolic blood pressures at rest and during 40% MVC contraction were higher in elderly subjects. They also attributed the higher blood pressure to increased peripheral resistance as a consequent to a decrease in arterial compliance and flexibility. Conversely, Goldstraw and Warren (1985) although observing significant differences in heart rate, systolic and diastolic blood pressure between young and old subjects at rest and during static exercise attributed these differences to a greater variability of response in the elderly. Since the systolic and diastolic blood pressures were significantly higher both at rest and during static contraction, it was suggested that the increment was equal. Goldstraw and Warren (1985) concluded that although there is an increase in blood pressure with age, the pattern of the response to isometric exercise did not change.

2.11.3 Effect of Training

Longhurst, Kelly, Gonyea and Mitchell (1981) compared the effects of isometric training and dynamic training on the hemodynamic response to static handgrip exercise. Following training a lower heart rate and myocardial oxygen demand was observed in the dynamic (endurance) trained subjects in comparison to the isometric (weight) trained and control (untrained) subjects. This difference was attributed to the much larger end-diastolic and end-systolic volumes which commonly accompany the structural changes to the heart with endurance training. In contrast, no significant difference were found with static exercise training since the weight-lifters responses were similar to the control group at rest and during exercise (Longhurst et al., 1981). An earlier study by this same group, Longhurst, Kelly, Gonyea and Mitchell (1980) found similar results when examining the hemodynamic responses to an isometric exercise between weight

trained and endurance trained athletes. An increased vascular resistance was noted in the endurance trained athletes and attributed to the lower heart rate at rest and during exercise which were thought to limit the increase in cardiac output during exercise. Takeshita, Jingu, Imaizumi, Kunihiko, Koyanagi and Nakamura (1986) similarly found a lower heart rate response and greater vascular resistance in trained subjects compared to controls. However, they suggested these differences were due to training, enhancing the inhibitory influence of the cardiopulmonary receptors. The increased cardiopulmonary receptor reflex was said to contribute to an increase in sympathetic nervous activity which has also been observed in trained athletes. Takeshita et al. (1986) therefore concluded, that the increased vascular resistance seen in trained athletes could be attributed to this altered sympathetic outflow due to training adaptations. Other studies have supported the findings that heart rate and blood pressure were lower in dynamically trained subjects during isometric exercise in comparison to untrained individuals as a result of an increase heart volume (Maiorano et al., 1989; Keul et al., 1981).

Thus, structural changes to the heart and cardiovascular system with disease, age or training, do not prevent the pressor response from occurring during isometric exercise. In coronary heart disease, there is an exaggerated sympathetically mediated vasoconstriction, enhancing the systemic vascular resistance, in order to compensate for the decrease in myocardial contractility. Conversely, the increase in sympathetic vasoconstriction in patients who suffer from hypertension is due to an increase in the sensitivity of the alpha adrenergic receptors. With age, there is a decrease in the contractility and compliance of the heart and blood vessels, as well as a decrease in sympathetic tone. Thus, similar to heart disease, the pressor response to static exercise is initiated by vasoconstriction. A difference in the mechanisms governing the pressor response are primarily found with dynamic exercise training. An enhanced sympathetic activation occurs to increase the cardiac output in the presence of an enlarged heart volume and slow heart rate.

2.12 Summary

The hemodynamic control mechanisms are similar for static and dynamic exercise, however the events that take place in the periphery govern the extent of the response. During static

exercise, an ischemic response occurs due to constant muscular contraction and compression of blood vessels within the muscle. The peripheral reflex acts on the cardiovascular centre to cause vasoconstriction which results in the pressor response increasing perfusion pressure. Conversely, during dynamic exercise, the rise in systolic blood pressure is primarily a consequent of an increase in cardiac output. Ischemia does not occur within the muscle as a result of the relaxation phases of dynamic exercise, and therefore vasoconstriction is not initiated by the cardiovascular centre. However, marked vasodilation does occur within the active muscle which decreases the peripheral vascular resistance and diastolic blood pressure during dynamic exercise. Therefore, when a static contraction is superimposed on dynamic exercise, a greater sympathetic activation is required to elicit the pressor response. Thus, the pressor response is elicited during isometric exercise primarily to increase the perfusion pressure to the active muscle regardless of any inhibitory influences.

III

METHODOLOGY

3.1 Introduction

The purpose of this study was to determine whether the magnitude of the pressor response is blunted when an isometric contraction is superimposed on dynamic exercise. This was accomplished through the continuous, noninvasive measurement of heart rate and blood pressure.

3.2 Subjects

Twenty moderately active, male volunteers (24 to 32 yrs old) participated in this study. A modified PAR-Q (appendix A) was utilized to determine the health status of the participants. Subjects selected for this study included those who met the following criteria:

1. male, age 24 to 32 years
2. answered "no" to all questions on the modified PAR-Q
3. non-smoker
4. a resting blood pressure lower than 140/90 mm Hg.
5. not taking prescribed medication which may alter cardiovascular response or physical performance.
6. normotensive blood pressure response to a moderate level (stage A of CAFT) of exercise.

