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PRESSOR RESPONSE TO ISOMETRIC HANDGRIP COMBINED WITH FOOT IMMERSION IN COLD WATER

by

Yuning Liu

In Partial Fulfilment Of
Master Of Science In Movement Studies

Presented to the School of Graduate Studies

University of Ottawa
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CONTENTS

APPENDICES

LIST OF TABLES AND FIGURES

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ARTICLE

Pressor Response to Isometric Handgrip Combined with Foot Immersion in Cold Water

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ABSTRACT

The purposes of this study were to (1) compare the pressor response between isometric exercise and a cold pressor test (CPT) and (2) examine the pressor response to isometric exercise at 33% of maximal voluntary contraction (MVC) combined with a CPT (cold foot immersion in ice-water) applied either at the onset or during the last minute of a 2-min CPT (2°C to 4°C). Ten normotensive male volunteers (mean age 26.7 ± 3.69 yrs) performed isometric handgrip (HG) at 33% MVC, cold foot immersion (CPT), HG combined with a simultaneous CPT, and HG performed during the last minute of a 2-min CPT in a random order over three days. Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and heart rate (HR) were recorded at rest and continuously throughout the tests using a Finapres 2300 BP monitor. Data were analyzed by paired t-tests and two-way repeated measures ANOVA with trend analysis designs. The results of this study indicate that (1) the pattern of HR response between the 2-min HG and the CPT was different; (2) DBP values during CPT for the initial 30s and the last 15s were significantly lower than DBP corresponding values during HG, while there were no significant differences between the CPT and HG with respect to SBP response; (3) when HG and CPT were performed simultaneously, the effects on SBP and HR were additive, whereas the effects on DBP and MAP were not; (4) CPT performed for 1 minute prior to HG attenuated the SBP and HR responses to HG at 33% MVC, and (5) although both HR and BP increased in response to HG at 33% MVC, only BP increased progressively in a linear fashion when combined with CPT. It is concluded that when HG (33% MVC) is combined with CPT, additive SBP and HR responses are elicited. However, when CPT is initiated 1 minute prior to combined HG and CPT, SBP and HR responses are attenuated.

Key words: Isometric exercise, cold pressor test, isometric stress, local cold stress, sympathetic nervous activity, blood pressure

INTRODUCTION

The cardiovascular response to isometric exercise is generally characterised by increases in systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP), with a relatively small increase in heart rate (HR) and minimal changes in stroke volume and total peripheral vascular resistance. The pressor response to isometric exercise is the result of an increase in cardiac output (Shepherd et al., 1981 and Laird et al., 1979), primarily the result of an increase in heart rate (Keul et al., 1981). The initial increase in HR is due to vagal withdrawal (Freyschuss, 1970), whereas sympathetic nervous activity (SNA) causes a further increase in HR (Martin et al., 1974 and Mark et al., 1985). Furthermore, the stimulation of group III and IV afferents from muscle contraction also causes a reflex cardiovascular response (McCloskey and Mitchell, 1972 and Waldrop et al., 1984). Muscle fibre type corporation has also been shown to influence the pressor response to isometric exercise (Petrofsky and Lind, 1980; Frisk-Holmberg et al., 1983, and White and Carrington, 1993)

The increases in HR and BP during a cold pressor test (CPT), on the other hand, are due to an increase in SNA and high cutaneous vasoconstriction (LeBlanc et al. 1978; LeBlanc et al., 1988; Frey et al., 1980 and Seals, 1989). Unlike the pressor response to isometric exercise, the pressor response to CPT is not related to muscle fibre type composition (Frisk-Holmberg et al., 1983). The cold stimulation of thermal receptors and nociceptors during CPT results in a reflex pressor response (Lloyd, 1986, p81 and Lovallo, 1975).

Various investigations have shown that during a 2-min CPT, a typical pressor response is characterised by an initial marked increase in BP; the response then levels off and drops slightly during the last part of CPT (LeBlanc, 1975). Research has also shown that during isometric exercise BP response, especially DBP response, increases markedly and progressively. Frisk-Holmberg and co-workers (1983) suggested that the magnitude of the arterial blood pressure response to CPT for 2 minutes was similar to isometric handgrip at 33% MVC for 2 minutes; however, the mechanisms eliciting the pressor response were different. Peikert and Smolander (1991) suggested that the blood pressure and heart rate responses to isometric exercise when combined with CPT was higher in comparison to isometric exercise performed without immersion.

However, no previous study has addressed the question as to whether HG, applied either at the onset or following 60 sec of CPT, influences the pressor response. In addition, limited information is available regarding the immediate and continuous measurement of the pressor response during CPT because most studies measured BP by auscultation, such that the immediate and continuous measurement of BP were not possible. Therefore, this study were designed to (1) compare the pressor response between CPT and isometric exercise (33% MVC); and (2) examine the pressor response to isometric exercise at 33% MVC applied either at the onset or during the last minute of a 2-min CPT.

METHODOLOGY

Subjects

Ten apparently healthy male volunteers between the ages of 22 to 31 years (mean age 26.7 ± 3.69 yrs) participated in the study. The subjects were normotensive and not taking medication. The study was approved by the Human Research Ethics Committee of the University of Ottawa. A written consent was obtained after the subjects had been informed of the procedures used in the experiments.

Materials and Procedures

Handgrip contraction (HG) was performed using a rubber bulb connected to an air pressure gauge (H. O. Trerice Co., Detroit, Michigan) by plastic tubing. The force was displayed to the subjects on a dial. The maximal voluntary contraction was taken as the average of three measured maximal dominant handgrip contractions with a 5-min rest between each contraction.

