Cardiovascular Responses To Physical Stressors In Normotensive And Exercise Hypertensive Individuals

by

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ABSTRACT

An exaggerated pressor response to specific physical stressors has been suggested as possible predictors for the development of future hypertension. This study compared the cardiovascular responses to the PWC 140, cold pressor test (CPT), and isometric hand grip (IHG), to evaluate the consistency in the blood pressure response among these physical stressors. Eighteen resting normotensive males (mean age 23.3 ± 1.7 yrs.) were classified as either normotensive at exercise (ΔSBP<45 mmHg, and ΔDBP<10 mmHg), or exercise hypertensive (ΔSBP≥45 mmHg, or ΔDBP≥10 mmHg) on the basis of their blood pressure response to the first stage of the Canadian Aerobic Fitness Test (CAFT) for their age group. Subjects were administered a 6-min PWC 140 bicycle ergometer test, a 120 sec CPT (forearm and hand immersion), and a 120 sec IHG at 30 % MVC. Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), heart rate (HR), stroke volume (SV), cardiac output (CO), and ejection fraction (EF) were recorded every 5 seconds during a one min resting period, during exercise, and for 3 min post exercise using a Finapress 2000 BP monitor and BoMed bioimpedance cardiac monitor. Delta scores (peak exercise - rest) were calculated for each variable. Data were analyzed using a 2-way factorial ANOVA for each exercise and recovery variable. Linear regression analyses were calculated to determine the slope of the SBP and DBP response to the CPT and IHG. The results of this study indicate that; 1) there were no significant differences between the two groups for the cardiovascular responses to the PWC 140, IHG, and CPT with the exception of a greater CO and SV in normotensives on the PWC 140, and a higher HR in exercise hypertensives on the CPT; 2) the exercise hypertensives had a greater rate of increase in SBP and DBP than the normotensives over the first 90 seconds of the CPT; 3) there was no consistent pattern in the individual responses to the CAFT, CPT, and IHG with respect to ΔSBP. It was concluded that within the context of this study, the blood pressure responses to dynamic exercise, isometric exercise, and cold stress were not consistent among the subjects and tests. These inconsistent responses may be due to the different mechanisms involved in the response to each of these physical stressors.

INTRODUCTION

Essential hypertension is one of the serious health concerns of our time. For years it has been known that hypertension is a leading contributor to cardiovascular disease. It is one of the primary risk factors associated with heart attack, stroke, heart and kidney failure, and is one of the leading diseases linked to premature death and disability in Canada (Canada Blood Pressure Survey, 1989).

It is estimated that 15 to 25 percent of individuals in the western world are hypertense (Kaplan, 1986). In Canada alone, two million adults aged 20-69 years are thought to be afflicted with the disease (Foder, 1980). Persons with hypertension face the risk of developing cardiovascular abnormalities 20 years earlier than their normotensive counterparts (Kaplan, 1983). One of the major concerns is that hypertension may lie asymptomatic for several years before any sign of cardiovascular dysfunction is evident. It has been estimated that of those who are hypertense, seventy-five percent are unaware of their condition or are not being treated for the disease (Kaplan, 1986). At present, there is no method of predicting an individual's risk of developing hypertension other than through repeated resting and ambulatory blood pressure readings. However, a number of tests that elicit a pressor response have been suggested as possible predictors for the development of future hypertension.

The Cold Pressor Test (CPT) which involves the immersion of an extremity in ice water, has long been used to evaluate the cardiovascular system's response to physical stress. Several studies (Ohlsson &

Henningsen, 1982; Menkes et al., 1989; Kasagi et al., 1995), have demonstrated that the strength of the pressor response is greater in hypertensives than in normotensives when exposed to the cold pressor test. Research by Wood et al., (1984), found that normotensives who show an exaggerated response to the CPT are more likely to develop future hypertension than individuals who respond normally, suggesting the potential use of the CPT as a predictor for hypertension.

Similar work has been reported using isometric exercise to elicit a pressor response. Several studies have evaluated the cardiovascular response to the isometric hand grip (Laird et al.,1979; Cantor et al., 1987). Again hypertensives demonstrated an exaggerated cardiovascular response to isometric exercise when compared to normotensives.

These findings have lead researchers to believe that an exaggerated cardiovascular response to the CPT or isometric exercise in normotensive individuals may be a predictor of the future development of sustained hypertension.

Research by Wilson and Meyer (1981), Jackson, Squires, and Beard, (1983), and Dlin et al., (1983), examined the use of blood pressure in response to dynamic exercise as an early predictor for the future development of hypertension. These studies reported differences in the cardiovascular response to aerobic exercise between hypertensives and normotensives. In addition, normotensives who possessed an exaggerated response to dynamic exercise were more likely to develop resting sustained hypertension than normal responders during a follow-up.

Although there has been considerable research into the blood pressure response to aerobic exercise, a standardized protocol had not been developed. Jetté et al., (1991) quantified the blood pressure response to a sub-maximal stepping exercise using the Canada Aerobic Fitness Test (CAFT). Individuals were classified according to their blood pressure response following the first three-minute stage (Stage A) of the CAFT, for their age group and gender. An increase in systolic blood pressure ≥ 45 mmHg (or ≥ 10 mmHg diastolic) over their resting value was classified as an exaggerated response. This test could provide a simple tool to identify individuals who may be at an increased risk for the development of hypertension.

Each of these physical stressors have been used to assess an individuals risk of developing future sustained hypertension based on their ability to place an increased demand on the cardiovascular system. It has been indicated that hypertensives have a greater response to these physical stressors than normotensives due primarily to an increased sympathetic tone typically found in hypertensives (Julius, 1990). As well, it has been reported that normotensive individuals who have an exaggerated response to physical stressors also have an increased sympathetic tone which is magnified at exercise. Thus these exaggerated responders are thaught to be in a 'transitional stage' and are more likely to develop hypertension in the future than those who exhibit a normal response to physical stressors.

It is evident that although each of the physical stressors outlined above place an increased demand on the cardiovascular system, the mechanisms governing these responses appear to be different. However, it would be expected that those individuals who show an exaggerated response to one of these stressors would show a similar response to another stressor. An examination of the current research appears to indicate that the hemodynamic response in individuals with an exaggerated blood pressure response to exercise may in fact differ from individuals with a normal blood pressure response to exercise. As far as it can be determined, no study has compared the blood pressure response to the CPT, isometric hand grip, and dynamic exercise in the same individuals.

The purpose of this study was to compare the cardiovascular profiles to dynamic exercise (PWC 140), isometric exercise (hand grip), and cold pressor tests among individuals who are:

1) normotensive at rest and at exercise;

2) normotensive at rest but hypertensive at exercise;

as classified on the basis of their blood pressure response to Stage A of the CAFT.

METHODOLOGY

This study was approved by the Human Research Ethics Committee of the University of Ottawa.

Subjects

Eighteen normotensive male volunteers who met the following criteria served as subjects for this experiment:

1) between the ages of 20 and 35;

2) resting blood pressure below 140/90 mmHg;

3) non-smokers;

4) body mass index < 27 kg/m^2;

5) not currently taking any medication that may alter cardiovascular function.

Subjects were selected according to their resting blood pressure and their blood pressure response to Stage A of the Canadian Aerobic Fitness Test (CAFT) for their age group. All subjects were classified into one of the following two categories:

1) normotensive at both rest and exercise;

2) normotensive at rest, hypertensive at exercise;

The criteria used for determining a hypertensive response to exercise was an increase of 45 mmHg systolic and/or an increase of 10 mmHg diastolic in response to Stage A of the CAFT, recorded on two separate occasions.

Apparatus

Heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were monitored using the Finapres 2300 blood pressure monitor (Ohmeda, Missisauga, Ont.). The Finapres 2300 is a non-invasive, continuous blood pressure monitor based on the Penaz technique (Penaz, 1973). The technique involves the measurement of changes in vascular volume within the finger. A photoplethysmograph, consisting of a light source and a photoelectric cell are incorporated into an inflatable finger cuff which is placed around the finger. A constant blood volume is determined by the photoplethysmogram and becomes a 'set point' for a servo loop. As the photoplethysmogram varies from the 'set point' , the pressure is increased or decreased to maintain the set point. The resulting cuff pressure is measured by an electronic pressure transducer and displayed as the arterial pressure. Blood pressure measured by the Finapres 2300 has been strongly correlated (0.98) to intra-arterial blood pressure (Hartman & Bassenge, 1989).

Cardiac output, stroke volume, and ejection fraction were monitored using the BoMed NCCOM3-R7 cardiac monitor (BoMed Manufacturing Ltd., Irvine, CA, U.S.A.). The BoMed operates on a bioimpedance model. Since the plasma within the blood is an electrically conductive substance within the body, the impedance of tissue can be measured to give measures of volume and velocity. The increase in blood volume and blood velocity during systolic upstroke contribute to the change in bioimpedance. The accuracy of the BoMed has been compared to the widely accepted thermodilution procedure (r=.88). However, as with thermodilution, the average expected error can be ± 15 to 20% (Bearnstein, 1986). All cardiovascular variables were collected and stored on a Tatung TCS 7000, personal computer (Tatung Co., Taipei).

Resting blood pressure and blood pressure recordings following each stage of the CAFT were measured using a sphygmomanometer (Tycos, Arden North Carolina). Isometric hand grip was performed on a dynamometer constructed from an air pressure gauge (H.O. Trerice Co., Detroit Michigan) connected by surgical tubing to a rubber squeeze bulb. The PWC 140 was performed on a Monark Ergomedic 818 E bicycle ergometer (Monark, Sweden). Cadence during the PWC 140 was maintained using a Seiko SQM 349, quartz metronome (Seiko, Japan).

Procedure

Introductory Session

After arrival at the laboratory, the subjects were briefed on the nature of the study and were given a letter of information outlining the requirements of the study. Subjects were asked to comply to the following instructions for the experimental session:

1) refrain from physical activity on day of the experimental session;
2) refrain from caffeine for 6 hours prior to the experimental session;
3) eat a light meal 4 hours prior to testing;
4) have at least 8 hours of sleep the night before testing.

Subjects were then asked to complete and sign the PAR-Q, and consent form. Subjects then remained seated, resting for 10 minutes, after which resting blood pressure was taken using a sphygmomanometer. Following an additional 5 minute rest, a resting heart rate was taken along with a resting blood pressure verification.

Upon completion of the PAR-Q, consent form and resting blood pressure recordings, subjects were administered the CAFT according a modified protocol (Jetté et al., 1991). Heart rates were taken between 5-15 seconds following completion of Stage A, and blood pressure was measured by sphygmomanometer between 15 and 45 seconds following the completion of the stage. If heart rates were below the predetermined ceiling values, the subject performed stages B and C. Heart rates and blood pressures were again recorded as described above following stages B and C. The CAFT was used to classify the subject's blood pressure response to exercise, and to determine a predicted VO_2 max.

A series of questionnaires were then completed by the subject. Questionnaire material included demographic data, personal and family blood pressure history, and lifestyle information.

Maximum voluntary contraction (MVC) on the isometric hand grip dynamometer was then determined. Subjects, using their dominant hand, and placing their arm unsupported at their side at a 90° angle, were asked to perform three trials with a one minute rest between trials. The average of the best two trials was recorded as his MVC.

Subjects were then initiated to a 120 second (continuous) cold pressor test (CPT). In a seated position, subjects immersed their dominant hand and forearm, up to their elbow, in a basin of ice-water (2°-4°) for a period of 120 seconds.

Following the completion of the cold pressor test, subjects were initiated to the PWC 140 test on the bicycle ergometer. During the first three minutes of the test, subjects cycled at a rate of 60 rpm, in cadence with a metronome, during which time the tension was adjusted (beginning at 1 kpm), to achieve a steady state heart rate of 140 beats per minute. Subjects continued to pedal at this intensity for the remainder of the 6-minute test. Heart rate was monitored throughout the test using a Polar Vintage XL (Polar USA Inc., Stamford Ct.) heart rate monitor.

Experimental Session

Subjects, upon arrival at the laboratory, remained seated for 10 minutes after which, resting blood pressure was measured and verified again following an additional 5-minute rest. Stage A of the CAFT was then re-administered to confirm the subject's blood pressure response at exercise.

Subjects were then required to rest (seated) for 15 minutes during which time the subject was prepared for the isometric hand grip test. The Finapres 2300 blood pressure cuff was placed around the middle phalange of the third finger on the non dominant hand. The hand was positioned at heart level and held in place using a sling to support the arm. A total of eight electrodes were carefully positioned on the subject for monitoring of cardiac variables. The first electrode was attached at the intersection of a line encircling the root of the neck with the frontal plane. The second electrode was placed 5 cm directly above. The next electrode was attached at the mid-axillary line at the xiphoid process level. The final electrode was placed directly 5 cm below. This procedure was repeated for the opposite side of the body.

The subjects then performed the isometric hand grip test. Subjects were required to maintain 30% of MVC for a period of 120 seconds using his dominant hand. A gauge was placed within the subjects view, with his 30% MVC clearly indicated to ensure that this level was maintained. Cardiovascular measures were recorded for one minute prior to the test, throughout the duration of the test, and for three minutes following the test.

Following a 15-minute seated rest, the subject then performed the CPT as described above. Cardiovascular variables were again monitored for one minute prior to, during, and for three minutes following the CPT. Following a 15 minute seated rest, the subject then completed the PWC

140 test as described above. Cardiovascular responses were monitored for one minute prior to, during, and for three minutes following the test.

Statistical Analysis

Delta scores (Δ) were calculated for heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP), for each stage of the CAFT. ΔHR was calculated by subtracting the resting HR from the ten second HR taken 5 - 15 seconds following each stage (converted to beats/min). ΔSBP and ΔDBP were calculated by subtracting the resting blood pressure from the blood pressure taken 15 - 45 seconds following each stage. All variables for the CAFT were analyzed using a two-way factorial analysis of variance (ANOVA) to determine if there were any differences ($p<.05$) between the groups (exercise normotense, exercise hypertense).

Exercise delta scores (Δ) were also calculated for all cardiovascular variables during the PWC 140, IHG, and CPT, by determining the change from rest to peak level at exercise. Resting values were calculated by taking the average of the 15-45 second period prior to exercise. Peak exercise values were determined by taking the peak response during exercise. Total systemic resistance (TSR) was calculated using the formula TSR=MAP/CO * 79.92 (Eliot, 1988). Recovery delta scores were determined by subtracting the resting response from the recovery response (calculated by averaging the last 15 seconds of recovery). All variables were analyzed using a two way ANOVA to determine if there were any differences ($p<.05$) between the two groups (exercise normotense, exercise hypertense).

Linear regression analyses were performed for SBP and DBP on each subject during the IHG and CPT to determine the slope of these responses. Group means were calculated for each test and a t-test was performed to determine if there were significant differences ($p<.05$) between the groups. All statistical analysis was performed using the Statsview 4.01 statistical package for Macintosh computers.

RESULTS

The eighteen subjects who participated in this study were classified as either normotensive at exercise or exercise hypertensive on the basis of their blood pressure response to stage A of the CAFT. As a result, 10 subjects were classified as exercise normotensive and 8 as exercise hypertensive. The mean age and physical characteristics of the subjects are shown in Table 1. There were no significant differences between groups for age, height, weight, body mass index (BMI), waist to hip ratio (WHR), sum of skinfolds (SOS), percent body fat, predicted VO_2 max, and isometric maximum voluntary contraction (MVC).

There were also no significant differences for resting heart rate (58.8 ±6.8 bpm; 59.5 ±7.8 bpm), resting systolic blood pressure (123.8 ±5.8 mmHg; 124.8 ±3.5 mmHg) or resting diastolic blood pressure (80.4 ±3.5 mmHg; 82.1 ±5.1 mmHg) between the normotensives and exercise hypertensives respectively (Table 2).

The mean systolic and diastolic blood pressure responses to stages A, B, and C, of the CAFT are shown in Figures 1 & 2. The mean delta scores (post stage - resting) for heart rate (ΔHR), systolic (ΔSBP) and diastolic

(ΔDBP) blood pressures in response to stages A, B, and C, of the Canadian Aerobic Fitness Test (CAFT) were calculated for each group and are presented in Table 2. The exercise hypertensives had a significantly higher ΔHR in response to stage A ($p=.023$) and stage C ($p=.050$) of the CAFT, in relation to the normotensives. The exercise hypertensives also had a significantly higher ΔSBP in response to stage A ($p=.0009$), stage B ($p=.0007$), and stage C ($p<.0001$) as well as a greater ΔDBP in response to stage B ($p=.043$) of the CAFT.

The mean systolic (SBP), diastolic (DBP) and mean (MAP) blood pressures, heart rate (HR), cardiac output (CO), stroke volume (SV), ejection fraction (EF), and total systemic resistance (TSR), in response to the PWC 140, isometric hand grip, and cold pressor tests for each group are shown in Table 3. Significant differences were found between groups on CO ($p=.016$) and SV ($p=.017$) for the PWC 140, with the normotensives eliciting the greater response. The exercise hypertensive subjects had a greater HR response to the CPT ($p=.025$). No other differences were found between groups on the PWC 140 or CPT. Also, no significant differences were found between groups with respect to the variables for the isometric hand grip.

Mean delta recovery data for all cardiovascular variables in response to the PWC 140, IHG, and CPT are presented in Table 4. Delta scores were calculated by subtracting the average of the last 15 seconds of recovery from the resting value (average of 15-45 seconds prior to exercise) for each variable. There were no significant differences found between groups.

Regression analysis was performed to determine the slope of the SBP and DBP responses for each subject on the CPT and isometric hand grip at the 90th second of exercise (Table 5). A t-test was performed to determine if there were any significant differences in slopes between the groups (Table 5). The exercise hypertensives had a greater rate of increase (slope) than the normotensives (Figures 10 & 11) on the CPT for both SBP ($p=.0001$) and DBP ($p=.008$). There were no significant differences between groups on slope for both SBP and DBP on the isometric hand grip.

The individual systolic blood pressure response for each test is presented in Table 6. Subjects were initially classified as either normotensive or exercise hypertensive based on their blood pressure response to the CAFT as described above. Individuals were then classified as either exaggerated responders or normal responders to the IHG and CPT. An exaggerated SBP response on the IHG was a $\Delta SBP \geq 45$ mmHg. On the CPT an exaggerated response was a $\Delta SBP \geq 50$ mmHg. As a result, 60% of the exercise normotensive responders to the CAFT, responded with a normal blood pressure to the isometric hand grip, and 50% responded with a normal blood pressure to the cold pressor test (Figure 2). Of the exaggerated responders to the CAFT 25% had an exaggerated response to the isometric hand grip, and 37.5% had an exaggerated response to the cold pressor test.