All subjects gave informed written consent to participate in the study prior to testing (Appendix B).

3.3 Testing Protocol

The testing protocol was conducted over two days.

DAY 1

Upon entering the laboratory, volunteers were briefed as to the nature of the testing and their involvement in the study. Subjects were requested to complete a modified PAR-Q (Appendix A), an informed written consent (Appendix B) prior to participation and a health questionnaire (Appendix C). They were then seated for a five minute period. The resting heart rate and blood pressure measurements were recorded using a standard stethoscope and sphygmomanometer at the end of the rest period, to ensure subjects were normotensive.

Subjects then performed the modified Canadian Aerobic Fitness Test (CAFT)(Jetté, Landry and Sidney, in press). The initial stage performed by the subjects was determined according to their gender and age. Each stage consisted of three minutes of stepping up and down two steps (20.3 cm high) to a six beat musical rhythm. With each successive stage there was an increase in the stepping rhythm. Heart rate and blood pressure were measured using a sphygmomanometer and stethoscope at rest, prior to commencing the test and following each stage of the CAFT. Immediately after each exercise stage, heart rate was recorded between the 5th and 15th second; systolic and diastolic (D5) blood pressure were measured between the 15th and 45th second. If the heart rate did not exceed a predetermined limit following the first two stages and no detrimental symptoms were observed, the participant was permitted to continue to the second and third stage of the CAFT. The blood pressure response to the first stage was utilized to screen subjects for an exaggerated blood pressure response (sbp measurement >150 mmHg) to dynamic exercise (Jetté et al., in press). The results of the last stage completed by the subject were used to calculate the predicted maximum aerobic capacity (max VO_2) using the procedure of Jetté (Appendix D). These calculations were performed to compare the 100 bpm exercise heart rate to a corresponding percent of the max VO_2.

The subject's age, height, weight and girths (chest waist and hip) were then measured (Jetté, 1983). From these measurements the following were later calculated: body mass index (BMI), chest minus waist (CMW) and the waist to hip ratio (WHR). These anthropometric measurements along with the results of the CAFT were evaluated to determine whether subjects represented a normal sample of the population.

The subjects were instructed as to the proper handgrip technique using the Martin Vigorometer. To prevent subjects from performing the Valsalva manoeuvre, they were instructed to exhale during the handgrip contraction . All handgrip contractions were performed in the upright, standing position. Subjects were given several trials to familiarize themselves with the dynamometer. Three maximum voluntary handgrip contractions (MVC) of the left hand were performed with a two-minute recovery between each trial. The maximum of the three consecutive contractions was used to determine the MVC.

Subjects then practised maintaining handgrip contractions at 30% and 40% MVC for one minute durations while continuous heart rate and blood pressure changes were recorded using the Finapres monitor (Hartmann and Bassenge, 1989). This was done to allow subjects to become familiar with the technique of maintaining the handgrip contraction and to become accustomed to the Finapres monitor.

Next, subjects were given a demonstration as to the proper walking technique on the Quinton Treadmill Model 643 (Quinton Instruments, Seattle, Washington). Subjects walked at 0% slope with progressive increases in treadmill speed until their exercising heart rate stabilized at 100 beats per minute, at which time the speed of the treadmill and their stepping rate per minute were recorded. Subjects then, practised walking while squeezing the Martin Vigorometer at 30% and 40% MVC.

DAY 2

The subjects, upon entering the laboratory, had their resting heart rate and blood pressure measurements recorded twice: after five minutes in the seated position and then following one minute standing. The standing heart rate and blood pressure measurements were utilized for comparison with the responses to the handgrip tests which were performed also in the upright position.

Subjects were then randomly divided into two equal groups and instructed that group A would perform all exercises initially utilizing a handgrip contraction of 30% MVC

followed by 40% MVC while group B would perform handgrip contractions initially at 40% MVC followed by 30% MVC. Thus, the protocol for group A is outlined below with group B simply performing the exercises in the opposite order. The subjects then performed a handgrip contraction at 30% MVC for one minute while in the standing position. After a five minute rest, the handgrip contraction was repeated at 40% MVC for one minute. Heart rate and blood pressure measurements were recorded at two second intervals during each handgrip contraction and 15 seconds into the recovery.

Next, subjects performed dynamic exercise on the treadmill at the predetermined speed (corresponding to 100 bpm). At the start of the fourth and last minute of walking exercise, a handgrip contraction of 30% MVC was performed and maintained for a one minute duration. Subjects stopped both exercises at the end of this minute. One minute following the cessation of exercise, the subjects repeated only the static handgrip contraction at 30% MVC for an additional minute. Heart rate and blood pressures were recorded continuously two second intervals from the start of the third minute of walking exercise until the end of the last static contraction and 15 seconds into the recovery. This procedure was then repeated using a handgrip contraction of 40% MVC.

3.5 Data Analysis

A descriptive analysis of the group will include the following means: age, height, weight, BMI and girth measures (CMW and WHR).

The cardiovascular response to handgrip contraction was compared according to the three times taken: alone (at rest) vs. during the fourth minute of dynamic exercise vs. one minute following dynamic exercise. A 3x2 factorial design analysis of variance (ANOVA) with repeated measures on two factors was used to determine significant differences between the means. The independent variables (IV) included two levels of intensity (30% and 40% MVC) and the 3 static exercise bouts (rest, during dynamic exercise and one minute following) and the dependent variables (DV) were heart rate, systolic and diastolic blood pressure. Significant differences were

assessed at an alpha level of < 0.05 for significance. When a significant difference was found, the Least Means square analysis was utilized to determine specifically between which means the difference existed.

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Appendix A

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 ofmedication, whether are prescribed or "over the counter"? If yes, please specify:	Yes	No

Reason: _____

Name: _____

Dosage: _____

Signature: _____

Date: _____

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

Appendix B

DEPARTMENT OF HUMAN KINETICS THESIS RESEARCH CONSENT FORM

DEPARTMENT OF HUMAN KINETICS THESIS RESEARCH CONSENT FORM

Whenever a study involves human subjects, the Ethics Committee of the University of Ottawa requires written consent of the participants. I, _____, authorize Jackie Vanderluit (230-1611) of the School of Human Kinetics, University of Ottawa, to administer and conduct the following fitness test and dynamic and static exercises. This study will be conducted under the supervision of Dr. M. Jetté, School of Human Kinetics (564-9108).

The purpose of this study is to determine whether dynamic exercise has a blunting effect on the pressor response to static exercise, during combined dynamic and static exercise in normotensive male volunteers age 24 to 35 years.

I understand that the testing will be conducted over two consecutive days. On the first day, prior to testing I will complete the modified Physical Activity Readiness Questionnaire (PAR-Q). My resting heart rate and blood pressure, height, weight and girths (chest, waist and hips) will be measured before I participate in the exercise tests.

I will step up and down on double 20.3 cm. steps for three stages (4, 5 and 6) of the Canadian Aerobic Fitness Test. My blood pressure and heart rate will be measured before commencing the test, in the one minute interval between each stage and during the three-minute recovery following exercise. I understand I will step up and down for three minutes during each stage and remain standing for the one minute interval between stages. I will continue with the following tests if my blood pressure at exercise does not exceed a predetermined limit.

Next, I will be given twenty (20) minutes to familiarize myself with the different apparatus: handgrip dynamometer and treadmill that I will be using in the exercise protocol. Following this I will perform three consecutive maximum handgrip contractions with my dominant hand in order to determine my maximum voluntary contraction (MVC). I will then proceed to walking on the treadmill (at 0% grade) to determine the speed at which my heart rate stabilizes at 100 beats per minute.

Following these preliminary tests, I will be requested to maintain a static handgrip contraction with my dominant hand at 30% MVC for one minute while continuous heart rate and blood pressure measurements will be recorded using the Finapres monitor.

On the second day of testing, I will perform ten minutes of walking on the treadmill at the speed at which my heart rate is 100 bpm. At the beginning of the fourth minute of exercise I will perform a static handgrip contraction at 30% MVC for one minute while continuing to walk on the treadmill. After one minute of recovery, I will repeat the static handgrip contraction at 30% MVC for another full minute. My heart rate and blood pressure will be measured continuously from the start of walking on the treadmill until the cessation of my second handgrip contraction.

The testing will be performed in such a way as to minimize any discomfort and risk. I understand that I may experience some light muscular fatigue. I understand that the testing personnel are assuming that I am a healthy subject who does not suffer from any chronic medical problems. I further understand that it is my responsibility to inform the testing personnel of any injury, illness, infection, or other condition, which would prevent me from fully participating in this session.

I understand all information collected will be kept confidential and recorded in an anonymous form. In agreeing to such an examination, I waive any legal recourse against the testing personnel and staff of the department of Human Kinetics from any and all claims resulting from personal injuries sustained resulting from these tests. This waiver shall be binding upon my heirs and personal representatives.

I understand that I have the right to withdraw from this study at any time.

DATE: _____ SUBJECT: _____

WITNESS: _____

Appendix C

HEALTH QUESTIONNAIRE

It would be greatly appreciated if you would answer the following questions by checking the appropriate response: :

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

2. If you are a smoker: cigarettes: no./day ___
   pipe: bowls/day ___
   cigars: no./day ___

3. If you are an ex smoker, please indicate the number ofyears as:
   an ex smoker: _____ smoker: _____

4. Which 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

   Nota:
   one drink = beer: 12-oz bottle/can
   wine: 4-oz
   liquor: 1 1/2 oz

Appendix D

DAY ONE & TWO TESTING SHEETS

School of Human Kinetics

University of Ottawa

Day One Testing Protocol

Name: _____ Date: _____

Age: _____yrs. Time: _____

Temp: _____

Anthropometric Measurements

Height: _____cm. Weight: _____kg.

Chest: _____cm. Waist: _____cm.

Hip: _____cm.