DISCUSSION

There is general agreement that hypertensives have a higher cardiovascular reactivity to physical stressors than normotensives. As well, it has been suggested that normotensives who exhibit an exaggerated blood pressure response to dynamic exercise (Wilson & Meyer, 1981; Jetté, et al., 1991), isometric exercise (Cantor et al.,1987; Chaney & Eyman, 1988), or the cold pressor test (Hines & Brown, 1936; Kasagi et al., 1995) are more likely to develop future sustained hypertension. However, no study has assessed the cardiovascular response to these three physical stressors in the same individuals. As such, the purpose of this study was to compare the cardiovascular responses to submaximal dynamic exercise (PWC 140), submaximal isometric exercise (IHG), and the cold pressor tests (CPT) in normotensive individuals classified as either normal or exaggerated responders on the basis of their blood pressure response to the CAFT. The results of this study indicate that 1) there were no significant differences between groups with respect to their cardiovascular response to the PWC 140, IHG, CPT (with the exception of CO and SV on the PWC 140 and HR on the CPT); 2) subjects classified as exercise hypertensive showed a steeper rate of increase over the first 90 seconds of the CPT; 3) there was no consistency in the individual blood pressure classification (normal or exaggerated) to the CAFT, CPT, and IHG with respect to their ΔSBP.

Blood Pressure Response To The CAFT

Jetté et al., (1991), examined the blood pressure response to a submaximal stepping exercise (CAFT) to develop provisional norms for the immediate post-exercise blood pressure response to help identify individuals who may be at an increased risk for developing hypertension. Based on their results, an increase in systolic blood pressure ≥ 45 mmHg (mean ± 1 SD) or an increase in diastolic blood pressure ≥ 10 mmHg to the first stage of the CAFT, was classified as an exaggerated response. Applying these criteria to this study, ten resting normotensives were classified as exercise normotensive and eight were classified as exercise hypertensive. The exercise hypertensives exhibited a significantly higher ΔSBP, ΔDBP, and ΔHR for all stages of the CAFT with the exception of ΔDBP at stage A&C and ΔHR at stage B, all of which showed a similar trend ($p>.05 \leq .10$). Thus, the results from this study indicate that in a relatively young, healthy and fit group, a large proportion of the subjects will show an exaggerated blood pressure response to moderate dynamic exercise. In fact, 45% of the normotensive subjects in this study exhibited an exaggerated blood pressure response to moderate dynamic exercise.

Several studies have indicated that normotensive individuals who demonstrate an exaggerated blood pressure response to dynamic exercise may be at an increased risk for the development of sustained hypertension (Davidoff et al., 1982; Dlin et al., 1983; Jetté et al., 1991). As an example, Dlin et al., (1983), evaluated the blood pressure response of 150 normotensive individuals seventy-five of whom were exaggerated responders to submaximal bicycle ergometry. The best predictor for the development of future hypertension (average follow-up of 5.8 yrs), was an exaggerated blood pressure response to bicycle ergometry. However, not all studies have agreed that the blood pressure response to dynamic exercise is a valuable predictor of future hypertension (Fixlar et al., 1983; Goble & Sheiken, 1991).

Hemodynamic Comparison of Normotensives vs. Exercise Hypertensives

A comparison of the cardiovascular response of normotensives and exercise hypertensives to the PWC 140, IHG, and CPT indicated that there were no significant differences between groups during exercise with the exception of ΔCO and ΔSV on the PWC 140, and ΔHR on the CPT, or during recovery. The normotensives had a greater ΔCO and ΔSV in response to the PWC 140 whereas the exercise hypertensives elicited a greater ΔHR in response to the CPT, although there was no difference in SBP. The greater ΔCO and ΔSV by the normotensives on the PWC 140 appears to contradict previous research which indicates that it is the young hypertensives who typically demonstrate a greater cardiac output in response to dynamic exercise as a result of their hyperkinetic circulation (Julius & Conway, 1968; Lund Johansen 1991). The increased cardiac output in these individuals has been reported to be primarily a result of an elevated heart rate and stroke volume in relation to a given workload and has been attributed to an increased sympathetic tone coupled with a decreased parasympathetic tone (Julius, 1990).

Our study produced similar results in response to the CAFT where heart rate was elevated in exercise hypertensives in response to a standardized workload. With respect to the PWC 140, however, it should be noted that individuals were not working at a predetermined workload. Instead, subjects were evaluated in response to a set heart rate of 140 bpm. Therefore, since heart rate was controlled for, the lower cardiac output in the exercise hypertensives would be a result of a decreased stroke volume. This may be an indication of increased left ventricular hypertrophy (LVH) which has been found in individuals who are at an increased risk for the development of sustained hypertension (Devereux et al., 1991). In fact, some studies have indicated an increased LVH may be present in individuals long before an elevation in resting systolic blood pressure is evident (Mahoney et al., 1988;). Furthermore, LVH has been associated with an increased exercise SBP (Janz et al., 1995). An increased LVH also results in a decreased left ventricular capacity and has been associated with a decrease in stroke volume and cardiac output. Thus, it would be expected that combined with the increased sympathetic activity, the decreased LV function which may be present in these exercise hypertensives would result in these individuals working at a lower relative workload than the normotensives. Although this was observed in this study, the difference was not significant.

While it might be expected that physical fitness is associated with LVH, Janz et al (1995) found that after correcting for body composition, none of the physical fitness parameters (peak VO^2, peak work, and maximal grip strength) were not associated with LV mass. As well, these physical parameters were not associated with resting systolic blood pressure. As such, it was concluded that physical fitness is not an important predictor of LVH or resting SBP. However, peak systolic blood pressure was associated with LVH. While there were no differences in resting SBP or physical fitness between the normotensives and exercise hypertensives in this study, the exercise hypertensives did have a greater increase in blood pressure in response to Stage A of the CAFT. This may be an indication of an increased LVH in these individuals placing them at an increased risk for the future development of hypertension.

The cardiovascular responses to the CPT, however, were quite different than that of the PWC 140. The exercise hypertensives had a greater ΔHR to the CPT than the normotensives along with an increased ΔSV and ΔCO ($p>.05 < .10$). This could indicate the hyperkinetic state typical of the pre-hypertensives reported by others (Hines, 1940; Kasagi et al., 1995). The initial response to the CPT is generally characterized by an increase in both HR and blood pressure. However, results from this study and other work done in our laboratory (Liu, 1994) have shown an initial decrease in both SBP and DBP over the first 5-20 seconds of the CPT, followed by an increase typically reported by others. This observation which has not been reported elsewhere. The decreased SBP and DBP was evident in both the normotensives and exercise hypertensives, although the exact mechanism responsible for this response is still not clear. Increases in HR and SBP typically reported in response to the CPT are said to be a result of an immediate increase in sympathetic activity (Seals, 1990). This increased sympathetic activity may be due in part to the cold sensation, or feeling of pain associated with cold exposure (Seals, 1990). Individuals who have an increased cardiovascular reactivity to the CPT may have an increased sympathetic tone, resulting in their response to be elevated in comparison to a normotensive individual. This could in fact be an indication of the hemodynamic changes which may place these individuals at an increased risk for the development of hypertension (Kasagi et al., 1995).

SBP and DBP response to CPT & IHG

To examine the blood pressure response to the CPT and IHG, linear regression slopes for SBP and DBP over the first 90 seconds for both the

CPT and IHG were calculated. Results indicate that individuals who were classified as exercise hypertensive on the basis of their blood pressure response to the CAFT had a steeper rate of increase for both SBP and DBP on the CPT. This may indicate a similarity in blood pressure response in exaggerated responders to the CAFT and the blood pressure response over the first 90 seconds of the CPT. Thus, it appears from these results that the blood pressure of exercise hypertensives may in fact increase at a steeper rate on the CPT, although no significant differences were observed at the second minute of exercise.

Other studies have reported a difference in the rate of increase (slope) in blood pressure, in response to exercise (Lund-Johansen, 1988), and cold stress (Hines, 1940) between normotensives and hypertensives. Hypertensives have been shown to have a steeper increase in blood pressure in response to a stimulus, possibly due to an exaggerated sympathetic tone (Conway, 1984; Julius 1990). However, Fagus et al., (1989), reported a gradual decrease in sympathetic activity after the first 60 seconds of the CPT. This may provide a possible explanation for the results in this study in which a significant difference in the ΔSBP response was observed after 90 seconds of the test, but not at the 2-min point. The drop in sympathetic activity may have been slightly greater in the exercise hypertensive group and could have masked the differences by the end of the test. Thus, it could be that those who have an initial exaggerated response to the CPT, may have an exaggerated sympathetic tone, which could place them at an increased risk of developing hypertension.

Although there was a difference between groups on the ΔSBP response to the CPT, similar results were not observed on the IHG. The initial response to isometric exercise is a decreased vagal tone as a result of decreased parasympathetic activity (Freyschuss, 1970). The decreased parasympathetic activity increases HR and blood pressure over the first minute of isometric exercise (Mitchell et al., 1983). Further increases are then a result of increased sympathetic activity (Mark et al., 1985). Thus, the differences in the SBP response to the CPT and IHG may be due in part to the different central control mechanisms governing the response.

Individual ΔSBP classification in response to the CAFT, CPT and IHG.

The primary purpose of this study was to determine if an individual classified on the basis of their blood pressure response to the CAFT, would exhibit a consistent blood pressure classification in response to the CPT, and IHG. A closer evaluation of the individual systolic blood pressure responses indicates that there is no apparent consistency in the pressor response to the CAFT, IHG, and CPT. Individuals were initially classified on the basis of their blood pressure response to stage A of the CAFT, with an increase of 45 mmHg systolic, or 10 mmHg diastolic resulting in an exaggerated response (Jetté et al., 1991). Using recently collected data from our laboratory (Liu, 1994) individuals were classified as exercise hypertensive on the IHG and CPT if their ΔSBP increased by 1 SD above the mean, as previously done by Jetté et al., (1991) for the CAFT. This resulted in an increase of 45 mmHg on the IHG and 50 mmHg on the CPT being considered an exaggerated response. Using this criteria, only 2 individuals were normal responders to all three physical stressors, while none were classified as exercise hypertense on all three stressors (Table 6). In fact of those individuals who had an exaggerated response to the CAFT, 75% had a normal response to the IHG and 62.5% had a normal response to the CPT (Figure 2). As such, the results from this study indicates that there is no consistency among the blood pressure response to these three physical stressors.

Conclusions

The results from this study indicate that in a relatively young, healthy and fit group, a large proportion (45%) of the subjects will show an exaggerated blood pressure response to moderate dynamic exercise (CAFT). However, the pressor response to dynamic exercise, isometric exercise, and cold stress is not consistent among subjects and tests. When individuals were classified as exaggerated responders on the basis of their blood pressure response to dynamic exercise (CAFT), they did not necessarily respond in the same manner on the other physical stressors. This may be due to the different physiological mechanisms which are involved in each of these physical stressors. As well, there may be individual differences in response to these physical tests. Thus, the use of a combination of these physical stressors to assess an individual's susceptibility to sustained hypertension may be more appropriate. Prospective research could help to determine which of these the three physical stressors (or combination of physical stressors) would be most effective in assessing an individual's risk for the development of sustained hypertension.

REFERENCES

Bernstein, D.P., (1986). Continuous noninvasive real-time monitoring of stroke volume and cardiac output by thoracic electrical bioimpedance. Critical Care Medicine. 14(10), 898-901.

Bo Med NCCOM3-R7 Operator/Service Manual. (1987). BoMed Ltd: Irvine CA.

Cantor, A., Gold, B., Gueron, M., Cristal, N., Prajgrod, G., & Shapiro, Y., (1987). Isotonic (dynamic) and isometric (static) effort in the assessment and evaluation of diastolic hypertension: correlation and clinical use. Cardiology. 74, 141-146.

Chaney, R. H., & Eyman, R. K., (1988). Blood pressure at rest and during maximal dynamic and isometric exercise as predictors of systemic hypertension. The American Journal of Cardiology. 62. 1058-1061.

Conway, J., (1984). Hemodynamic aspects of essential hypertension in humans. Physiological Reviews. 64(2), 617-660.

Davidoff, R., Schamroth, C.L., Goldman, A.P., Diamond, T.H., Cilliers, A.J., & Myburg, D.P., (1982). Postexercise blood pressure as a predictor of hypertension. Aviation Space and Environmental Medicine. 53 (6), 591-594.

Devereux, R.B., de Simone, G., Koren, M.J., Roman, M.J., & Laragh, J.H., (1991). Left ventricular mass as a predictor of development of hypertension. American Journal of Hypertension. 4, 603s-607s.

Dlin, R.A., Hanne, N., Silverberg, D.S., & Bar-Or, O., (1983). Follow up of normotensive men with exaggerated blood pressure response to exercise. American Heart Journal. 106, 316-320.

Goble, M.M. & Schieken, R.M. (1991). Blood pressure response to exercise: A marker for future hypertension? American Journal of Hypertension. 4, 617S-620S.

Fagius, J., Karhuvaara, S. & Sundlof, G., (1989). The cold pressor test: effects on sympathetic nerve activity in human muscle skin nerve fascicles. Acta Physiology Scand, 137: 324-334.

Fixlar, D.E., Laird, W.P., & Dana, K. (1985). Usefulness of exercise stress testing gor prediction of blood pressure trends. Pediatrics. 75(6), 1071-1075.

Freyschuss, U. (1970). Elicitation of heart rate and blood pressure increase on muscle contraction. Journal of Applied Physiology, 28(6), 758-761.

Hartmann, B., & Bassenge, E., (1989). Nichtinvasive, kontinuierliche Messung des Fingerarteriendrucks mit dem Servo-Plethysmo-Manometer Finapres®. Herz. 14(4), 251-259.

Hines, E.A., & Brown, G.E., (1936). The cold-pressor test for measuring reactability of the blood pressure: data conserning 571 normal and hypertensive subjects. American Heart Journal. 11, 1-9.

Hines, E. A., (1940). Range of normal blood pressure and subsequent development of hypertension: a follow-up of 1522 patients. Journal of the American Medical Association. 115. 271-278.

Jackson, A.S., Squires, W.G., & Beard, E.F., (1983). Prediction of hypertension from exercise blood pressure. Journal of Cardiac Rehabilitation, 3. 263-268.

Janz, K.F., Burns, T.L., & Mahoney, L.T., (1995). Predictors of left ventricular mass and resting blood pressure in children: the Muscatine Study. Medicine and Science in Sport and Exercise. 27(6), 818-825.

Jetté, M., Landry, F., & Sidney, K., (1991). Blood pressure response to the Canadian areobic fitness test. Canadian Journal of Public Health, 82, 267-276.

Julius, S., & Conway, J. (1968). Hemodynamic studies in patients with borderline blood pressure elevation. Circulation. 38, 282-288.

Julius, S., (1990). Changing role of the autonomic nervous system in human hypertension. Journal of Hypertension. 8(7): S59-S65.

Kasagi, F., Akahoshi, M., & Shimaoka, K., (1995). Relation between cold pressor test and development of hypertension based on a 28-year follow-up. Hypertension. 25(1), 71-76.

Liu, Y., (1994). Pressor response to isometric hand grip combined with foot immersion in cold water. Unpublished master's thesis, University of Ottawa, Ottawa, ON.

Lund-Johansen, P., (1988). Hemodynamics in essential hypertension at rest and during exercise- a 20-year follow-up study. Annals of Clinical Science. 48, 31-38.

Lund-Johansen, P., (1994). Newer thinking on the hemodynamics of hypertension. Current Opinion in Cardiology. 9, 505-511.

Mahoney, L.T., Clarke, W.R., Burns, T.L., & Lauer, R.M. (1991). Childhood predictors of high blood pressure. American Journal of Hypertension. 4, 608S-610S.

Mark, A.L., Victor, R.G., Nerhed, C., & Wallin, B.G. (1985). Microneurographic studies of sympathetic nerve responses to static exercise in humans. Circulation Research. 57 (3): 461-469.

Mitchell, J.H., Kaufman, M.P., & Iwamloto, G.A., (1983). the exercise pressor reflex: its cardiovascular effects, afferent mechanisms, and central pathway. Annual Review of Physiology. 45, 229-242.

Seals, D.R. (1990). Sympathetic activation during the cold pressor test: influence of stimulus area. Clinical Physiology, 10, 123-129.

Wilson, N. V., & Meyer, B. M., (1981). Early prediction of hypertension using exercise blood pressure. Preventitive Medicine. 10, 62-68.

Table 1. Mean age and physical characterists of subjects (± SD).

	Normotensives	Exercise Hypertensives
Age (yrs)	23.1 (± 1.2)	23.5 (± 2.3)
Height (cm)	179.6 (± 5.1)	174.7 (± 9.0)
Weight (kg)	72.5 (± 4.5)	72.8 (± 8.1)
BMI (wt/ht^2)	22.5 (± 1.7)	23.8 (± 1.7)
WHR	0.82 (± 0.04)	0.85 (± 0.03)
Sum of Skinfolds (mm)	25.8 (± 4.5)	29.0 (± 8.1)
% Body Fat	11.7 (± 2.03)	12.9 (± 2.9)
VO_2 Max (mL/kg·min)	58.2 (± 1.4)	55.6 (± 4.6)
MVC (kPa)	91.9 (± 12.7)	89.9 (± 14.2)
Work (watts/kg)	2.7 (± 0.2)	2.4 (± 0.7)

Table 2. Mean delta (Δ) blood pressure* and heart rate** response to the Canadian Aerobic Fitness Test (CAFT).

	Normotensives	Exercise Hypertensives
HR Rest (beats/min)	58.8 (± 6.8)	59.5 (± 7.8)
SBP Rest (mmHg)	123.8 (± 5.8)	124.8 (± 3.5)
DBP Rest (mmHg)	80.4 (± 3.5)	82.1 (± 5.1)
ΔSBP (Stage A) †	27.6 (± 9.5)	47.8 (± 11.0)
ΔDBP (Stage A)	0.6 (± 7.0)	6.8 (± 8.5)
ΔHR (Stage A) †	33.0 (± 13.3)	47.8 (± 11.1)
ΔSBP (Stage B) †	34.8 (± 7.0)	51.8 (± 10.3)
ΔDBP (Stage B) †	0.2 (± 7.7)	10.0 (± 11.1)
ΔHR (Stage B)	58.8 (± 16.1)	73.3 (± 16.1)
ΔSBP (Stage C) †	40.8 (± 4.4)	54.3 (± 3.2)
ΔDBP (Stage C)	0.6 (± 7.7)	8.9 (± 9.6)
ΔHR (Stage C) †	73.8 (± 8.7)	86.9 (± 17.0)

* Delta scores (Δ) were calculated by subtracting subjects resting blood pressure from the blood pressure taken 15-60 seconds following each stage.

** Delta scores (Δ) for heart rates were calculated by subtracting the subjects resting HR from the heart rate taken 5-15 seconds following each stage (converted to bpm).

† Indicates a significant difference ($p<.05$) found between groups.

Table 3. Mean delta scores* for the PWC 140, isometric hand grip and cold pressor tests.