Resting Measurements

RHR: _____bpm. RSBP: _____mmHg. RDBP: _____mmHg.

CAFT: Stage 4		Stage 5		Stage 6	
HR:	_____bpm.	HR:	_____bpm.	HR.	_____bpm.
SBP:	_____mmHg.	SBP:	_____mmHg.	SBP:	_____mmHg.
DBP:	_____mmHg.	DBP:	_____mmHg.	DBP:	_____mmHg.

MVC

Trial 1: _____ MVC: _____

Trial 2: _____ 30% MVC: _____

Trial 3: _____ 40% MVC: _____

Treadmill (120 bpm) _____mV

_____steps/min

School of Human Kinetics
University of Ottawa

Day Two Testing Protocol

Name: _____ Date: _____
Age: _____yrs. Time: _____
Temp: _____

Resting Values: Seated
RHR: _____bpm.
RSBP: _____mmHg.
RDBP: _____mmHg.

Standing
RHR: _____bpm.
RSBP: _____mmHg.
RDBP: _____mmHg.

30% MVC
HG at Rest
HR: _____bpm.
SBP: _____mmHg.
DBP: _____mmHg.

HG with Dynamic Exercise
HR: _____bpm.
SBP: _____mmHg.
DBP: _____mmHg.

HG post Dynamic Exercise
HR: _____bpm.
SBP: _____mmHg.
DBP: _____mmHg.

40% MVC
HG at Rest
HR: _____bpm.
SBP: _____mmHg.
DBP: _____mmHg.

HG with Dynamic Exercise
HR: _____bpm.
SBP: _____mmHg.
DBP: _____mmHg.

HG post Dynamic Exercise
HR: _____bpm.
SBP: _____mmHg.
DBP: _____mmHg

Appendix E

PERMISSION FOR COPYRIGHTED MATERIAL

May 26, 1991 88

The American Heart Association,
7320 Grenville Ave.,
Dallas, TX 75231

To whom it may concern,

I am writing a thesis on the effect of dynamic exercise on the blood pressure response to isometric exercise in normotensive males for my Masters degree in Human Kinetics at the University of Ottawa. I would like to use a figure from an article in Circulation Research in my review of literature. I am referring specifically to figure 12 on page I-75 from the article Response of Arterial Blood Pressure to Static Exercise in Relation to Muscle Mass, Force Development, and Electromyographic Activity" by Mitchell et al. (1981) in Circulation Research 48 (6). This figure would be used in the review of literature section of my thesis and not for publication in any journal. I am requesting permission to reproduce this figure for my thesis.

Thank you for your attention to this matter.

Sincerely,

Circulation Research 1981; 48:I-75

J. Vanderluit

Jackie Vanderluit
R.R. # 1
Hampton, ON
Canada L0B 1J0

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

PREDICTED MAXIMUM AEROBIC CAPACITY EQUATION

Predicted Maximum Aerobic Capacity Equations

Stage Completed

5th MaxV02 = 42.5 + [16.6 * 2.0066] - [0.12 * weight (kg)] - [0.12 * HR] - [0.24 * Age]

6th MaxV02 = 42.5 + [16.6 * 2.3453] - [0.12 * weight (kg)] - [0.12 * HR] - [0.24 * Age]

7th MaxV02 = 42.5 + [16.6 * 2.7657] - [0.12 * weight (kg)] - [0.12 * HR] - [0.24 * Age]

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

Appendix G

RAW DATA & STATISTICAL ANALYSIS

	SBP						Input Column
	Low Intensity			High Intensity			
	R/HG1	W/WHG	RC/HG2	R/HG1	W/WHG	RC/HG2	
1	47.24	34.63	7.01	30.68	22.58	26.00	
2	7.49	23.99	13.47	23.30	21.90	7.47	
3	-5.64	9.93	7.03	4.70	19.33	9.00	
4	13.14	9.77	-1.03	21.24	14.07	7.53	
5	12.27	-13.73	-4.33	10.93	13.96	15.06	
6	14.76	3.27	-9.33	23.82	12.27	2.30	
7	14.67	-1.10	-2.20	9.63	.37	6.20	
8	3.07	16.67	2.90	10.10	40.50	8.77	
9	21.43	12.10	3.76	13.67	9.40	5.74	
10	17.96	6.23	-6.62	18.76	14.76	-1.47	
11	23.63	12.05	20.71	22.13	17.27	20.40	
12	22.81	3.57	.30	22.53	11.70	15.80	
13	24.09	17.10	3.93	30.45	16.83	9.40	
14	24.93	11.27	13.50	19.23	13.47	21.04	
15	8.60	4.03	23.66	6.66	-2.57	24.03	
16	16.33	13.00	21.50	12.41	17.60	31.50	
17	26.76	9.40	5.20	19.16	18.64	9.50	
18	-2.20	5.77	5.40	11.79	11.17	13.37	
19	8.90	-8.68	8.97	18.46	13.00	22.83	
20	23.00	13.24	3.21	25.12	21.43	23.44	