	PWC 140		Isometric Hand Grip		Cold Pressor Test	
	Normotensive	Exercise Hypertensive	Normotensive	Exercise Hypertensive	Normotensive	Exercise Hypertensive
ΔSBP (mmHg)	96.2 (± 16.5)	88.2 (± 21.4)	40.3 (± 16.2)	32.5 (± 10.0)	44.1 (± 20.1)	49.6 (± 23.6)
ΔDBP (mmHg)	39.3 (± 13.2)	34.4 (± 12.5)	27.7 (± 8.2)	23.3 (± 7.6)	33.7 (± 17.7)	32.7 (± 15.0)
ΔMAP (mmHg)	50.3 (± 13.2)	46.5 (± 13.1)	32.7 (± 7.9)	27.5 (± 7.9)	36.8 (± 16.4)	37.8 (± 18.2)
ΔHR (beats/min)	75.2 (± 10.6)	77.8 (± 9.3)	21.9 (± 13.0)	17.5 (± 7.0)	† 16.0 (± 6.0)	† 27.5 (± 12.9)
ΔCO (L/min)	† 17.5 (± 4.8)	† 11.7 (± 3.7)	0.4 (± 0.7)	0.6 (± 0.8)	0.5 (± 0.5)	1.6 (± 1.7)
ΔSV (mL)	† 71.1 (± 19.3)	† 42.2 (± 18.6)	0.9 (± 8.7)	0.5 (± 4.6)	5.2 (± 10.5)	7.2 (± 12.6)
ΔEF (%)	15.0 (± 11.4)	16.2 (± 16.5)	3.5 (± 4.2)	3.8 (± 3.8)	1.3 (± 3.3)	5.3 (± 4.5)
ΔTSR (dynes-s-cm^{-5})	678.7 (± 232.2)	855.7 (± 228.3)	-315.0 (± 185.5)	-264.4 (± 247.8)	-341.8 (± 194.1)	-172.9 (± 503.0)

* Delta scores (Δ) were calculated by subtracting subjects resting value from the peak value achieved at exercise.

† Indicates a significant difference (p<.05) found between groups.

Table 4. Mean recovery delta scores* for the PWC 140, isometric hand grip and cold pressor tests.

	PWC 140		Isometric Hand Grip		Cold Pressor Test	
	Normotensive	Exercise Hypertensive	Normotensive	Exercise Hypertensive	Normotensive	Exercise Hypertensive
ΔSBP (mmHg)	19.3 (± 17.6)	12.5 (± 26.1)	12.1 (± 15.0)	0.5 (± 10.8)	5.2 (± 17.8)	7.5 (± 12.6)
ΔDBP (mmHg)	17.9 (± 19.8)	10.8 (± 16.3)	5.0 (± 7.6)	0.7 (± 8.7)	6.6 (± 12.2)	4.6 (± 5.6)
ΔMAP (mmHg)	18.2 (± 15.7)	11.1 (± 15.3)	6.8 (± 8.4)	1.2 (± 8.3)	7.0 (± 12.4)	5.7 (± 7.0)
ΔHR (beats min)	7.1 (± 12.3)	16.7 (± 7.2)	-0.3 (± 9.2)	-1.8 (± 6.3)	-8.7 (± 7.7)	-8.7 (± 9.1)
ΔCO (L min)	3.0 (± 1.5)	2.5 (± 5.6)	0.2 (± 0.6)	-0.2 (± 0.3)	-0.3 (± 0.7)	-0.3 (± -0.4)
ΔSV (mL)	29.3 (± 21.6)	16.0 (± 28.5)	0.9 (± 14.2)	1.0 (± 8.6)	-1.4 (± 14.0)	-0.5 (± 4.6)
ΔEF (%)	3.2 (± 11.4)	4.9 (± 6.4)	1.9 (± 3.0)	0.7 (± 1.9)	0.2 (± 3.6)	0.7 (± 3.2)
ΔTSR (dynes-s-cm^{-5})	-195.8 (± 106.5)	-327.2 (± 228.5)	55.5 (± 110.8)	68.6 (± 161.7)	140.6 (± 204.3)	221.3 (± 374.3)

* Delta scores (Δ) were calculated by subtracting subjects resting value from the average recovery value between 2:45 and 3:00 following exercise.

Table 5. Mean linear regression slopes for systolic (SBP) and diastolic (DBP) blood pressures on the cold pressor and isometric hand grip tests.

	Normotensives		Exercise Hypertensives	
	Slope	R^2	Slope	R^2
Cold Pressor Test				
SBP †	0.23	0.81	0.57	0.90
DBP †	0.19	0.91	0.35	0.94
Isometric Hand Grip				
SBP	0.18	0.73	0.22	0.80
DBP	0.18	0.89	0.19	0.90

† Indicates a significant difference ($p<.05$) found between the groups.

Table 6. Delta systolic blood pressure responses to the Canadian Aerobic Fitness Test (CAFT)[1], isometric hand grip (IHG)[2], and cold pressor tests (CPT)[3].

Subject	Group*	CAFT†	IHG†	CPT†
1	Normotense	24	**54**	**51**
2	Normotense	16	**46**	36
3	Normotense	26	42	**52**
4	Ex. Hypertense	**22**	**46**	16
5	Ex. Hypertense	**58**	28	**91**
6	Ex. Hypertense	**48**	27	47
7	Normotense	34	18	16
8	Normotense	20	**48**	35
9	Normotense	12	32	**52**
10	Normotense	32	24	**92**
11	Normotense	38	41	29
12	Ex. Hypertense	**56**	36	**68**
13	Ex. Hypertense	**48**	25	27
14	Normotense	40	**73**	**40**
15	Ex. Hypertense	**50**	24	**61**
16	Ex. Hypertense	**50**	24	46
17	Ex. Hypertense	**46**	**49**	42
18	Normotense	34	26	38

[1] An increase in systolic blood pressure ≥ 45 mmHg or diastolic blood pressure ≥ 10 mmhg in response to stage A of the CAFT was classified as an exaggerated response.

[2] An increase in systolic blood pressure greater than 45 mmHg in response to the IHG was classified as an exaggerated response.

[3] An increase in systolic blood pressure greater than 50 mmHg in response to the CPT was classified as an exaggerated response.

* Blood pressure classification according to the CAFT.

† Exaggerated responders in bold.

Figure 1. Mean systolic blood pressure response to the CAFT.

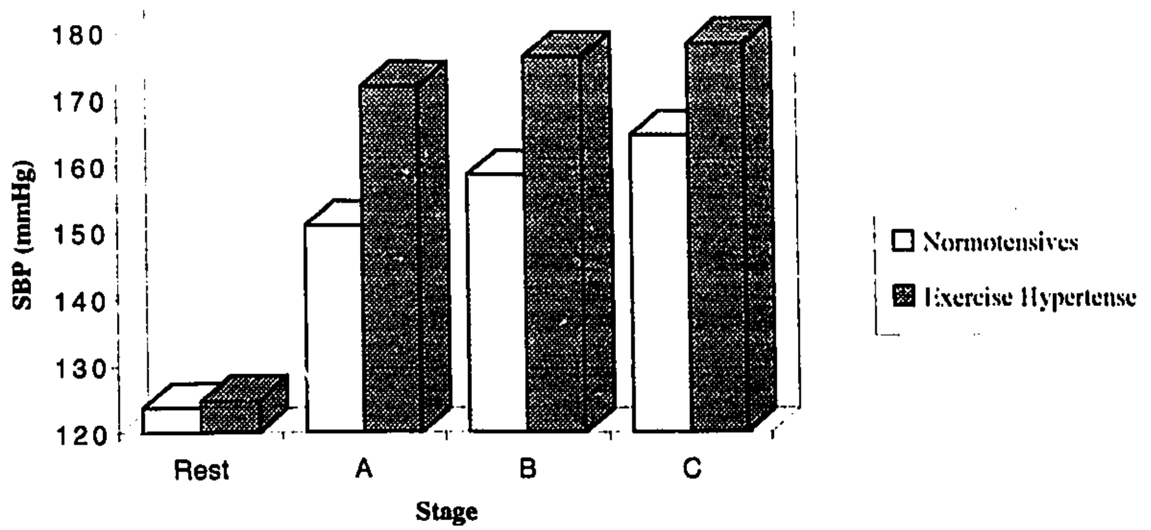


Figure 2. Mean diastolic blood pressure response to the CAFT.

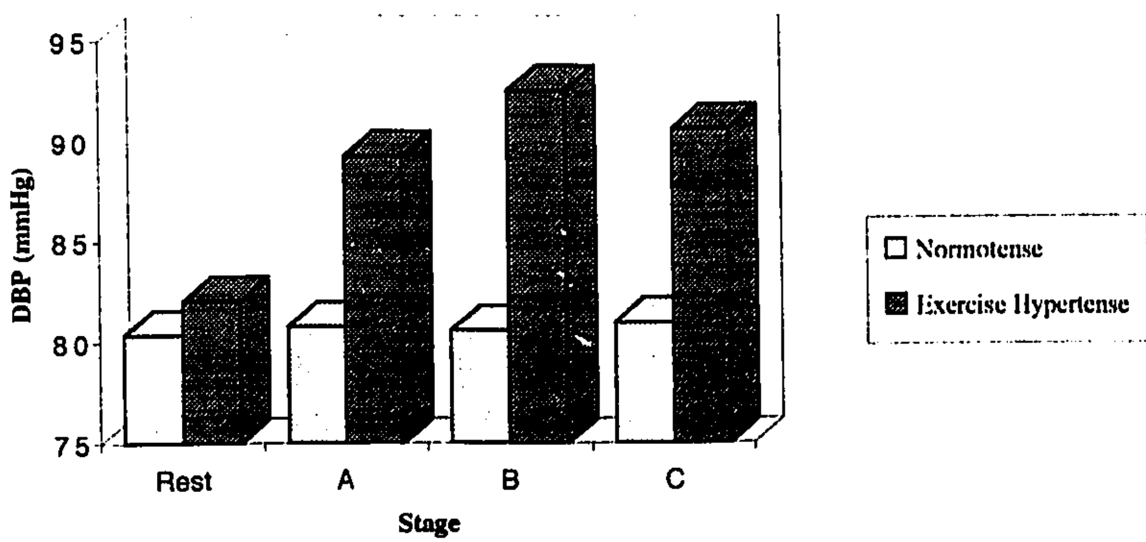
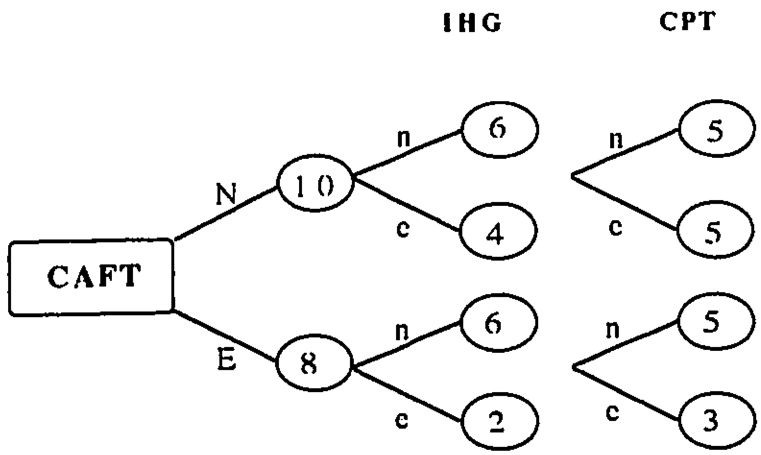


Figure 3. Systolic blood pressure response to the isometric hand grip (IHG), and cold pressor test (CPT), based on the blood pressure classification to the Canadian Aerobic Fitness Test (CAFT).



N= normal response to CAFT (Δ SBP < 45 mmHg).
E = exaggerated response to CAFT (Δ SBP > 45 mmHg).
n= normal response.
e= exaggerated response.

REVIEW OF LITERATURE

2.1 Introduction

The purpose of this study was to examine the hemodynamic response to three physical stressors in individuals who are normotensive at rest and exercise and individuals who are normotensive at rest but hypertensive at exercise. The following review will provide a background into: 1) the hemodynamics of essential hypertension; 2) the control of the cardiovascular system; 3) the cardiovascular response to the cold pressor test, isometric exercise, and dynamic exercise; 4) the use of these tests in the early detection of hypertension.

Hypertension was recognized as a specific disease around the beginning of the twentieth century, shortly after the invention of the sphygmomanometer. Since the discovery of hypertension there has been a vast amount of research directed towards this phenomenon in an attempt to understand it's hemodynamics and pathophysiology. This research has led to a greater understanding of the elements associated with the disease. However, many questions relating to it's cause still remain unknown.

In the late 1930's and early 1940's some landmark observations were made in relation to hypertension and external stressors. Hines and Brown (1936), found that individuals with hypertension exhibited an excessive blood pressure response to a cold stressor. As well, normotensives who showed an exaggerated response to the CPT were more likely to develop future hypertension (Hines, 1940). Shortly after, Levey et al., (1944), found acute emotional stress also elicited an excessive blood pressure response in hypertensive individuals. These early researchers opened the doors to various physical and emotional stressors as potential tests to identify individual who may be at an increased risk for the development of hypertension. Recently, much interest has been directed into the use of an exaggerated blood pressure response to exercise in normotensive individuals as an early predictor in the development of sustained hypertension (Wilson & Meyer, 1981; Jetté, Landry, Sydney, & Blumchen, 1988; Jetté, Landry, & Sydney, 1991).

2.2 Hemodynamics of Hypertension

Essential hypertension is generally referred to as a sustained arterial blood pressure increase of unknown cause. While there may be no direct known cause there are several characteristics that are common among hypertensives.

2.2.1 Early stages of hypertension

For researchers one of the biggest challenges in the study of essential hypertension is to identify a starting phase in the disease. The development of hypertension can begin at an early age. The earliest stage of hypertension is often said to begin in early childhood (Mahoney et al., 1991). This early stage is often characterized by a hyperkinetic circulation (Lund-Johansen, 1984). During this phase, cardiac index, heart rate and oxygen consumption are elevated whereas peripheral resistance is within normal values when compared to normotensives (Lund-Johansen 1988).

This pattern is believed to be a result of an increased sympathetic nervous activity (Lund-Johansen, 1987).

Schicken et al., (1986) investigated the mechanisms regulating resting blood pressure in 264 children (9-18 years). Individuals with the highest blood pressure had a higher heart rate and cardiac index when compared to children with the lowest blood pressure. The older children also had a lower cardiac output and increased peripheral resistance than the younger children. Through echocardiography, they observed an increased left ventricular mass in those individuals with the highest blood pressure. These observations led them to conclude that hypertension develops from a hyperkinetic state in early childhood to a state of lower cardiac output and increased peripheral resistance at an older age.

This early stage of hypertension is said to be a result of a hyperkinetic circulation. According to the concept of 'whole body autoregulation' the theory of a hyperkinetic circulation would result in an increased vascular resistance in order to protect the tissues from over perfusion, thereby maintaining BP when CO subsequently fell (Lund-Johansen, 1984, Conway, 1984). These studies, and others (Folkow 1982; Page & Mc Cubbin 1983) indicate that the increased blood pressure is likely due to medial hypertrophy of the arteriolar walls, resulting in an elevation of total peripheral resistance.

2.2.2 Established hypertension

Several studies have documented the hemodynamic profiles of established hypertensives in adults (>40 years) and elderly patients. The primary hemodynamic disturbance in these individuals is an increased total peripheral resistance as compared to normotensive individuals (Conway, 1984; Lund-Johansen, 1987). At this stage of hypertension there is a general decrease in the cardiac output and low to normal stroke volume when compared to normotensives or younger hypertensives (Lund-Johansen 1980). There is an increased wall to lumen ratio, caused by extensive medial vascular hypertrophy in developed hypertensives (Page & Mc Cubbin, 1983). Studies using echocardiography techniques have shown an increased left ventricular wall thickness (Hammond et al., 1986), resulting in decreased left ventricular filling time and compliance among hypertensives (Lund-Johansen, 1987). As hypertension progresses, patients generally exhibit an enormous increase in total peripheral resistance which can be more than three times greater than in normotensives (Conway, 1984).

2.2.3 Summary

The development of hypertension may begin at a very young age. Some but not all believe that early hypertensives are characterized by a hyperkinetic circulatory state. Typically in young hypertensives there is a high cardiac output and a low to normal total peripheral resistance. As the disease progresses, there is a shift from this hyperkinetic state to one of a lower than normal cardiac output to an increased total peripheral resistance which can be up to three times greater than in normotensives.

2.3 Cardiovascular Control

The cardiovascular system is a complex system comprised of two major components; the *heart*, and an intricate network of *blood vessels*. Together, these components work as a vehicle to deliver essential nutrients throughout the body. The control of this system is governed by a network of mechanisms that has the capability to adapt to constant changes and variations in an attempt to maintain homeostasis.

2.3.1 Control of heart rate

Heart rate is maintained spontaneously through the rhythmic discharge of the AS node, in the absence of nervous or hormonal influences. However, the sympathetic and parasympathetic nervous system play primary roles in the regulation of heart rate. In general it can be stated that an increase in sympathetic activity will cause an increase in heart rate and force of contraction, while an increase in parasympathetic activity will result in a decrease in the activity of the heart (Guyton, 1982).

Stimulation of the sympathetic nervous system results in the release of norepinepherine at the sympathetic nerve endings which are located around the heart with a concentration of sympathetic nerves located on the left ventricle. The release of norepinepherine results in an increased permeability of the fibbers to sodium and calcium resulting in an increased heart rate (Vander et al., 1975). Stimulation of the sympathetic nervous system has four main effects on heart rate. First, it increases the rate of S-A nodal discharge. Second, it increases the excitability and rate of conduction of the entire heart. Third, it increases the force and strength of the contraction up to 100 percent greater than normal. Finally, it increases the excitability of the A-V node, thus decreasing the time of conduction from the atria to the ventricles. (Guyton, 1982).

The stimulation of the parasympathetic nervous system has essentially the opposite effect. The parasympathetic nerves (vagi) are located primarily around the S-A and A-V nodes. An increase in parasympathetic nervous activity results in the release of acetylcholine at the vagal nerve endings. The result is a decrease in the excitability of the S-A node, causing reduction in heart rate, as well as a decrease in the activity in the A-V junctional fibbers which results in a reduced rate of cardiac impulse into the ventricles. At rest, the parasympathetic system is the dominant control mechanism up to a heart rate of about 100 bpm (Vander et al., 1975).

The hormonal influence in the control of heart rate is also very important. The primary hormone that influences heart rate is epinephrine. Elevated plasma levels of epinephrine secreted by the adrenal medulla causes an increased activity of the heart, that could last up to ten times longer than direct sympathetic stimulation due to its slow rate of removal from the blood stream (Vander, et al., 1975).