	SL?						Input Column
	Low Intensity			High Intensity			
	R/HG1	W/WHG	RC/HG2	R/HG1	W/WHG	RC/HG2	
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 Fl...	Free Format Fl...	Free Format Fl...	Free Format Fl...	Free Format Fl...	Free Format Fi...
Dec. Places:	2	2	2	2	2	2	3
Mean:	16.16	9.13	5.85	17.75	15.39	13.90	.
Std. Deviation:	11.68	10.52	9.09	7.47	8.70	8.87	.
Std. Error:	2.61	2.35	2.03	1.67	1.94	1.98	.
Variance:	136.48	110.62	82.57	55.82	75.66	78.77	.
Coeff. of Variation:	72.28	115.25	155.28	42.09	56.53	63.87	.
Minimum:	-5.64	-13.73	-9.33	4.70	-2.57	-1.47	.
Maximum:	47.24	34.63	23.66	30.88	40.50	31.50	.
Range:	52.88	48.36	32.99	26.18	43.07	32.97	.
Count:	20	20	20	20	20	20	.
Missing Cells:	0	0	0	0	0	0	.
Sum:	323.24	182.51	117.04	354.97	307.76	277.91	.
Sum of Squares:	7817.32	3767.25	2253.77	7360.79	6173.29	5358.24	.

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Subject	19	3776.850	198.782		
Intensity	1	841.905	841.905	18.196	.0004
Intensity * Subject	19	879.102	46.269		
Change	2	1038.696	519.348	4.416	.0189
Change * Subject	38	4469.208	117.611		
Intensity * Change	2	222.433	111.216	3.730	.0332
Intensity * Chang...	38	1133.092	29.818		

Dependent: SBP

Table of Epsilon Factors for df Adjustment
Dependent: SBP

	G-G Epsilon	H-F Epsilon
Intensity	1.000	1.000
Change	.960	1.066
Intensity * Change	.938	1.038

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

Least Squares Means Table
Effect: Change
Dependent: SBP

	Vs.	Diff.	Std. Error	t-Test	P-Value
R/HG1	W/WHG	4.698	2.425	1.938	.0601
	RC/HG2	7.081	2.425	2.920	.0059
W/WHG	RC/HG2	2.383	2.425	.983	.3320

Least Squares Means Table
Effect: Intensity * Change
Dependent: SBP

	Vs.	Diff.	Std. Error	t-Test	P-Value
Low Intensity, R/HG1	Low Intensity, W/WHG	7.036	1.727	4.075	.0002
	Low Intensity, RC/HG2	10.310	1.727	5.971	.0001
	High Intensity, R/HG1	-1.587	1.727	-.919	.3640
	High Intensity, W/WHG	.774	1.727	.448	.6565
	High Intensity, RC/HG2	2.267	1.727	1.313	.1972
Low Intensity, W/WHG	Low Intensity, RC/HG2	3.274	1.727	1.896	.0656
	High Intensity, R/HG1	-8.623	1.727	-4.994	.0001
	High Intensity, W/WHG	-6.262	1.727	-3.627	.0008
	High Intensity, RC/HG2	-4.770	1.727	-2.762	.0088
Low Intensity, RC/HG2	High Intensity, R/HG1	-11.897	1.727	-6.889	.0001
	High Intensity, W/WHG	-9.536	1.727	-5.522	.0001
	High Intensity, RC/HG2	-8.043	1.727	-4.658	.0001
High Intensity, R/HG1	High Intensity, W/WHG	2.361	1.727	1.367	.1797
	High Intensity, RC/HG2	3.853	1.727	2.231	.0316
High Intensity, W/WHG	High Intensity, RC/HG2	1.493	1.727	.864	.3928

	DBP						Input Column
	Low Intensity			High Intensity			
	R/HG1	W/WHG	RC/HG2	R/HG1	W/WHG	RC/HG2	
1	10.10	40.46	37.72	24.84	29.31	27.87	
2	6.49	9.14	20.17	14.46	12.61	18.54	
3	6.33	6.67	16.46	9.50	6.60	16.30	
4	9.03	6.90	8.83	14.00	12.86	13.70	
5	11.64	4.40	10.10	10.87	6.37	16.57	
6	2.00	3.67	3.77	6.68	6.00	9.46	
7	6.40	4.80	7.07	10.83	2.86	12.40	
8	9.90	6.28	11.43	9.94	17.71	13.23	
9	10.47	3.30	12.37	10.67	9.00	11.60	
10	5.30	6.66	7.28	9.54	8.57	11.67	
11	17.00	9.02	19.97	14.27	16.64	17.50	
12	15.46	3.44	12.70	12.92	9.47	17.43	
13	15.51	9.00	13.97	19.85	7.20	19.47	
14	16.46	11.13	12.87	6.70	11.53	18.47	
15	5.53	.41	20.40	10.40	4.17	23.30	
16	8.43	6.30	20.23	8.07	12.33	29.80	
17	21.00	5.50	6.64	7.40	15.10	9.20	
18	4.94	5.87	12.07	18.18	17.05	22.87	
19	8.60	.46	16.03	17.14	12.05	19.18	
20	20.81	15.23	11.42	29.92	18.74	25.10	