2.3.2 Control of stroke volume

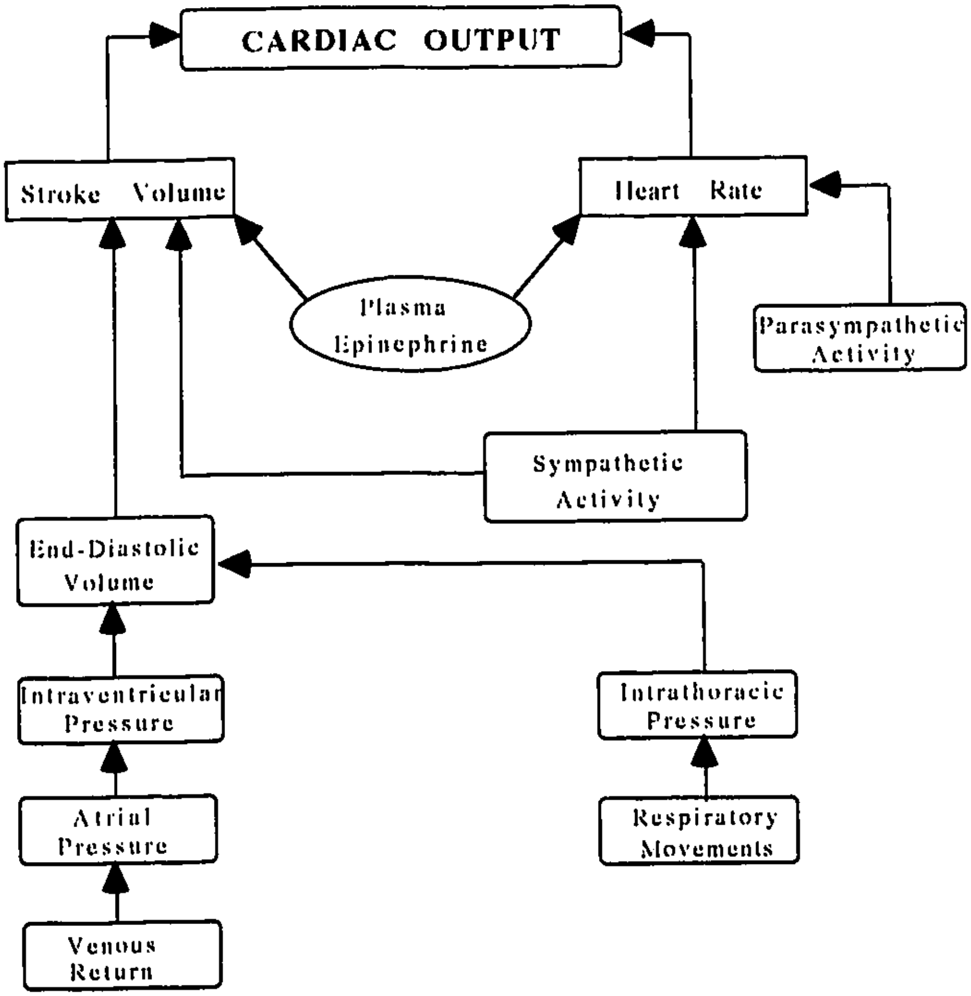
Stroke volume is mediated by both intrinsic (venous return) and extrinsic (sympathetic activity) control mechanisms. As venous return is increased more blood is pumped from the heart. The intrinsic control of stroke volume is dependent on it's ability to regulate the strength of ventricular contraction to adapt to the increase in blood returning to the heart. The result is that as venus return increases, end diastolic volume increases. This increase is causes an increase in the tension of the cardiac muscle which increases stroke volume on the subsequent cycle. The stroke volume ultimately increases so that stroke volume is equal to diastolic filling. This intrinsic mechanism is known as the Frank Starling Mechanism (Sherwood, 1995).

The extrinsic control of stroke volume is regulated by sympathetic activity. An increase in sympathetic activity or elevated levels of plasma epinephrine result in an increased contractility of the heart. Therefore, the heart is able to deliver a greater percentage of blood, resulting in a more complete ejection. Thus, although end diastolic volume remains the same stroke volume increases resulting in a lower end systolic volume and an increased ejection fraction. Sympathetic stimulation also results in vasoconstriction which increases venous return and therefore causes a further increase in stroke volume.

2.3.3 Control of cardiac output

Cardiac output is essentially the product of heart rate and stroke volume. Thus, any factors which may influence either heart rate or stroke volume will also have an effect on cardiac output. The factors which control cardiac output are best illustrated in Figure 1 which also includes those factors that influence both heart rate and stroke volume.

Figure 1. Major factors influencing cardiac output.



(Adapted from Vander et al., 1975, pg.247).

2.3.4 Control of blood pressure

Arterial blood pressure is an intricate system that is regulated by several interrelated mechanisms. The two major systems that regulate blood pressure can be divided into; 1) those mechanisms which are responsible for the short term regulation of blood pressure and; 2) those mechanisms that regulate the long term control of blood pressure.

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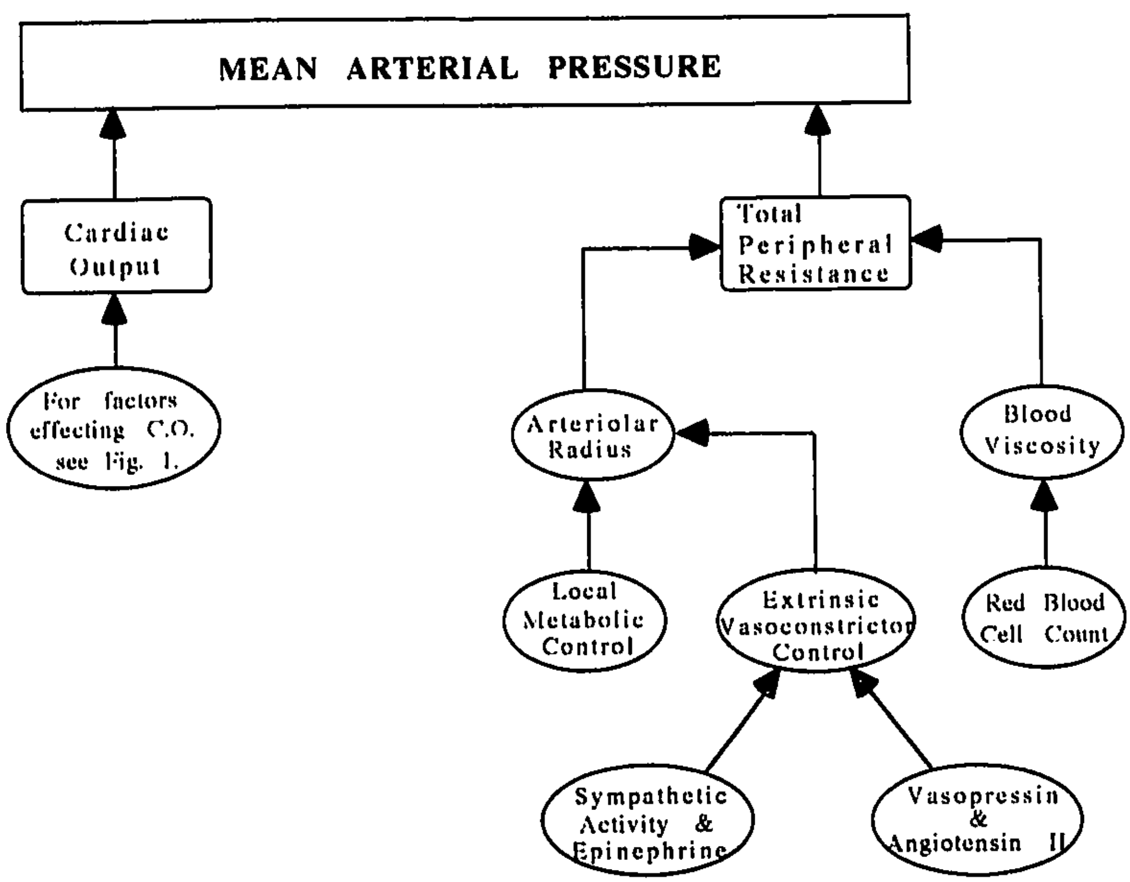
The first mechanism that is activated in the short term regulation of blood pressure is the baroreceptor reflex. Baroreceptors are fast acting spray type nerves that are located throughout walls of large systemic arteries, with the major regions located in the carotid sinuses and walls of the aortic arch (Guyton, 1982). Baroreceptors are pressure activated. When an increase in arterial pressure is detected by the baroreceptors, an excitation of baroreceptor impulses results. These baroreceptor impulse inhibit the vasoconstrictor center of the medulla and excite the vagal center resulting in vasodilatation and a decreased heart rate which decreases blood pressure back towards normal values. Thus, it is often said that the main function of the baroreceptor system is to reduce the daily variation in arterial pressure (Guyton, 1982).

Two hormonal mechanisms are also involved in the short term rapid control of arterial blood pressure. The norepinepherine/epinephrine vasoconstrictor mechanism causes excitation of the heart and constriction of most blood vessels resulting in an increased blood pressure (Vander et al., 1975). These release of these hormones act in a similar fashion of direct sympathetic stimulation. However, they can remain in the bloodstream for several minutes resulting in a prolonged effect.

There are several other factors that are important in the short term control of blood pressure. These include the renin-angiotensin system, atrial and pulmonary reflexes, vasopressin, salt/fluid imbalances etc. A summary of the factors that affect arterial blood pressure are illustrated in Figure 2.

The primary mechanism responsible for the long-term control of arterial pressure is the renal-body fluid system. The renal-body fluid system essentially controls the excretion of water and salt from the body via the kidneys. This controls the amount of extracellular fluid volume within the body, thereby controlling blood pressure through the manipulation of blood volume. This is a very powerful mechanism, aided by the renin-angiotensin system, aldosterone system, and sympathetic nervous system. It should be noted that this system only takes effect when the short term systems cannot adequately maintain blood pressure (Guyton, 1982).

Figure 2. Major factors influencing blood pressure.



(Adapted from Sherwood, 1995, pg. 266).

2.3.5 Summary

The regulation of heart rate, stroke volume, cardiac output, and blood pressure is a complex process that can be affected by many factors both intrinsically and extrinsically. Each of these functions are interrelated, therefore, a change in one has a direct impact on these other variables. This is especially evident when we examine the effect of physical stressors on the cardiovascular system.

2.4 Cardiovascular Response to Physical Stressors

Several changes occur in the cardiovascular system in response to physical stressors. Dynamic exercise, isometric exercise, and the cold pressor test each tax the cardiovascular system in a different manner. Given the different mechanisms that are involved, each of these tests can provide unique information regarding an individuals cardiovascular fitness. Although the mechanisms regulating the cardiovascular response to these tests are different, there may be some similarities that exist between an individuals response on these stressors which could provide some insight into the state of ones cardiovascular system. Thus, the combined results of each of these tests may provide a useful information for evaluating the early onset of hypertension.

2.4.1 Cold pressor test

The cold pressor test (CPT) has been used for several decades as a means of elevating blood pressures in the study of hypertension. The CPT involves the immersion of one of the extremities in ice water for a period of time typically ranging from 30 seconds to 3 minutes to elicit a pressor response. The CPT was first used by Hines and Brown (1936), in which they observed a dramatic increase in both the systolic and diastolic pressures of individuals, which they hypothesized to be a result of general vascular constriction initiated through a neurogenic reflex arc. There is an increase in sympathetic activity in response to the CPT. The initial response to the cold pressor test is cutaneous vasoconstriction, increased heart rate, and an increased blood pressure (Lovallo, 1975). It has been suggested that individuals who elicit a high response to the CPT may have a sympathetic predominance when exposed to stress whereas low responders have a parasympathetic predominance (LeBlanc, 1975).

Heart Rate

There is a gradual increase in heart rate following exposure to the cold pressor test (Seals, 1990). This increase is greatest between 60 to 80 seconds following immersion, after which heart rate tends to remain constant (Zbrozyna & Krebble, 1985), or decrease slightly (Greene et al., 1965). The increases in heart rate are due to increased levels of plasma epinephrine (LeBlanc, 1979).

Stroke Volume

There is little change in stroke volume in response to the CPT. Stroke volume tends to remain constant or decrease slightly over prolonged localized cold exposure (Greene, 1965).

Cardiac Output

The CPT elicits an increase in cardiac output in response to the increased heart rate. This increase may be minimal if the increase in heart rate is coupled with the decreased stroke volume often associated with the CPT. The increases in cardiac output usually peak by the first minute and level off during prolonged exposure (Greene, 1965).

Blood Pressure

Increases in both systolic and diastolic blood pressure are observed following exposure to the CPT (Hines & Brown, 1936; Godden et al., 1955; Greene, 1965; LeBlanc et al., 1975). Observed increases in both systolic and diastolic pressures have ranged from 10mmHg in normotensics (Thomas et al., 1982), to as much as in 40 mmHg in hypertensives (Hines & Brown 1936). The increase in blood pressure in response to the cold pressor test is primarily a result of increased levels of norepinepherine causing widespread vasoconstriction (LeBlanc et al., 1979). It has been suggested that the increases in the muscle sympathetic nervous activity (MSNA) are responsible for the increases in blood pressure (Fagius et al., 1989). However, the extent to which MSNA effects blood pressure may be influenced by the size of the area that is exposed to the cold stimulus.

Total Peripheral Resistance

Exposure to the CPT induces widespread neural stimulation causing peripheral vasoconstriction resulting in a slight elevation in total peripheral resistance (Green, et al., 1965).

Use as a Predictor of Hypertension

The early work of Hines and Brown (1936), provided the first evidence for the use of the cold pressor test as a possible predictor of hypertension. They observed an increased blood pressure response to the CPT in hypertensives, and in some normotensives they classified as 'hyperreactors'. They hypothesized that these 'hyperreactors' were in a prehypertensive state and would likely develop hypertension in the future. This hypothesis was later confirmed in a follow-up study (Hines, 1940).

This innovative work on the CPT by Hines helped pave the way for much research in this area. Several studies have since examined the usefulness of the CPT in the prediction of hypertension. Wood et al., (1984), found that 71% of those who showed a hyperreactive response to the CPT later developed hypertension, compared to only 19% of the normal responders. This finding suggested that there may be a relationship between an exaggerated BP response to the CPT and the future development of hypertension.

A recent study by Kasagi et al., (1995) further supports this hypothesis. Eight hundred twenty-four normotensive individuals were exposed to the CPT during a 28 year prospective study. The relative risk for hyperreactors was 1.37 when compared to the normal reactors. Systolic blood pressure during the CPT was found to be an independent and significant predictor. Thus, they concluded that those individuals who were classified as hyperreactors to the CPT were more likely to develop sustained hypretension. However, the effectiveness of the CPT as a predictor of future hypertension is not agreed upon by all (Greene, 1965; Thomas & Duszynski, 1982).

2.4.2 Isometric exercise

Isometric exercise involves primarily changes in muscle tension with no change in muscle length. An exercise pressor response results in a rise in both systolic and diastolic pressures in response to isometric exercise that increases with the magnitude of and duration of the contraction. This pressor response can be observed in sustained contractions as low as 15-20% MVC (Lind et al., 1964). Both hypertensives and normotensives, exhibit a pressor response with a greater increase typically seen among hypertensives (Laird et al., 1979).

Three possible mechanisms were proposed as possible explanations for the cardiovascular response to the isometric hand grip: 1) humoral, 2) central drive 3) reflex activity. However, it appears that peripheral reflex activity is the mechanism involved in the pressor response (Shepherd et al., 1981). Contraction of skeletal muscle during isometric exercise causes changes in the efferent sympathetic and parasympathetic outputs to the cardiovascular system that are in turn responsible for increases in arterial blood pressure, heart rate, contractile state of the left ventricle, cardiac output and changes in the distribution of blood flow reflexly induced from contracting skeletal muscle (Mitchell, 1983). Evidence also suggests that the reflex activity originates in the exercising muscle due to the release of anaerobic muscle metabolites, primarily potassium, which as a result alters cardiovascular function (Shepherd et al., 1981). The increased cardiac output appears to be the result of an increased heart rate with little or no change in stroke volume (Palatini, 1988). As well, there appears to be little change in total peripheral resistance (Perez-Gonzales et al., 1981) in response to isometric exercise.

Heart Rate

During isometric exercise there is an immediate increase in heart rate to accommodate the demands of the working muscle. The initial increase in heart rate during isometric exercise is due primarily to vagal withdrawal, without an increase in muscle sympathetic nervous activity (Freyschuss, 1970). The magnitude of the increase at the onset of contraction varies with the percentage of MVC (Shepherd et al., 1981). The increase in HR is linear and appears to level off within 2 minutes of the onset of exercise (Asmussen, 1981).

Stroke Volume

During isometric exercise, there is little or no change in stroke volume possibly due to the fact that there is no change in total peripheral resistance and therefore no change in the amount of venous blood returning to the heart (Laird et al., 1979). Stroke volume has been reported to remain constant at moderate intensities (30%) (Laird et al., 1979), but may decrease slightly at higher intensities (50%) (Lind et al., 1981).

Cardiac Output

Since there is little or no change in stroke volume, any changes in cardiac output during isometric exercise are a result of the heart rate response. Cardiac output increases with isometric exercise, mediated by the increase in heart rate (Palatini, 1994). The increase in cardiac output is primarily directed through non-working muscle and body parts, since the pathway to the working muscle is partially blocked by a localized increase in resistance (Asmussen, 1981).

Blood Pressure

Isometric exercise elicits an increase in both systolic and diastolic blood pressure (Asmussen, 1981). A restriction in blood flow to the working muscle as a result of the muscular contraction causes an increase in blood pressure in an attempt to maintain the blood supply by overcoming the intramuscular pressure on the local vessels (Lind et al., 1967). Since total peripheral resistance remains primarily unchanged during isometric exercise, the changes in blood pressure is primarily a result of an increase in cardiac output (Laird et al., 1979). Blood pressure increases linearly with isometric exercise and is affected by the magnitude and duration of the contraction. At moderate intensities (30% MVC), blood pressure appears to peak by the second minute of exercise (Jones et al., 1986).

Total Peripheral Resistance

Total peripheral resistance remains unchanged during isometric exercise (Laird et al., 1979; Pollock and Schmidt, 1979). Although some researchers have reported localized increases in peripheral resistance (Saito et al., 1990), reflex vasodilatation from baroreceptor stimulation maintains total peripheral resistance (Martin et al., 1974).

Use as a Predictor of Hypertension

The isometric hand grip has been used for several years in the diagnosis of cardiac performance and in the diagnosis of hypertension. A study by Cantor et al., (1987) examined the usefulness of the hand grip in comparison to dynamic exercise in the diagnosis of hypertension in 140 males and 10 females. Their hypothesis was that the hand grip could evaluate the same parameters as dynamic exercise yet is much easier and less expensive to administer. Of the 150 subjects involved 62 were found to be normotensive and 88 were borderline hypertensive. Following a 90 second isometric hand grip test at 30% MVC, the borderline hypertensives had a diastolic pressure 10 mmHg higher than the normotensives. However, there was no significant difference in the diastolic pressures following dynamic exercise (Bruce Protocol). The authors concluded that the hand grip was more effective in evaluating diastolic blood pressure than was dynamic exercise.

Similar results were noted by Chaney and Eyman, (1988). Resting blood pressures, and blood pressures during isometric and dynamic work were evaluated to determine the best predictor of hypertension within a 14-year follow up. The isometric hand grip performed at 30% MVC, when combined with resting measures was able to predict 88% of the hypertensives and 80% of the normotensives.

2.4.3 Dynamic exercise

During dynamic exercise, there is increased oxygen to the working muscles, an increased need to dispose of waste products, and to regulate temperature, resulting in widespread vasodilatation to increase blood flow to the muscles. Vasoconstriction of the non-working muscles and the splanchnic bed occurs simultaneously. The result is an increased venous return thereby increasing stroke volume. This increased stroke volume, accompanied by an increased heart rate produces an increased cardiac output (Palatini, 1988). The combination of these factors produces a change in blood pressure which is generally characterized by a rise in systolic pressure with little or no change in diastolic pressure. The magnitude of these changes is dependent on 1) the intensity of the exercise; 2) the individuals cardiovascular characteristics (i.e.. level of fitness).

Heart Rate

Heart rate increases dramatically at the onset of dynamic exercise and plateaus as the oxygen demands of the working muscles are met. Initial increases in heart rate are primarily a result of a decreased parasympathetic activity or vagal withdrawal (Rowell, 1986). Later increases in heart rate (above 100 bpm) have been associated with increases in sympathetic activity (Rowell, 1986). Thus, vagal withdrawal appears to be significant at lower exercise intensities and during the early stages of intense dynamic exercise, whereas increases in sympathetic activity are primarily responsible for greater increases in heart rate at higher exercise levels (Rowell, 1986).

Stroke Volume

Stroke volume is determined by the rate at which blood is returned to the heart through venous return (Patton et al., 1989). Stroke volume increases sharply at the onset of exercise, due to an increase in venous return. Stroke volume increases linearly with oxygen uptake until about 40% of V02 max. is reached, after which stroke volume increases only slightly (Åstrand & Rodahl 1970).

Cardiac Output

The onset of dynamic exercise is characterized by a rapid increase in cardiac output. Cardiac output continues to rise during dynamic exercise until a plateau or steady state is achieved. Cardiac output increases linearly with the oxygen demands of the body and will plateau once the oxygen demands of the working muscle are met. Cardiac output during dynamic exercise can increase from a resting level of about 5 l/min, to levels as high as 35 l/min. in trained individuals (Powers & Howley, 1990).

Total Peripheral Resistance

Initially there is little or no change in total peripheral resistance during dynamic exercise. As exercise progresses, an increase in sympathetic activity results in vasoconstriction of the non-working muscles and viscera. Within the working muscles, withdrawal of sympathetic outflow to arterioles results in local vasodilatation. As exercise progresses, this vasodilatation is maintained and increased through intrinsic metabolic control (autoregulation). The overall result may be a slight decrease in peripheral resistance to accommodate the oxygen demands of the working muscles (Powers & Howley, 1990).

Blood Pressure

The increased sympathetic activity during dynamic exercise results in mass vasoconstriction in the non-working muscles and thus, an increase in blood pressure to accommodate the oxygen demands of the working muscles (Palatini, 1988). Although there may be a slight decrease in overall peripheral resistance and a decrease in diastolic pressure during dynamic exercise, the increase in cardiac output results in an increase in systolic pressure well above resting levels. Systolic blood pressure increases as exercise intensity increases until oxygen demands are met.

Use as a Predictor of Hypertension

The use of dynamic exercise in the prediction of hypertension is well documented. Many (Wilson and Meyer, 1981; Jackson et al., 1983; Dlin et al., 1983; Tanji et al., 1989) have found the blood pressure response to dynamic exercise to be a strong predictor of future hypertension, while others (Fixlar et al., 1985; Goble and Schieken, 1991) have not had the same success.

Research by Wilson and Meyer (1981), provided evidence for the use of dynamic exercise blood pressures in the prediction of hypertension. Subjects (3395 males and 425 females) were exposed to dynamic exercise (Balke treadmill) administered over a 32 month period. Results indicated that the relative risk of developing sustained hypertension in individuals who were normotensive at rest but hypertensive at exercise was 2.17 for men and 3.33 for women when compared to individuals who were normotensive at both rest and exercise. They concluded that exercise blood pressure is a strong predictor for the later development of sustained hypertension.

These findings have since been supported in several similar studies. Jackson et al., (1983), retrospective study on 4856 individuals found the development of hypertension to be greater in those who were exaggerated responders to exercise (52%) when compared to normal responders (15%). Dlin et al., (1983) also found an increased incidence of hypertension in those with exaggerated blood pressure responses to exercise. Following subjects over an average of 5.8 years, they observed 10.6% of the exaggerated responders develop hypertension while none of the controls developed hypertension over the same period.

Recently, Tanji et al., (1989), examined the blood pressure response to dynamic exercise using the Harvard Step Test. Ninety-one percent of the individuals diagnosed as exercise hypertense developed sustained hypertension 10 years later. Only 1 of the 15 individuals who were normotensive at exercise developed hypertension over that same period. As such, it appears that an exaggerated blood pressure response to dynamic exercise may represent an early stage in the development of hypertension, and may provide a means for the early identification of individuals predisposed to hypertension.

2.4.4 Canadian Aerobic Fitness Test

Recently researchers have examined the blood pressure response to the Canadian Aerobic Fitness Test (CAFT) (Jetté et al., 1988; Jetté et al., 1991). The CAFT is a standardized sub-maximal test that consists of a stepping sequence performed on two 20.3 cm steps in cadence with a six-count musical rhythm that increases in tempo as the stages progress. Blood pressures (measured by sphygmomanometer) and heart rate are taken following each stage. There are a possible three stages, with post exercise heart rates determining whether or not individuals proceed to subsequent stages.

In a study by Jetté et al. (1991), normative data was established to identify individuals who have an abnormal response to the CAFT. Nine hundred eighty-six men and women between the ages of 20-69 were examined. Criteria for inclusion in this study were blood pressures <150/100 mmHg for males, and <160/100 mmHg for females. They concluded that an increase in systolic blood pressure of 45 mmHg (+1 standard deviation) or 10 mmHg diastolic in response to the first stage of the CAFT constituted an exaggerated response. Using this criteria, as many as 500 000 Canadians could be identified as exercise hypertense (Jetté et al., 1988).

Individuals with an exaggerated blood pressure response to exercise may be undergoing some of the cardiovascular changes associated with hypertension and have a 2-10 times increased risk of developing future sustained hypertension (Wilson & Meyer 1981; Davidoff et al., 1982; Jackson et al., 1983). Thus, the CAFT could be effective in identifying individuals who may be at risk, yet were not identified as such through resting measures alone.

2.4.5 Summary

The CPT, isometric exercise and dynamic exercise have each been shown to induce a pressor response, although the mechanisms that are involved appear to be different. However, one would expect that individuals who show a specific response to one of these stressors would also show a similar response to the other stressors.

There appears to be some controversy over the usefulness of exercise blood pressure in the prediction of future hypertension. Although many researchers have indicated an exaggerated blood pressure response to exercise to be a strong predictor for future hypertension (Hines, 1940; Wilson & Meyer, 1981; Jackson et al., 1983; Dlin et al., 1983; Chaney & Eyman, 1988; Tanji et al., 1989; Jetté 1991) others have not (Greene, 1965; Thomas & Duszynski, 1982; Fixlar et al., 1985; Goble & Schieken, 1991). However, it appears that exercise blood pressure measurements may indicate the possibility of prehypertensive changes that may be in progress long before they can be detected using resting measures. With further research, exercise hypertension may provide a useful tool in the early detection of hypertension.

REFERENCES

Åstrand, P.O., Rodahl, K., (1970). Textbook of Work Physiology. Toronto: McGraw-Hill Book Company.

Asmussen, E. (1981). Similarities and dissimilarities between static and dynamic exercise. Circulation Research, 48(6), I3-I10

Canadian Blood Pressure Survey. (1989). Department of Health and Welfare Canada. Ottawa: Minister of National Health and Welfare.

Canadian Standardized Test of Fitness (3rd ed.). (1986). Ottawa: Fitness and Amateur Sport.

Cantor, A., Gold, B., Gueron, M., Cristal, N., Prajgrod, G., & Shapiro, Y., (1987). Isotonic (dynamic) and isometric (static) effort in the assessment and evaluation of diastolic hypertension: correlation and clinical use. Cardiology. 74, 141-146.

Chaney, R. H., & Eyman, R. K., (1988). Blood pressure at rest and during maximal dynamic and isometric exercise as predictors of systemic hypertension. The American Journal of Cardiology. 62. 1058-1061.

Conway, J., (1984). Hemodynamic aspects of essential hypertension in humans. Physiological Reviews. 64(2), 617-660.

Davidoff, R., Schamroth, C.L., Goldman, A.P., Diamond, T.H., Cilliers, A.J., & Myburg, D.P., (1982). Postexercise blood pressure as a predictor of hypertension. Avation Space and Environmental Medicine. 53 (6), 591-594.

Dlin, R.A., Hanne, N., Silverberg, D.S., & Bar-Or, O., (1983). Follow up of normotensive men with exaggerated blood pressure response to exercise. American Heart Journal. 106, 316-320.

Fagius, J., Karhuvaara, S. & Sundlof, G., (1989). The cold pressor test: effects on sympathetic nerve activity in human muscle skin nerve fascicles. Acta Physiology Scand, 137: 324-334.

Fixlar, D.E., Laird, W.P., & Dana, K. (1985). Usefulness of exercise stress testing gor prediction of blood pressure trends. Pediatrics. 75(6). 1071-1075.

Foder, G., (1980). L'hypertension au Canada: mise a jour. Maladies Chroniques au Canada, 1, 9-10.

Folkow, B., Physiological aspects of primary hypertension. Physiology Reviews. 27, 19-22.

Freyschuss, U. (1970). Elicitation of heart rate and blood pressure increase on muscle contraction. Journal of Applied Physiology, 28(6), 758-761.

Goble, M.M. & Schieken, R.M. (1991). Blood pressure response to exercise: A marker for future hypertension? American Journal of Hypertension. 4, 617S-620S.

Godden, J.E., Roth, G.M., & Hines, E.A., (1955). The changes in the intra-arterial pressure during immersion of the hand in ice-water. Circulation. 12, 963-973.

Guyton, A.C., (1982). Human Physiology and Mechanisms of Disease. Toronto: W.B. Saunders Company.

Hammond, I.W., Devereux, P.B., Alderman, M.H., & Laragh, J.H. (1986). The prevelance and correlates of exocardiographic left ventricular hypertrophy among employed patients with uncomplicated hypertension. Journal of the American College of Cardiology. 7, 639-650.

Hines, E.A., & Brown, G.E., (1936). The cold-pressor test for measuring reactability of the blood pressure: data conserning 571 normal and hypertensive subjects. American Heart Journal. 11, 1-9.

Hines, E. A., (1940). Range of normal blood pressure and subsequent development of hypertension: a follow-up of 1522 patients. Journal of the American Medical Association. 115. 271-278.

Jones, R., Lahiri, A., Cashmin, P.M.M., Dore, C., Raftery, E.B., (1986). Left ventricular function during isometric hand grip and cold stress in normal subjects. British Heart Journal. 55, 246-252.

Jackson, A.S., Squires, W.G., & Beard, E.F., (1983). Prediction of hypertension from exercise blood pressure. Journal of Cardiac Rehabilitation, 3. 263-268.

Jetté, M., Landry, F., Sidney, K., & Blumchen, G. (1988). Exaggerated blood pressure response to exercise in the detection of hypertension. Journal of Cardiopulmonary Rehabilitation. 8, 171-177.

Jetté, M., Landry, F., & Sidney, K., (1991). Blood pressure response to the Canadian areobic fitness test. Canadian Journal of Public Health, 82, 267-276.

Kaplan, N.M., (1986). Clinical Hypertension, 4th ed. Baltimore: Williams & Wilkins.

Kaplan, N.M., (1983). Hypertension. In: Kaplan, N.M., & Stannler, J. (eds), Prevention of Coronary Heart Disease. Practical Management of Risk Factors. Philedelphia: WB Saunders.

Laird, W.P., Fixler, D.E., & Huffines, F.D., (1979). Cardiovascular response to isometric exercise in normal adolescents. Circulation. 59, 651-654.

LeBlanc, J., Côté, J., Jobin, M., & Labrie, A. (1979). Plasma catecholamine and cardiovascular responses to cold and mental activity. Journal of Applied Physiology: Respiration and Environmental Exercise Physiology, 47(6), 1207-1211.

Levey, R. L., Hillman, C. C., Stroud, W. D., & White, P. D., (1944). Transient hypertension: its significance in terms of later development of sustained hypertension and cardiovascular-renal diseases. Journal of the American Medical Association. 126. 829-833.

Lind, A.R., Taylor, S.H., Humphreys, P.W., Kennelly, B.M., and Donald, K.W. (1964). The circulatory effects of sustained voluntary muscle contractions. Clinical Science. 27, 229-244.

Lind, A.R., & McNicol,G.W. (1967). Circulatory responses to sustained hand-grip contraction performed during other exercise both rhythmic and static. Journal of Physiology. 192, 595-607.

Lind, A.R., Dahms, T.E.., Williams, C.A., and Petrofsky, J.S. (1981). The blood flow through the "resting" arm during hand-grip contractions. Circulation Research. 48(6), 1104-1109.

Lovallo, W. (1975). The cold pressor test and autonomic function: A review and integration. Psychophysiology. 12(3), 269-282.

Lund-Johansen, P. (1980). State of the art review. Hemodynamics in hypertension. Clinical Science. 59, 343S-354S.

Lund-Johansen, P., (1984). Hemodynamic concepts in essential Hypertension. Triangle. 23(1), 13-23.

Lund-Johansen, P., (1987). Hemodynamics in hypertension at rest and during Exercise. Journal of Cardiovascular Pharmacology. 10(suppl.11) , 51-55.

Lund-Johansen, P., (1988). Hemodynamics in essential hypertension at rest and during exercise- a 20-year follow-up study. Annals of Clinical Science. 48, 31-38.

Mahoney, L.T., Clarke, W.R., Burns, T.L., & Lauer, R.M. (1991). Childhood predictors of high blood pressure. American Journal of Hypertension. 4, 608S-610S.

Martin, C.E., Shaver, J.A., Leon, D.F., Thompson, M.E., Reddy, P.B., and Leonard, J.J. (1974). Autonomic mechanisms in hemodynamic responses to isometric exercise. Journal of Clinical Investigation. 54, 104-115

Menkes, M.S., Matthews, K.A., Krantx, D.S., Lundberg, U., Mead, L.A., Qaqish, B., Liang, K.R., Thomas, C.B., & Pearson,T.A., (1989). Cardiovascular reactivity to the cold pressor test as a predictor of hypertension. Hypertension. 14(5), 524-530.

Mitchell, J.H., Kaufman, M.P., & Iwamloto, G.A., (1983). the exercise pressor reflex: its cardiovascular effects, afferent mechanisms, and central pathway. Annual Review of Physiology. 45, 229-242.

Ohlsson, O., & Henningsen, N.C., (1982). Blood pressure, cardiac output and systemic vascular resistance during rest, muscle work, cold pressure test and psychological stress. Acta Medical Scandinava. 212, 329-336.

Page, I.H., & Mc Cubbin, J.W., (1983). The physiology of arterial hypertension. In: Handbook of Physiology and Circulation. Washington D.C.: American Physiological Society.

Palatini, P. (1988). Blood pressure behaviour during physical activity. Sports Medicine. 5, 353-374.

Palatini, P. (1994). Exercise haemodynamics in the normotensive and the hypertensive subject. Clinical Science. 87, 275-287.

Patton, H.D., Fuchs, A.F., Hille, B., Scher, A.M., & Stuner, R. (1989). Textbook of Physiology. Philadelphia : W.B. Saunders company.

Perez-Gonzales, J.F., Schiller, N.B., & Parmley, W.W., (1981). Direct and noninvasive evaluation of cardiovascular response to isometric exercise. Circulation Research. 48(6), 1138-1148.

Pollock, M.L., & Schmidt, D.H., (1979). Response of patients with heart disease to dynamic and static exercise: In: Heart Disease and Rehabilitation. Toronto: John Wiley & Sons.

Powers, S.K., & Howley, E.T., (1990). Exercise Physiology : Theory and Application to Fitness and Performance. Dubuque: Wm. C. Brown Publishers.

Rowell, L.B. (1986). Human Circulation: Regulation During Physical Stress. New York: Oxford University Press.

Saito, M., Mano, T., & Iwase, S. (1990). Change in muscle sympathetic nerve activity and calf blood flow during static handgrip exercise. European Journal of Applied Physiology, 60, 277-281.

Schieken, R.M., Lauer, R.M., Clarke, W.R., (1986). Hemodynamics in childhood hypertension. Clinical and Experimental Hypertension-Theory and Practice. A8(4&5), 703-720.

Seals, D.R. (1990). Sympathetic activation during the cold pressor test: influence of stimulus area. Clinical Physiology, 10, 123-129.

Sherwood, L., (1995). Fundamentals of Physiology-A Human Perspective. New York: West Publishing Company.

Tanji, J.L., Champlin, J.J., Wong, G.Y., Lew, E.Y., Brown, T.C., & Amsterdam, E.A., (1989). Blood pressure recovery curves after submaximal exercise. American Journal of Hypertension. 2, 135-138.

Thomas, C.B., & Dusxinski, K.R., (1982). Blood pressure levels in young adulthood as predictors of hypertension and the fate of the cold pressor test. Johns Hopkins Medical Journal. 151, 93-100.

Vander, A.J., Sherman, J.H., & Luciano, D.D., (1975). Human Physiology-The Mechanisms of Body Function. Toronto: Mc Graw Hill.

Wilson, N. V., & Meyer, B. M., (1981). Early prediction of hypertension using exercise blood pressure. Preventitive Medicine. 10, 62-68.

Wood, D.L., Sheps, S.G., Elveback, J.R., & Schirger, A. (1984). Cold pressor test as a predictor of hypertension. Hypertension. 6, 301- 306.