	DBP						Input Column
	Low Intensity			High Intensity			
	R/HG1	W/WHG	RC/HG2	R/HG1	W/WHG	RC/HG2	
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 Fl...	Free Format Fl...	Free Format Fl...	Free Format Fl...	Free Format Fl...	Free Format Fl...
Dec. Places:	2	2	2	2	2	2	3
Mean:	10.52	7.93	14.07	13.31	11.81	17.68	.
Std. Deviation:	5.38	8.40	7.41	6.12	6.19	5.85	.
Std. Error:	1.20	1.88	1.66	1.37	1.38	1.31	.
Variance:	28.96	70.51	54.88	37.40	38.26	34.23	.
Coeff. of Variation:	51.15	105.86	52.63	45.95	52.38	33.09	.
Minimum:	2.00	.41	3.77	6.68	2.86	9.20	.
Maximum:	21.00	40.46	37.72	29.92	29.31	29.80	.
Range:	19.00	40.05	33.95	23.24	26.45	20.60	.
Count:	20	20	20	20	20	20	.
Missing Cells:	0	0	0	0	0	0	.
Sum:	210.40	158.64	281.50	266.18	236.17	353.66	.
Sum of Squares:	2763.56	2598.00	5004.65	4253.11	3515.75	6904.21	.

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Subject	19	2742.095	144.321		
Intensity	1	351.816	351.816	21.509	.0002
Intensity * Subject	19	310.773	16.356		
Change	2	746.684	373.342	10.811	.0002
Change * Subject	38	1312.316	34.535		
Intensity * Change	2	6.418	3.209	.186	.8309
Intensity * Chang...	38	655.275	17.244		

Dependent: DBP

Table of Epsilon Factors for df Adjustment
Dependent: DBP

	G-G Epsilon	H-F Epsilon
Intensity	1.000	1.000
Change	.980	1.092
Intensity * Change	.790	.850

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

Least Squares Means Table
Effect: Change
Dependent: DBP

	Vs.	Diff.	Std. Error	t-Test	P-Value
R/HG1	W/WHG	2.044	1.314	1.556	.1281
	RC/HG2	-3.964	1.314	-3.017	.0045
W/WHG	RC/HG2	-6.009	1.314	-4.573	.0001

	MAP						Input Column
	Low Intensity			High Intensity			
	R/HG1	W/WHG	RC/HG2	R/HG1	W/WHG	RC/HG2	
1	18.67	43.20	29.16	26.17	29.72	32.67	
2	5.60	13.70	16.77	15.43	12.68	16.20	
3	3.04	7.90	12.52	7.34	5.67	16.24	
4	9.50	6.04	5.80	15.73	12.10	14.40	
5	11.36	1.36	6.23	11.80	6.90	20.07	
6	4.20	3.23	1.50	-18.79	5.87	11.07	
7	6.80	4.40	4.14	9.90	2.96	13.50	
8	7.73	8.13	8.14	9.13	23.45	13.40	
9	10.93	5.97	10.13	10.30	8.46	12.46	
10	5.77	6.04	5.91	10.40	9.53	14.04	
11	17.47	9.91	21.17	16.83	16.56	21.40	
12	17.29	5.40	6.87	15.68	9.90	20.14	
13	17.35	12.84	12.73	22.25	9.23	19.60	
14	17.80	10.47	12.10	7.40	12.90	21.10	
15	5.60	1.59	19.29	9.37	1.13	26.97	
16	10.23	10.61	20.70	10.84	16.33	32.20	
17	25.86	7.26	5.30	10.26	13.40	11.07	
18	-1.16	5.93	7.70	14.89	18.36	22.13	
19	13.53	-.50	14.14	22.23	12.00	22.28	
20	25.01	16.12	10.03	29.16	20.74	26.79	

	MAP						Input Column
	Low Intensity			High Intensity			
	R/HG1	W/WHG	RC/HG2	R/HG1	W/WHG	RC/HG2	
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 Fl...	Free Format Fi...	Free Format Fl...	Free Format Fi...	Free Format Fi...	Free Format Fi...
Dec. Places:	2	2	2	2	2	2	3
Mean:	11.63	8.98	11.52	12.82	12.39	19.39	•
Std. Deviation:	7.32	9.10	6.97	9.69	7.02	6.52	•
Std. Error:	1.64	2.04	1.56	2.17	1.57	1.46	•
Variance:	53.52	82.88	48.61	93.84	49.26	42.49	•
Coeff. of Variation:	62.91	101.38	60.54	75.58	56.82	33.62	•
Minimum:	-1.16	-.50	1.50	-18.79	1.13	11.07	•
Maximum:	25.86	43.20	29.16	29.16	29.72	32.67	•
Range:	27.02	43.70	27.66	47.95	28.59	21.60	•
Count:	20	20	20	20	20	20	•
Missing Cells:	0	0	0	0	0	0	•
Sum:	232.58	179.60	230.33	256.32	247.89	387.73	•
Sum of Squares:	3721.53	3187.62	3576.28	5067.88	4008.33	8323.95	•