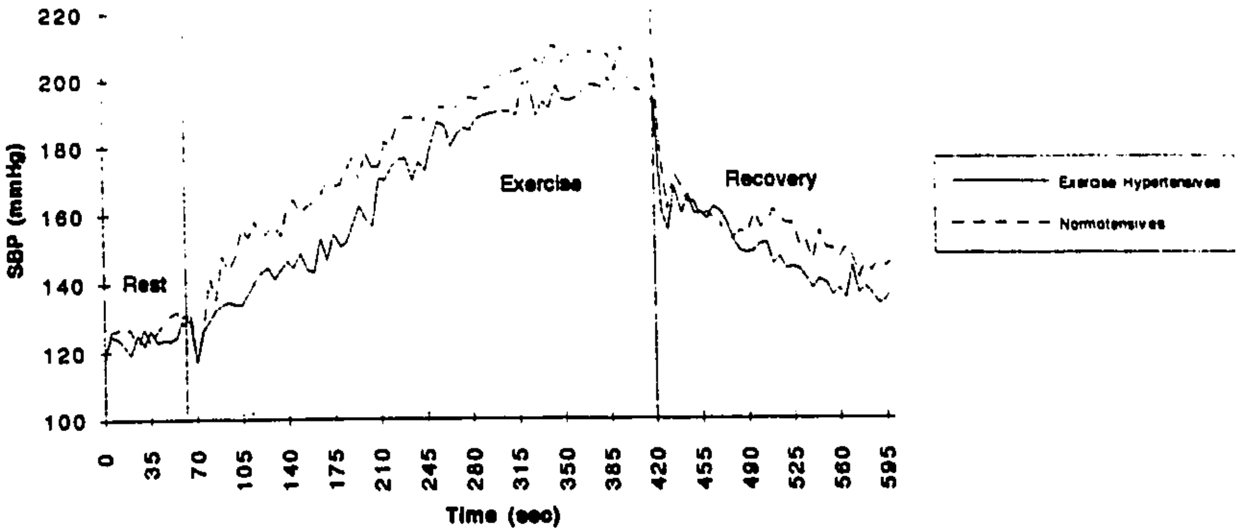
Zbrozyna, A.W., & Krebble, F. (1985). Habituation of the cold pressor response in normo- and hypertensive human subjects. European Journal of Applied Physiology, 54, 136-144.

APPENDICES

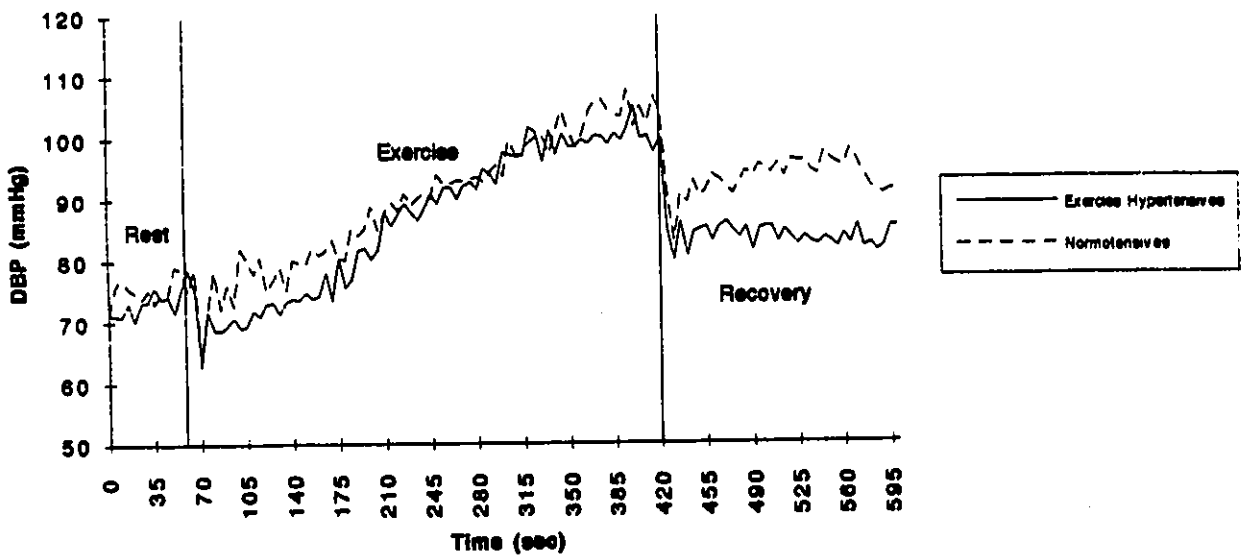
APPENDIX 1

MEAN CARDIOVASCULAR RESPONSE TO THE PWC 140

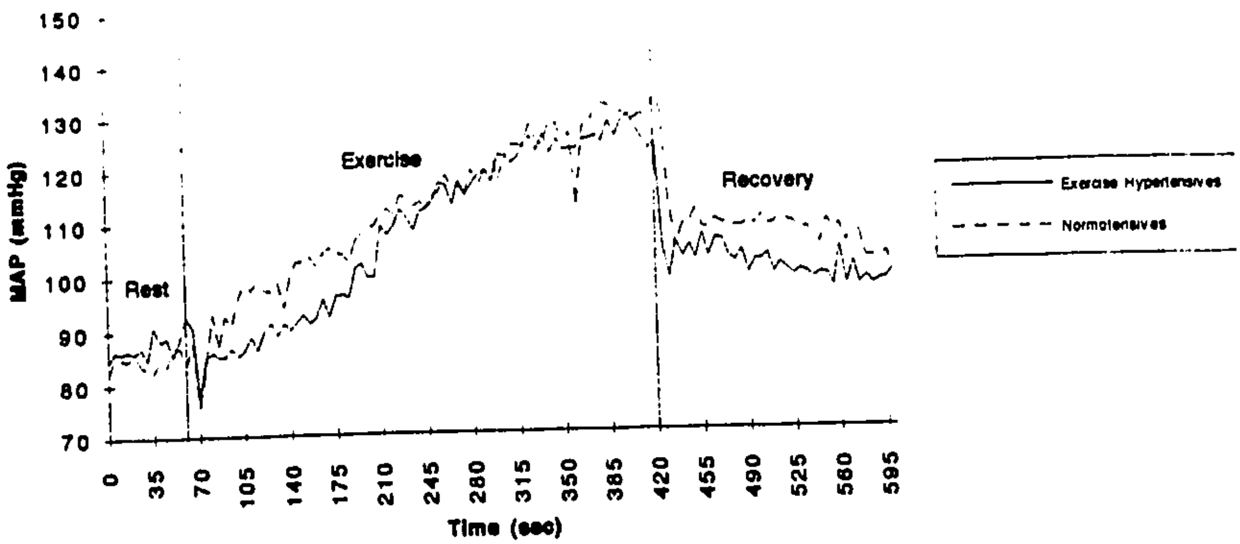
Appendix 1(a). Mean systolic blood pressure response to the PWC 140



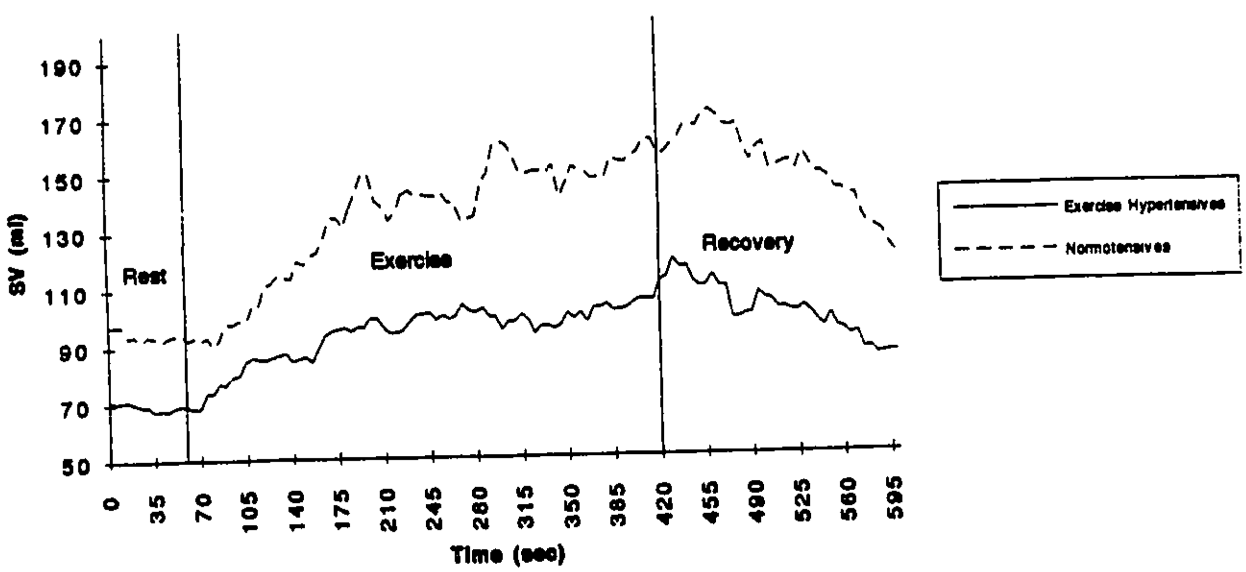
Appendix 1(b). Mean diastolic blood pressure response to the PWC 140



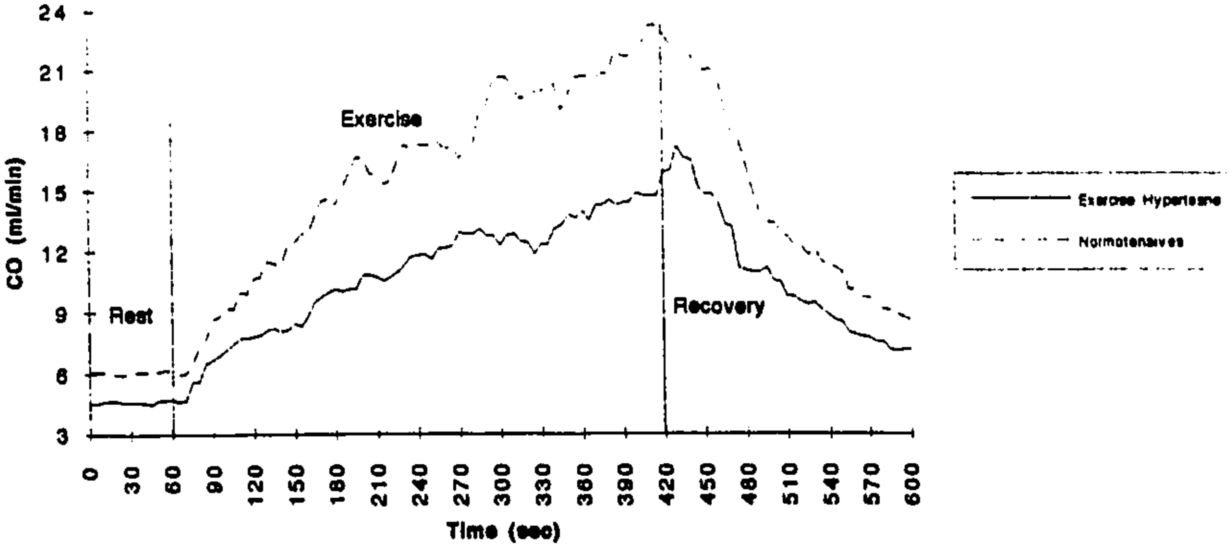
Appendix 1(c). Mean arterial blood pressure response to the PWC 140



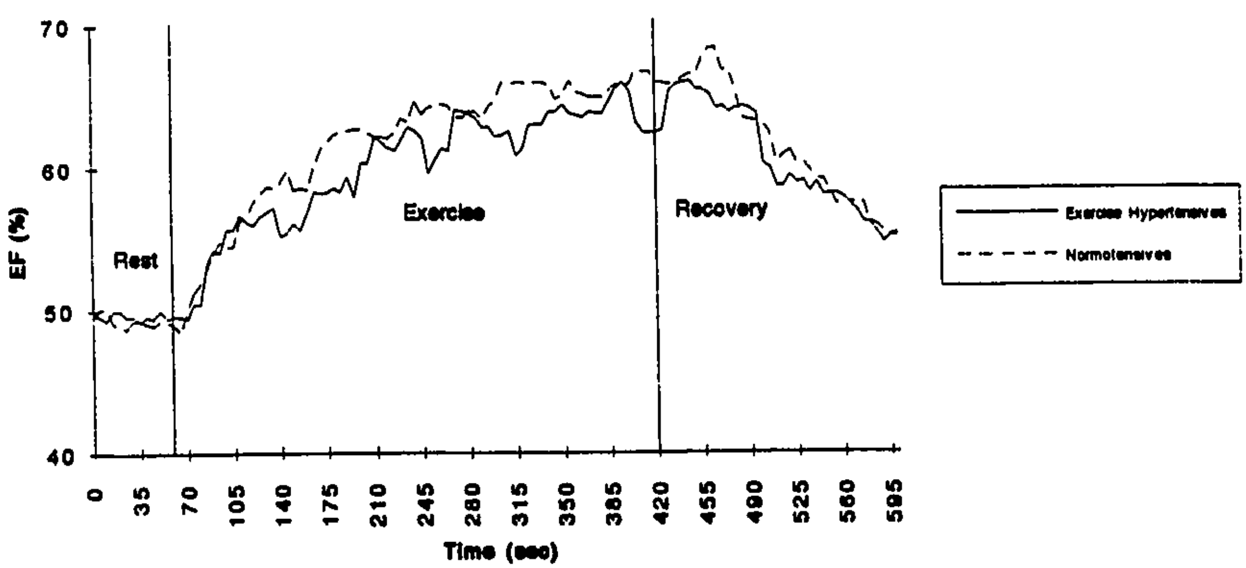
Appendix 1(d). Mean stroke volume in response to the PWC 140



Appendix 1(e). Mean cardiac output pressure response to the PWC 140



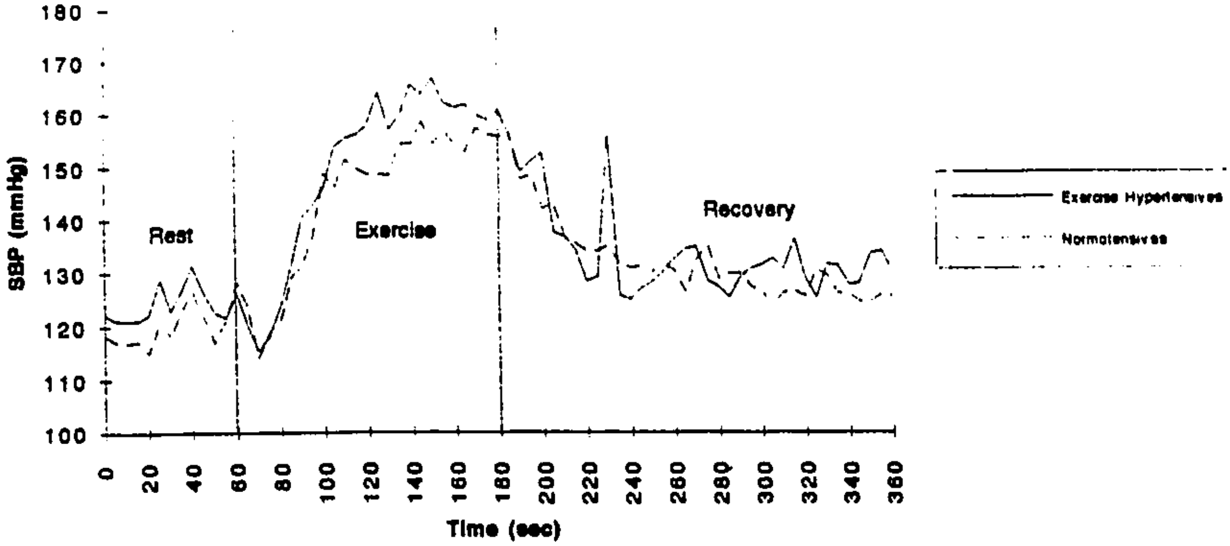
Appendix 1(f). Mean ejection fraction pressure response to the PWC 140



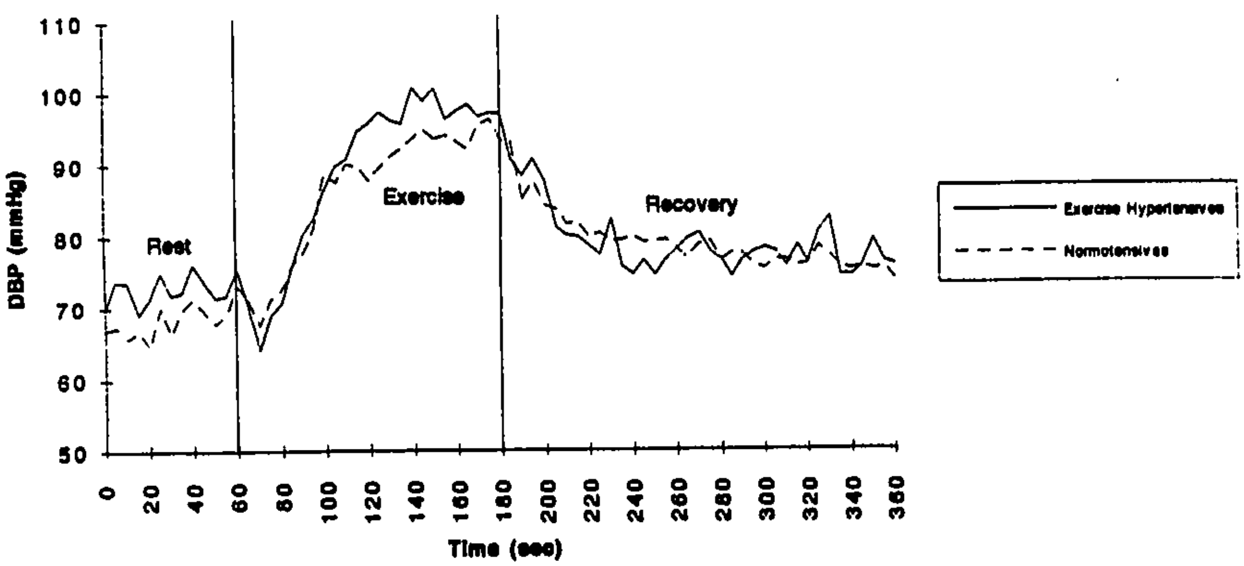
APPENDIX 2

MEAN CARDIOVASCULAR RESPONSE TO THE CPT

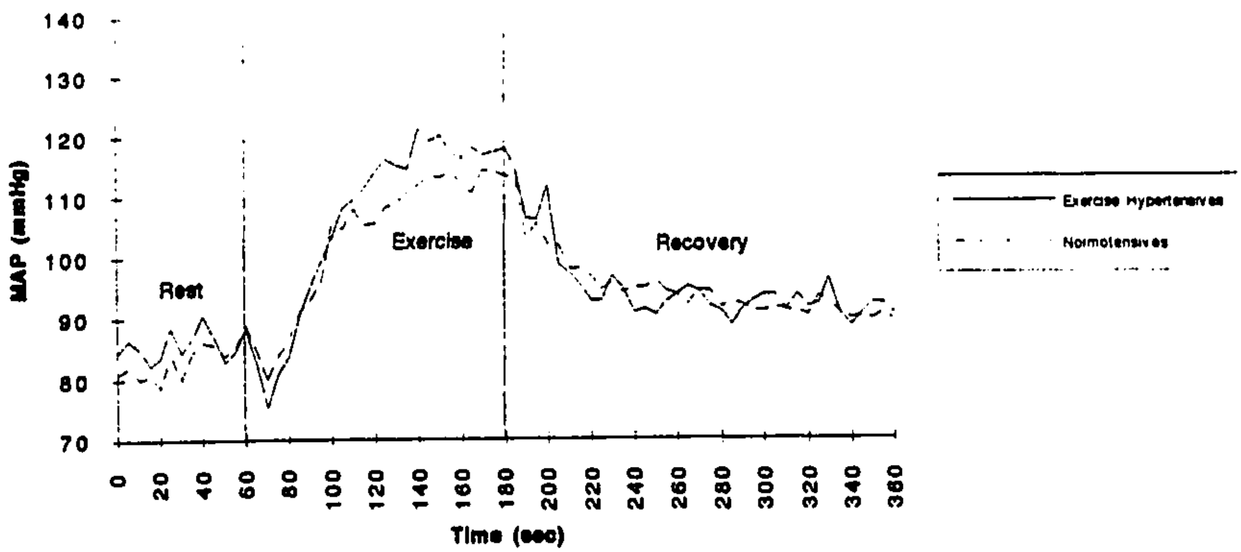
Appendix 2(a). Mean systolic blood pressure response to the CPT