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Subject	19	3888.734	204.670		
Intensity	1	518.461	518.461	19.687	.0003
Intensity * Subject	19	500.381	26.336		
Change	2	473.087	236.543	4.951	.0123
Change * Subject	38	1815.636	47.780		
Intensity * Change	2	231.586	115.793	5.260	.0096
Intensity * Chang...	38	836.562	22.015		

Dependent: MAP

Table of Epsilon Factors for df Adjustment
Dependent: MAP

	G-G Epsilon	H-F Epsilon
Intensity	1.000	1.000
Change	.975	1.085
Intensity * Change	.787	.846

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

Least Squares Means Table
Effect: Change
Dependent: MAP

	Vs.	Diff.	Std. Error	t-Test	P-Value
R/HG1	W/WHG	1.535	1.546	.993	.3269
	RC/HG2	-3.229	1.546	-2.089	.0434
W/WHG	RC/HG2	-4.764	1.546	-3.082	.0038

Least Squares Means Table
Effect: Intensity * Change
Dependent: MAP

	Vs.	Diff.	Std. Error	t-Test	P-Value
Low Intensity, R/HG1	Low Intensity, W/WHG	2.649	1.484	1.785	.0822
	Low Intensity, RC/HG2	.113	1.484	.076	.9400
	High Intensity, R/HG1	-1.187	1.484	-.800	.4287
	High Intensity, W/WHG	-.765	1.484	-.516	.6089
	High Intensity, RC/HG2	-7.758	1.484	-5.228	.0001
Low Intensity, W/WHG	Low Intensity, RC/HG2	-2.536	1.484	-1.710	.0955
	High Intensity, R/HG1	-3.836	1.484	-2.585	.0137
	High Intensity, W/WHG	-3.414	1.484	-2.301	.0270
	High Intensity, RC/HG2	-10.407	1.484	-7.014	.0001
Low Intensity, RC/HG2	High Intensity, R/HG1	-1.299	1.484	-.876	.3866
	High Intensity, W/WHG	-.878	1.484	-.592	.5575
	High Intensity, RC/HG2	-7.870	1.484	-5.304	.0001
High Intensity, R/HG1	High Intensity, W/WHG	.421	1.484	.284	.7779
	High Intensity, RC/HG2	-6.571	1.484	-4.428	.0001
High Intensity, W/WHG	High Intensity, RC/HG2	-6.992	1.484	-4.712	.0001

	HEART RATE						Input Column
	Low Intensity			High Intensity			
	R/HG1	W/WHG	RC/HG2	R/HG1	W/WHG	RC/HG2	
1	13.03	16.44	-5.25	10.87	20.17	20.23	
2	3.74	5.63	-3.07	9.38	2.91	-.54	
3	3.40	2.84	-3.28	4.26	-18.25	-11.03	
4	1.46	2.54	-11.23	4.36	6.47	-6.90	
5	2.27	-1.13	-12.90	4.97	4.86	-7.30	
6	-4.27	4.74	-19.70	-3.37	-1.86	-15.90	
7	-4.03	14.90	-7.00	-1.53	4.24	-6.94	
8	2.90	-5.37	-5.56	-1.10	-12.31	-1.60	
9	.76	4.90	-1.17	9.16	7.46	-3.20	
10	-1.70	3.30	-14.03	-5.64	6.24	-9.33	
11	.90	8.62	-1.76	2.43	12.56	1.43	
12	.57	2.97	3.87	-.93	4.63	7.50	
13	11.76	9.36	-7.20	11.56	6.30	-8.30	
14	2.33	9.10	2.53	2.50	21.04	4.37	
15	4.67	31.26	-4.50	11.50	-.56	-2.21	
16	-3.48	5.48	-1.47	-1.13	8.17	4.33	
17	-4.98	-2.00	-15.14	.15	-2.20	-15.47	
18	-.94	7.97	2.50	7.81	10.73	10.73	
19	-2.25	.20	-5.33	-.52	4.04	-6.49	
20	4.28	8.93	-.73	6.80	14.10	6.11	

	HEART RATE						Input Column
	Low Intensity			High Intensity			
	R/HG1	W/WHG	RC/HG2	R/HG1	W/WHG	RC/HG2	
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 Fl...	Free Format Fi...	Free Format Fi...	Free Format Fi...
Dec. Places:	2	2	2	2	2	2	3
Mean:	1.52	6.53	-5.57	3.58	4.94	-2.03	.
Std. Deviation:	4.76	7.88	6.33	5.28	9.34	9.05	.
Std. Error:	1.06	1.76	1.41	1.18	2.09	2.02	.
Variance:	22.66	62.11	40.03	27.92	87.24	81.83	.
Coeff. of Variation:	312.97	120.62	-113.57	147.74	189.19	-446.59	.
Minimum:	-4.98	-5.37	-19.70	-5.64	-18.25	-15.90	.
Maximum:	13.03	31 26	3.87	11.56	21.04	20.23	.
Range:	18.01	36.63	23.57	17.20	39.29	36.13	.
Count:	20	20	20	20	20	20	.
Missing Cells:	0	0	0	0	0	0	.
Sum:	30.42	130.68	-111.42	71.53	98.74	-40.51	.
Sum of Squares:	476.81	2034.05	1381.28	786.33	2145.13	1636.75	.