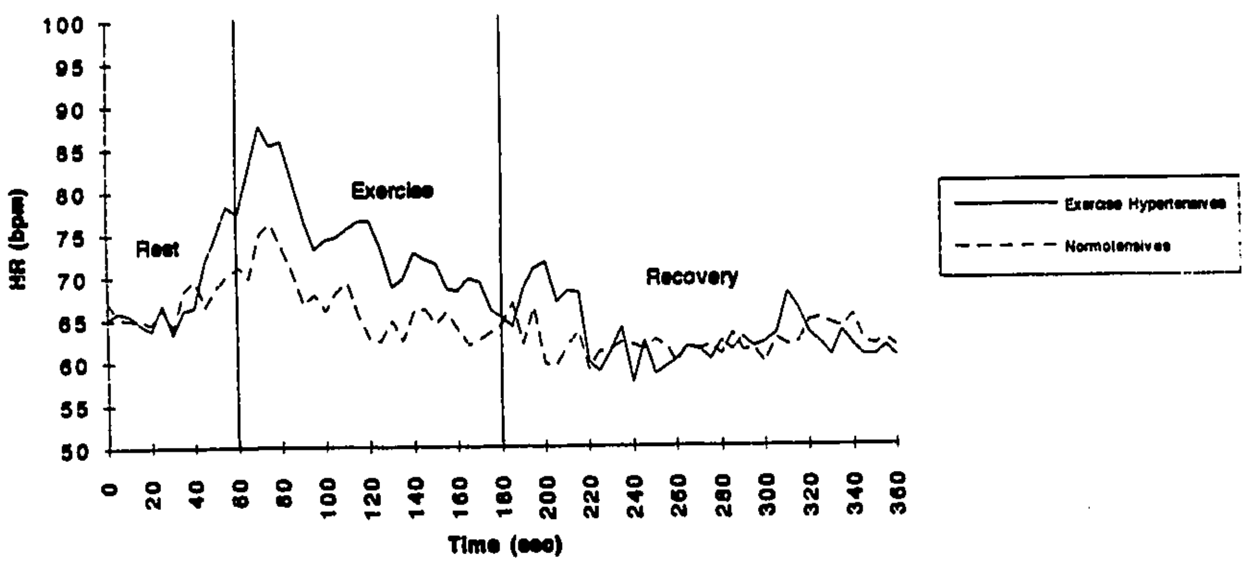
Appendix 2(b). Mean diastolic blood pressure response to the CPT



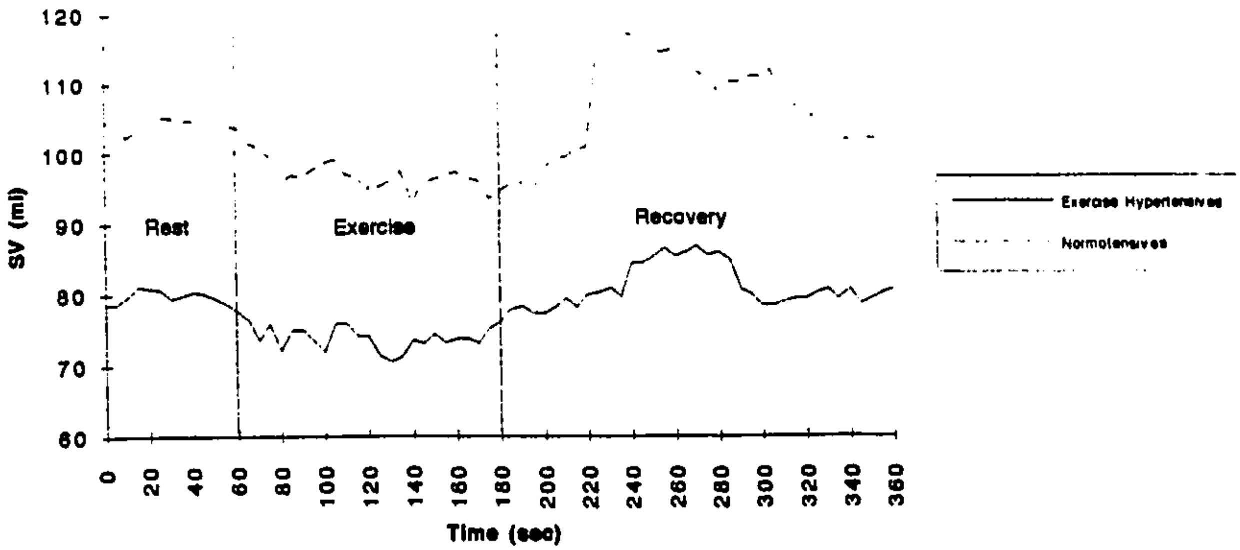
Appendix 2(c). Mean arterial blood pressure response to the CPT



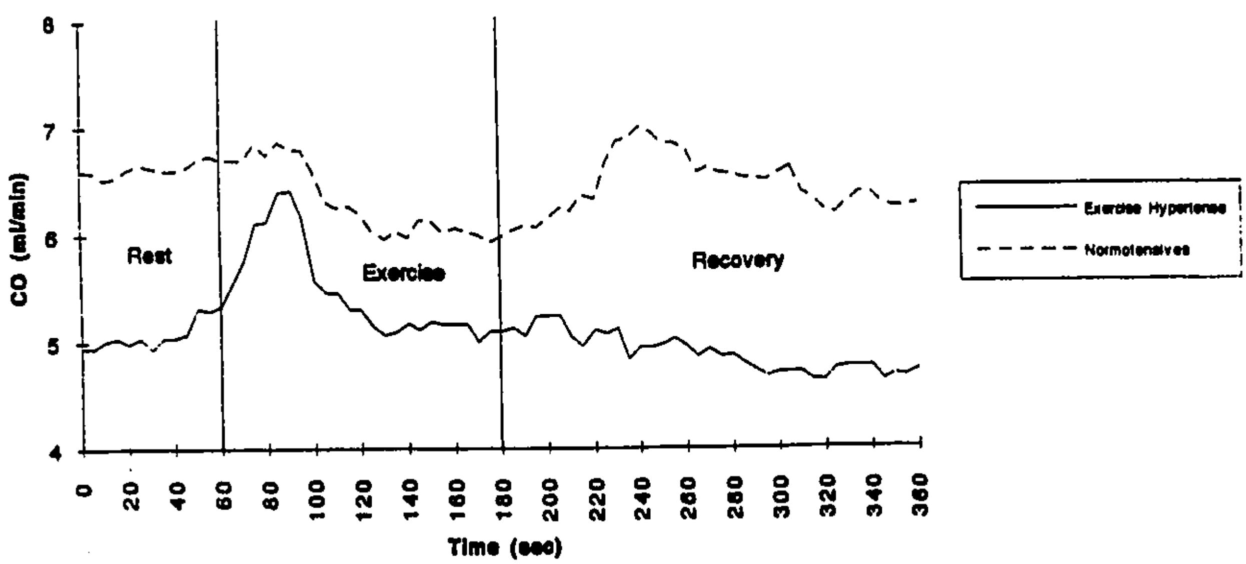
Appendix 2(d). Mean heart rate response to the CPT



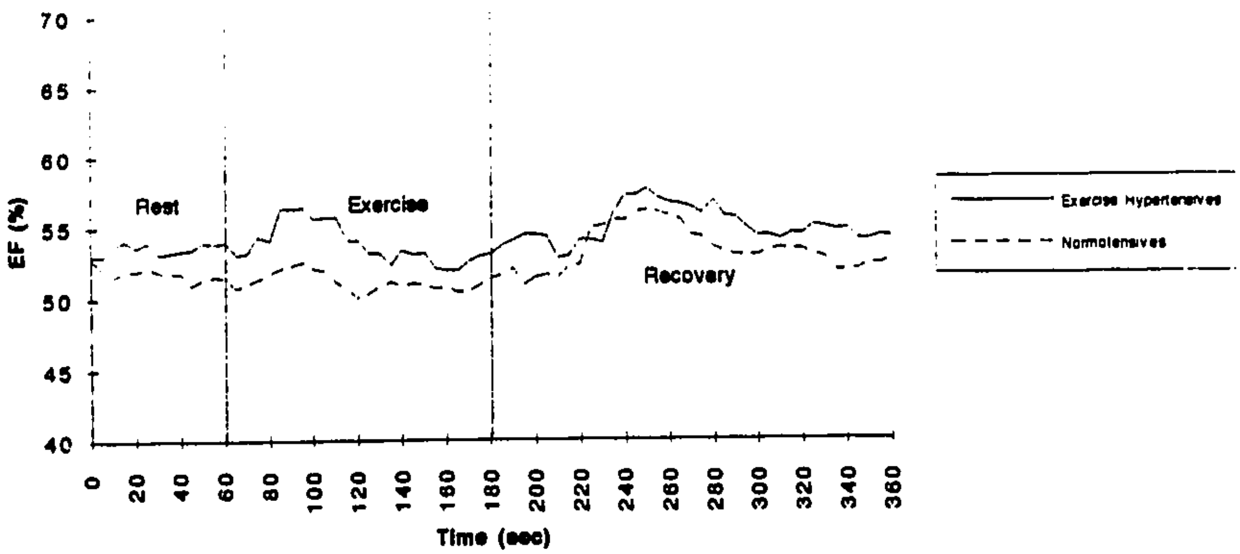
Appendix 2(e). Mean stroke volume response to the CPT



Appendix 2(f). Mean cardiac output response to the CPT



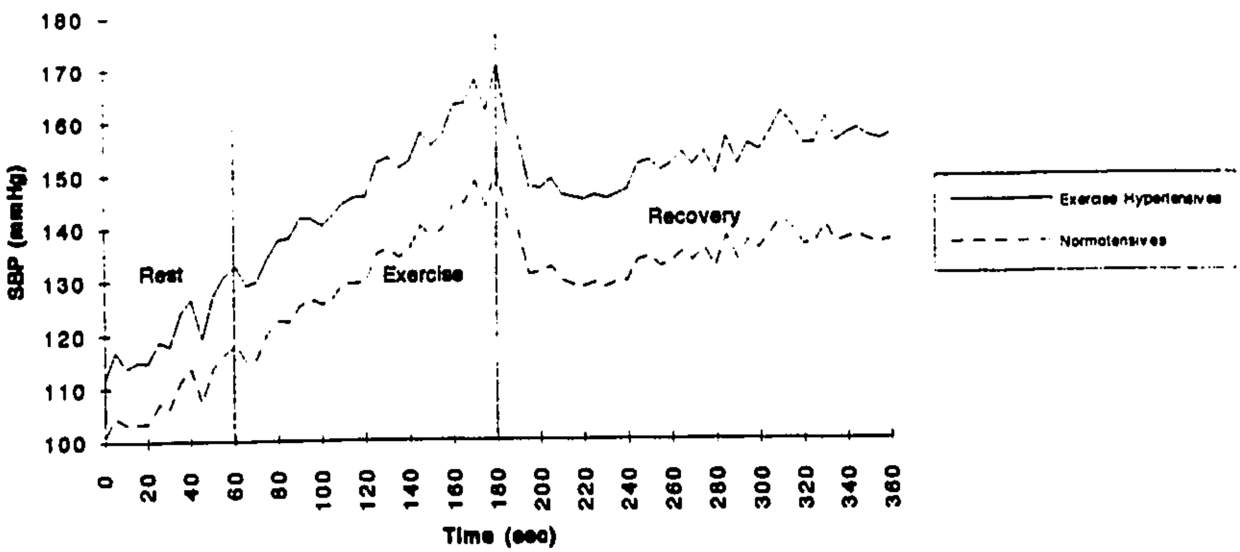
Appendix 2(g). Mean ejection fraction response to the CPT



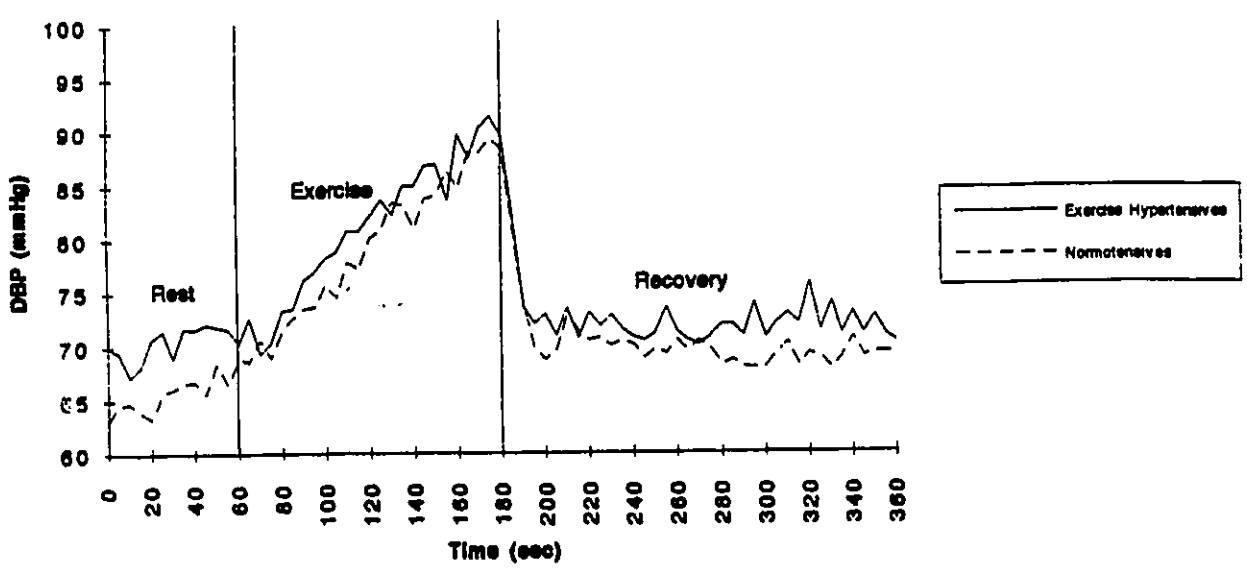
APPENDIX 3

MEAN CARDIOVASCULAR RESPONSE TO THE IHG

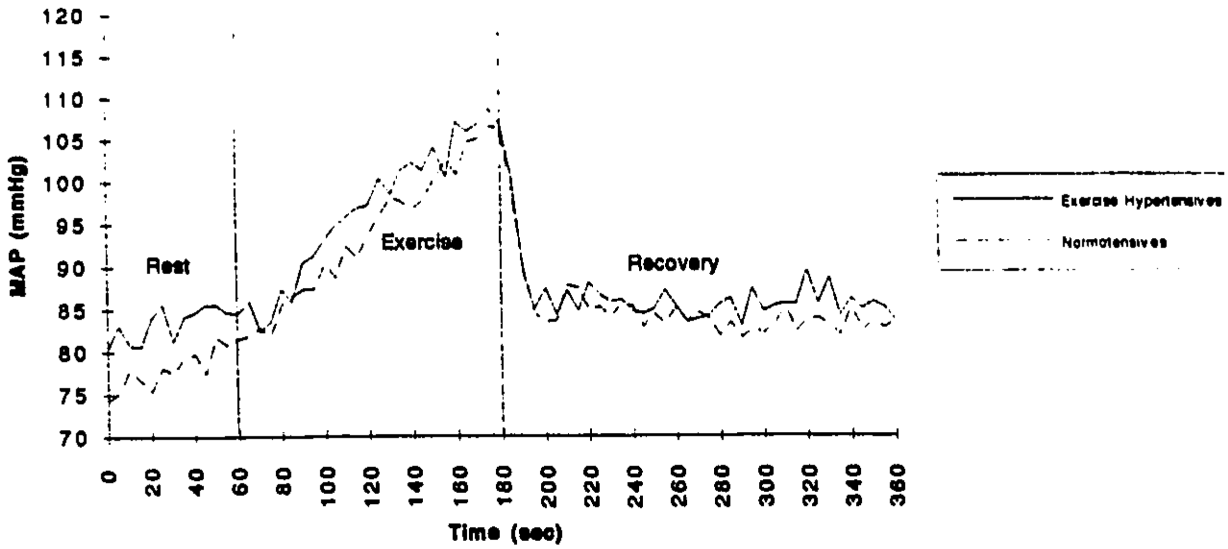
Appendix 3(a). Mean systolic blood pressure response to the IHG