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value
Subject	19	2946.775	155.093		
Intensity	1	53.440	53.440	1.818	.1934
Intensity * Subject	19	558.459	29.393		
Change	2	1884.429	942.214	21.000	.0001
Change * Subject	38	1704.921	44.866		
Intensity * Change	2	140.021	70.010	2.943	.0648
Intensity * Chang...	38	903.983	23.789		

Dependent: HEART RATE

Table of Epsilon Factors for df Adjustment
Dependent: HEART RATE

	G-G Epsilon	H-F Epsilon
Intensity	1.000	1.000
Change	.998	1.115
Intensity * Change	.873	.954

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

Least Squares Means Table
Effect: Change
Dependent: HEART RATE

	Vs.	Diff.	Std. Error	t-Test	P-Value
R/HG1	W/WHG	-3.187	1.498	-2.128	.0399
	RC/HG2	6.347	1.498	4.238	.0001
W/WHG	RC/HG2	9.534	1.498	6.365	.0001

variable	x2	xy	y2	b	(xy)2/x2	(y-y)2	df	F
SBP								
post 40	8990	3387.90	1371.75	0.38	1276.74	95.01	28.00	
ex 40	8990	3290.01	1305.55	0.37	1204.02	101.53	28.00	
rest 40	8990	2994.97	1033.11	0.33	997.76	35.35	28.00	
						231.89	84	2.76
pooled	26970	9672.9	3710.41	0.36	3469.21	241.20		
						9.31	2	4.65
								1.69
post 30	8990	2102.1	566.74	0.23	491.50	75.24	28.00	
ex30	8990	1048.2	362.78	0.12	122.22	240.56	28.00	
rest 30	8990	1580.4	315.79	0.18	277.82	37.97	28.00	
						353.76	84	4.21
pooled	26970	4730.7	1245.31	0.1754	829.779	415.53		
						61.77	2	30.88
								7.33

variable	x2	xy	y2	b	(xy)2/x2	(y-y)2	df	F
DBP								
post 40	8990	3634.4	1504.45	0.40	1469.24	35.21	28.00	
ex 40	8990	2789.7	891.23	0.31	865.68	25.55	28.00	
rest 40	8990	2873.3	962.1	0.32	918.31	43.79	28.00	
						104.55	84	1.24
pooled	26970	9297.3	3357.78	0.34	3205.04	152.74		
						48.19	2	24.09
								19.36
post 30	8990	2842.4	941.98	0.32	898.67	43.31	28.00	
ex30	8990	1812.9	395.66	0.20	365.57	30.09	28.00	
rest 30	8990	2026.1	470.66	0.23	456.63	14.03	28.00	
						87.43	84	1.04
pooled	26970	6681.3	1808.3	0.25	1655.18	153.12		
						65.69	2	32.84
								31.55

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= 2.00	
	A: R vs Ex	0.06	277.82	37.97	122.22	240.56	278.53	56	4.97	0.0002	0.03326	1.78	ns	
	B: Po vs R	0.06	491.50	75.24	277.82	37.97	113.21	56	2.02	0.0002	0.02121	2.74	sd	
	C: Po vs E	0.12	491.50	75.24	122.22	240.56	315.8	56	5.64	0.0002	0.03542	3.31	sd	
DBP 30														
	A: R vs Ex	0.02	456.63	14.03	365.57	30.09	44.12	56	0.79	0.0002	0.01324	1.79	ns	
	B: Po vs R	0.09	898.67	43.31	456.63	14.03	57.34	56	1.02	0.0002	0.01509	6.02	sd	
	C: Po vs E	0.11	898.67	43.31	365.57	30.09	73.40	56	1.31	0.0002	0.01708	6.71	sd	
DBP 40														
	A: R vs Ex	0.01	918.31	43.79	865.68	25.55	69.34	56	1.24	0.0002	0.0166	0.56	ns	
	B: Po vs R	0.08	1469.24	35.21	918.31	43.79	79.00	56	1.41	0.0002	0.01772	4.78	sd	
	C: Po vs E	0.09	1469.24	35.21	865.68	25.55	60.76	56	1.08	0.0002	0.01554	6.05	sd	
SBP 40														
	A: Po vs Ex	0.01	1204.02	101.53	1276.74	95.01	196.54	56	3.51	0.0002	0.02794	0.39	ns	
	B: Po vs R	0.04	997.76	35.35	1276.74	95.01	130.36	56	2.33	0.0002	0.02276	1.92	ns	
	C: Ex vs R	0.03	997.76	33.41	1204.02	101.53	134.94	56	2.41	0.0002	0.02315	1.42	ns	