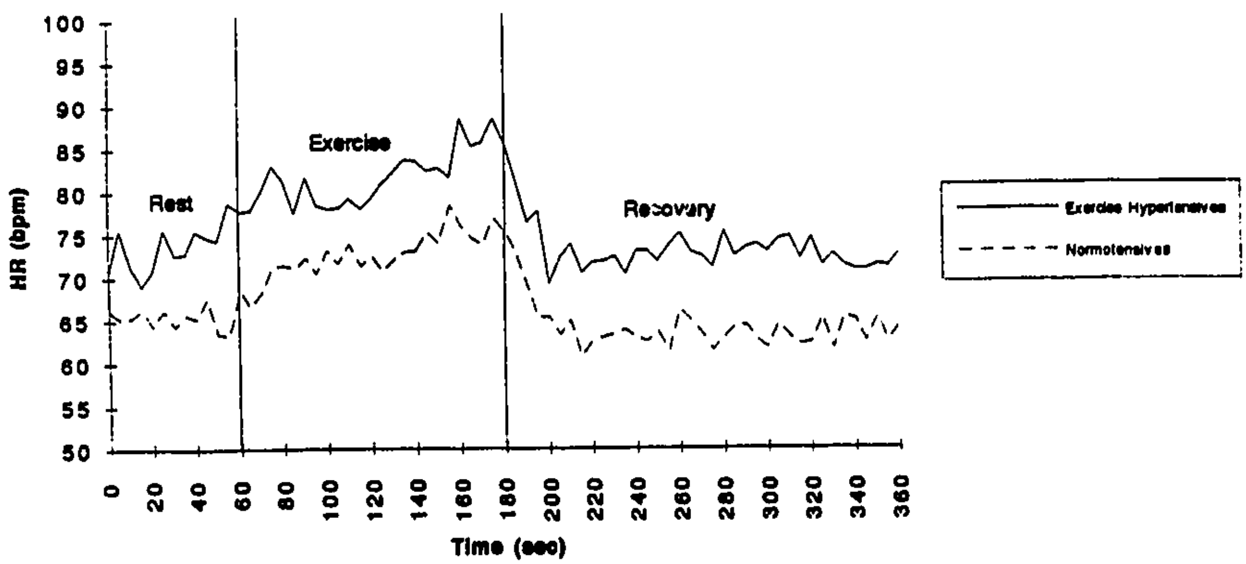
Appendix 3(b). Mean diastolic blood pressure response to the IHG



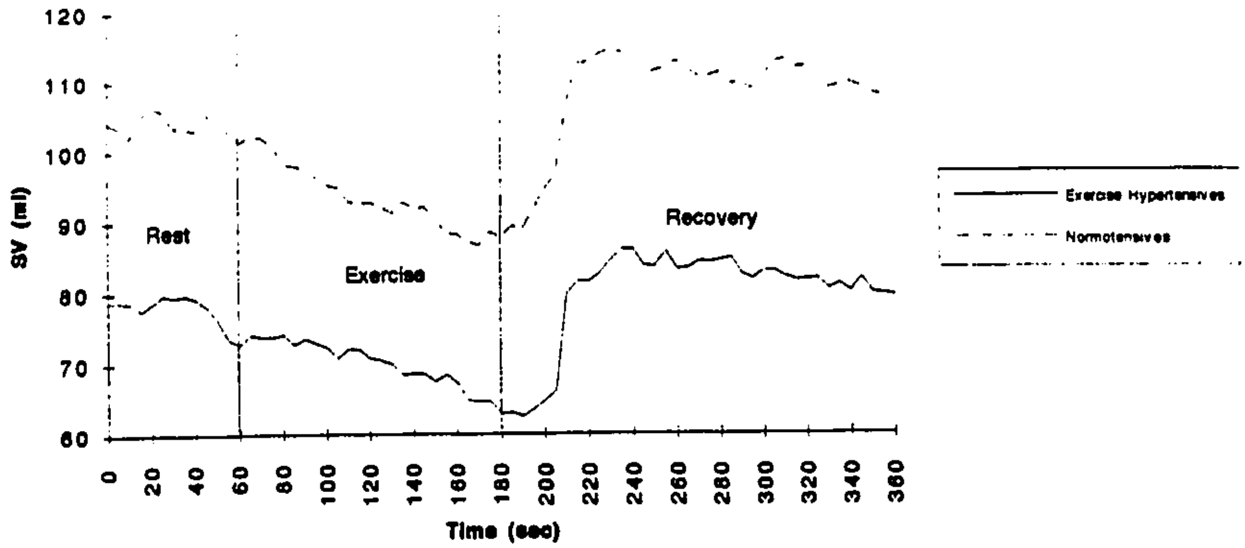
Appendix 3(c). Mean arterial blood pressure response to the IHG



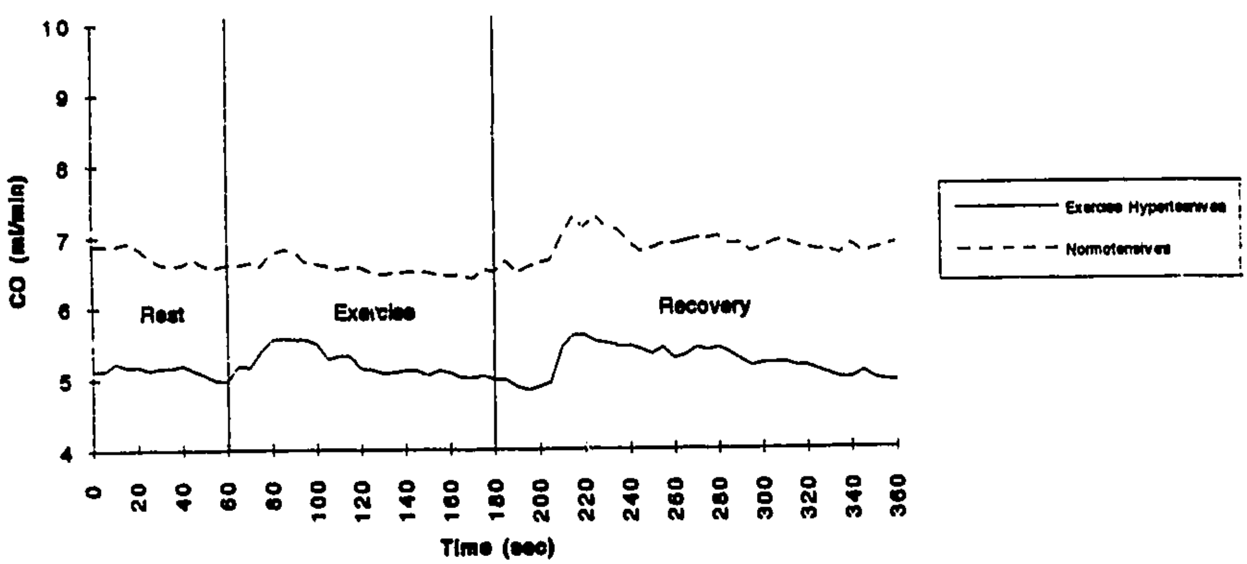
Appendix 3(d). Mean heart rate response to the IHG



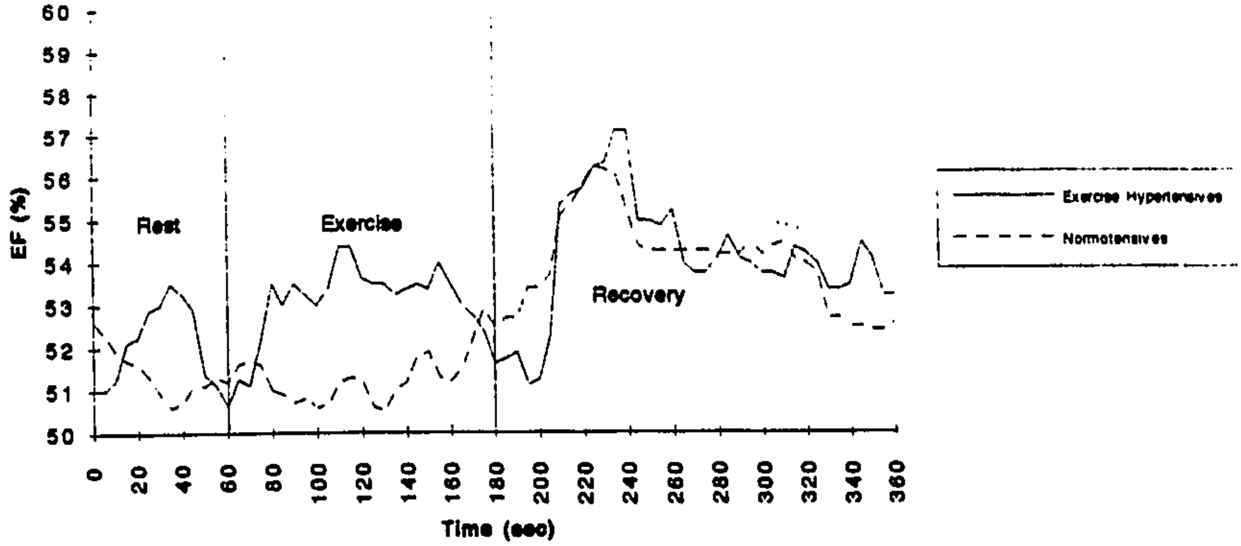
Appendix 3(e). Mean stroke volume response to the IHG



Appendix 3(f). Mean cardiac output response to the IHG



Appendix 3(g). Mean ejection fraction response to the IHG



APPENDIX 4

LETTER OF INFORMED CONSENT

UNIVERSITÉ D'OTTAWA
UNIVERSITY OF OTTAWA

FACULTÉ DES SCIENCES DE LA SANTÉ
FACULTY OF HEALTH SCIENCES

CONSENT FORM FOR CARDIOVASCULAR RESPONSES TO THREE PHYSICAL STRESSORS IN SUBJECTS CLASSIFIED ON THE BASIS OF BLOOD PRESSURE RESPONSE TO THE CAFT

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CHAIR OF FHS-HREC: Dr. F.D. Reardon, Chair
Faculty of Health Sciences
Human Research Ethics Committee
MNT 352, 125 University Pvt.
Ottawa, Ontario K1N 6N5
Tel: 564-9128

Date: ________________ Name of Volunteer:____________________

The title of this research is the "Cardiovascular response to three physical stressors in subjects classified on the basis of blood pressure response to the CAFT". The purpose of this study is to examine the cardiovascular responses to three physical stressors in young male volunteers, in order to improve the early prediction of hypertension.

I understand that I will be asked to participate in a preliminary and two experimental sessions lasting from 60 to 90 minutes. During the course of this study the following tests will be administered:

1) The Canadian Aerobic Fitness Test,
2) Eysenk Personality Questionnaire,
3) The Cold Pressor Test,
4) Isometric Hand Grip Test,
5) Physical Working Capacity at a heart rate of 140,
6) Lifestyle Questionnaire

There are minimal risks of discomfort associated with these tests as utilized in this study. These are: localized discomfort in the forearm such as a prickling or tingling sensation during the two-minute cold pressor test; episodes of transient lightheadedness, fainting, abnormal blood pressure, leg cramps and nausea during the CAFT and PWC 140 tests; and local muscle fatigue during the isometric handgrip test. It is understood that I can withdraw from the experiment at any time I may wish.

The confidentiality of my data will be maintained at all times. Only the principal investigators and the advisor will have access to the data. My name will not appear on any data sheet but will be identified by a coded number.

I MAY WITHDRAW this consent and can discontinue my participation at any time without prejudice.

Signature of volunteer: ________________________Date:_________

Signature of witness: ____

APPENDIX 5

LETTER OF INFORMATION

UNIVERSITÉ D'OTTAWA
UNIVERSITY OF OTTAWA

FACULTÉ DES SCIENCES DE LA SANTÉ
FACULTY OF HEALTH SCIENCES

LETTER OF INFORMATION

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Tel: 564-9128

This project is submitted in partial fulfilment of the degree of master of science in movement studies. The purpose of this study is to examine the cardiovascular responses to three physical stressors in young male volunteers, in order to improve the early prediction of hypertension. This study will help to provide insight into some of the risks associated with hypertension, and some of the relationships that exist between physical stressors and the mediating variable of personality. This study will add to the existing knowledge regarding the early prediction of hypertension.

Participation in this study involves a preliminary session, and two experimental sessions lasting between 60-90 min. each. You will be asked to complete the sessions within a 5 day period with no more than 48 hours seperating the experimental sessions. Total time commitment for this study will be no longer than 4.5 hours.

Preliminary Session

You will be briefed on the purpose of the study, and the protocols and procedures will be explained. You will be asked to sign an informed consent form.

After 10 minutes of rest, your blood pressure will be measured with a blood pressure cuff and verified 5 minutes later.

You will be asked to complete questionnaires regarding demographic data, medical history, family medical history, lifestyle data and a personality questionnaire.

You will complete the Canadian Aerobic Test of Fitness (CAFT). The CAFT is a submaximal test. It involves a maximum of three stages of three minutes each of stepping at a predetermined rate. Blood pressure and heart rate measurements will be taken following each stage of the test.

Experimental Session #1

You will remain seated for ten minutes, after which your blood pressure will be measured using a blood pressure cuff. Then anthropometric measurements (height, weight, skinfolds, and girths) will be taken. Skinfolds will be measured with calipers at four sites: biceps, triceps, suprailiac and subscapular. Girth measurements will be taken at the chest, waist, hips, and thigh.

You will repeat Stage A of the CAFT to verify the results of the preliminary session.

Your maximum handgrip strength will be determined. This will involve three trials of five seconds each of a maximal hand grip.

You will then be initiated to the cold pressor test in which the right forearm is immersed in ice water (2-5° C) for two minutes while cardiovascular responses are monitored continuously with the BoMed and Finapres.

You will then be initiated to the PWC 140 test in which you will pedal on a bicycle ergometer at a rate of 60 rpm for a six minute period, while maintaining a heart rate of 140 bpm. Cardiovascular responses will be monitored during the last minute of exercise and for three minutes following the exercise bout with the BoMed and Finapres.

Experimental Session #2

Your resting blood pressure and heart rate will be measured. You will perform Stage A of the CAFT to verify Session I results.

You will then will perform the handgrip test for two minutes at 33% MVC. Your heart rate and blood pressure will be monitored continuously during this test with the BoMed and Finapres.

You will then rest seated for fifteen minutes. Then you will be given the cold pressor test for two minutes. Heart rate and blood pressure will be monitored with Finapres and BoMed.

You will then perform a test of physical working capacity at a heart rate of 140 beats per minute. The test involves pedalling at a constant rate at a workload that elicits a heart rate of 140 bpm. The Finapres and BoMed will be used to measure heart rate and blood pressure.

There are minimal risks of discomfort associated with these tests as utilized in this study. These are: localized discomfort in the forearm such as a prickling or tingling sensation during the two-minute cold pressor test; episodes of transient lightheadedness, fainting, abnormal blood pressure, leg cramps and nausea during the CAFT and PWC 140 tests; and local muscle fatigue during the isometric handgrip test. It is understood that you may withdraw from the experiment at any time.

The confidentiality of your data will be maintained at all times. Only the principal investigators and the advisor will have access to the data. Your name will not appear on any data sheet but will be identified by a coded number. All data will be stored in a secured room.

Your participation in this study is on the understanding that you may withdraw your consent at any time without prejudice. All information gathered during the investigation will be kept in the strictest confidence.

APPENDIX 6

BLOOD PRESSURE / HEALTH SCREENING QUESTIONNAIRE

School of Human Kinetics
Faculty of Health Sciences
University of Ottawa

HYPER III STUDY

SUBJECT BP/HEALTH SCREENING

NAME: LAB #:

PHONE #: DATE:

ADDRESS:

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

   At present, are you taking medication for blood pressure? YES NO

   If yes, what medication? ______________________

   Dosage: ______________

5. Have either of your parents been told that they have high blood pressure? YES NO UNSURE

   If yes, at what age? ___________

   If yes, did they or are they taking medication for blood pressure? YES NO

   If yes, what medication? ______________________

   Dosage: ______________

   If yes, would it be possible to get in contact with either/both of them? YES NO

6. Do you suffer from any respiratory tract problem such as Chronic Bronchitis, Asthma, or Emphysema? YES NO

7. Have you ever had or are you now suffering from any nervous disorder? YES NO

8. Have you ever suffered from kidney disease? YES NO

9. Do you suffer from a bone or joint problem which either has been or may be irritated by an exercise session? YES NO

10. Do you know of a valid medical reason why you should NOT be involved in either a regular excercise program or an excercise testing session? YES NO

11. At present, are you taking any other type of medication, whether they are perscribed or "over the counter"? YES NO

If yes, for what reason? ______________________

what medication? ______________________

Dosage: ________________

Signature: ____________________

Date: ____________________

August 1993

APPENDIX 7

LIFESTYLE QUESTIONNAIRE

School of Human Kinetics
Faculty of Health Sciences
University of Ottawa

Lifestyle Questionnaire

NAME: LAB #:

AGE: DATE:

1. Do you take part in REGULAR physical activity? YES NO

   If yes, then list Type(s), Amount Weekly, Duration and Intensity

   Duration: refers to minutes per excercise session

   Intensity: light= slight change above normal state
   moderate= perspiration and breathing above normal
   heavy= heavy perspiration and heavy breathing

Activity	Amt. Weekly	Duration	Intensity

2. When did you last receive a medical examination?

   (month/year) ______________________

3. Please circle the MOST appropriate response

   Smoker Ex-smoker Non-Smoker

   If Smoker then Cigarettes Pipe Cigars Other

   Amount smoked per day ______________________

   Enter number of years as: an ex-smoker____ a smoker____

4. Do you drink coffee? YES NO cups per day ________

Do you drink tea? YES NO cups per day ________

5. Alcohol Consumption

a) Presently or Occasionaly drinks   b) Ex-drinker   c) Never drank

If a), then amount consumed in number of bottles/ounces per week:

Beer: _____ bottles Wine: _____ ounces Spirits ____ ounces

August 1993

APPENDIX 8

DATA COLLECTION SHEETS

School of Human Kinetics
Faculty of Health Sciences
University of Ottawa

HYPER III STUDY

EXERCISE

NAME: HEIGHT:

SEX: M __ F __ LAB #: WEIGHT:

PART A

CAFT

DATE:

PRE-TEST

HR _____ bpm

SBP ______mm Hg
DBP __________mm Hg

TESTING

STAGE A ___ HR A ____ b\10s BP A ____________mm Hg
____________mm Hg

STAGE B ___ HR B ____ b\10s BP B ____________mm Hg

STAGE C ___ HR C ____ b\10s BP C ____________mm Hg

VO2 MAX: _______mL\kg\min

PART B

ISOMETRIC HAND GRIP

DATE:

Determination of MVC

Trial 1 ______ Mean of trials: ______

Trial 2 ______

Trial 3 ______ 30% MVC _________

August 1993

School of Human Kinetics
Faculty of Health Sciences
University of Ottawa

HYPER III STUDY

ANTHROPOMETRIC DATA

NAME: LAB #:

DATE: AGE: SEX: M ___ F ___

RESTING BLOOD PRESSURE (mm Hg)

1) SBP __________mm Hg
DBP ________mm Hg

2) SBP __________mm Hg
DBP ________mm Hg

3) SBP __________mm Hg
DBP ________mm Hg

4) SBP __________mm Hg
DBP ________mm Hg

GIRTHS (cm)

WAIST: _____ _____

HIP: _____ _____

CHEST: _____ _____

THIGH: _____ _____

SKINFOLDS (mm)

TRICEPS: _____ _____ _____

SUPRAILIAC: _____ _____ _____

BICEPS: _____ _____ _____

SUBSCAPULAR: _____ _____ _____

Stage A CAFT Verification

SCREENING
SBP _________mmHg
DBP _______mmHg

PRELIMINARY SESSION
SBP __________mmHg
DBP ________mmHg

EXP SESSION I
SBP _________mmHg
DBP ________mmHg

EXP SESSION II
SBP __________mmHg
DBP ________mmHg