CPT was administered using a plastic container filled with ice-water (2 to 4° C). During the CPT, the right foot was immersed up to the ankle in the ice-water.

A Finapres Blood Pressure Monitor (Ohmeda Louisville, Co.) connected to an IBM computer was used to record HR, SBP, DBP and MAP responses during all of the experimental tests.

At a preliminary testing session, after a 5-min sitting rest, resting HR and BP values were measured using a standard sphygmomanometer and stethoscope to ensure that the subjects were normotensive. Height and weight were then measured.

The three experimental sessions were administered in a randomized fashion over three days. All tests were conducted in the afternoon during the second week of January to the third week of February. Subjects were requested not to participate in any form of physical activity or to drink caffeine-containing beverages for at least 3 hours prior to testing.

The experiments were performed in a sitting position. The subjects sat comfortably in the laboratory for 20 minutes prior to the tests. The Finapres cuff was then applied on the middle finger of the non-dominant hand. The subjects were reminded to relax, to breathe normally and to avoid a Valsalva manoeuvre during the immersion and the handgrip contraction. They were instructed to maintain the 33% MVC by observing the air pressure gauge. Resting HR and BP were recorded for a period of one minute at 5-second intervals prior to each test. HR, SBP, DBP, and MAP were recorded continuously at 5-second intervals during all testing, as well as for a 4-min period following the test.

Experimental session one: The subject performed the HG at 33% MVC for 2 minutes (condition 1). After a 15-min rest, the subject performed the CPT (condition 2).

Experimental session two: The subjects performed the HG at 33% MVC simultaneously with the CPT for 2 minutes (condition 3).

Experimental session three: The subjects performed the CPT for 2 minutes during which the HG at 33% MVC was administered during the last minute of the CPT (condition 4).

Statistical analyses

Statistical analyses were performed on the University mainframe computer using SPSSx.

Descriptive analysis of the subjects included: age, height, weight, resting HR, resting BP, and MVC.

The dependent variables of SBP, DBP, MAP and HR, were represented as the difference between the resting value and the response value (ie, as ΔSBP, ΔDBP, ΔMAP, and ΔHR). Pre-exercise resting values were the average of 15s measurements prior to the experimental tests. For statistical purpose, the average of three 5s values were utilized.

A student paired t-test was used to determine whether significant difference existed between CPT and HG (condition 1 vs. condition 2). A 2x8 repeated measures ANOVA with trend analysis design (Stevens, 1990) was used to determine if overall significant differences existed between condition 1 and condition 2 over the 2-min test. A 2x7 repeated measures ANOVA with trend analysis design was used to determine if there were significant differences between condition 1 and condition 2 during the 4-min recovery period.

A 3x4 repeated measures ANOVA with trend analysis design was used to determine if significant differences existed among the responses to conditions 1, 3, and 4. If an overall significant difference between-subjects factor was found, Tukey's technique was administered for post hoc analysis in order to localize the significant mean difference.

P values smaller than 0.05 were considered to be statistically significant.

RESULTS

The mean (±SD) age and physical characteristics of the subjects are shown in Table 1. Their mean height was 177.3 cm; mean weight, 74.0 Kg; mean resting HR, 70.8 beat/min; mean resting SBP, 113.7 mmHg; mean resting DBP, 63.0 mmHg and mean maximum dominant handgrip, 86.2 KiloPascals.

Comparison of the pressor response between HG and CPT

The mean ΔSBP, ΔDBP, ΔMAP and ΔHR responses to the 2-min HG at 33% MVC (condition 1) and the 2-min CPT (condition 2) are shown in Table 2.

There were significant increases in mean ΔSBP from rest for both CPT and HG (Fig 1).

However, the differences in mean ΔSBP value between HG and CPT during the 2-min tests were not significant (F=.00, P<.992).

The mean ΔDBP to HG showed a significant increase throughout the period of the 2-min test (Fig 2). However, mean ΔDBP to CPT showed an initial reduction during the first 15s, followed by a progressive increase and then a slight decrease during the last 15s. The results from the t-test (Table 2) show that the mean ΔDBP increases to HG were significantly higher than the response to CPT in the first 30s (P<.001 and P<.019) and during the last 15s of the test (P<.005).

Figure 3 shows that the mean ΔMAP values to HG increased progressively throughout the 2-min test, while the mean ΔMAP values to CPT decreased in the first 15s, then progressively increased to a plateau over the next 60 sec. The mean ΔMAP values for HG were significantly higher than values for CPT at the first and the last 30s of the tests (P<.002, P<.0048, P<.0038 and P<.007).

During CPT, mean ΔHR increased by 9 beat/min during the first 15s of the test, and then decreased in the following 30s, remaining 1 to 3 beat/min above resting for the duration of the immersion (Fig 4). On the other hand, during HG, mean ΔHR increased 3 to 8 beat/min. T-tests showed that the increase in mean ΔHR for HG at the first 15s was significantly lower than the increase for CPT (P<.006). Thereafter, however, the mean ΔHR for HG was generally higher than for CPT (Table 2).

Comparison of the pressor response between post-HG and post-CPT

The mean ΔSBP, ΔDBP, ΔMAP, and ΔHR values measured during the 4-min recovery period following CPT and HG are shown in Table 3. There were no significant difference in mean delta score between tests for HR and BP variables. Following the first 15s of post-CPT, mean ΔHR dropped slightly below resting levels until the second minute of the recovery and then returned to resting level (Fig 5). Both mean ΔSBP and mean ΔDBP decreased rapidly over the first 15 to 30s of recovery; thereafter, values averaged 1 to 4 mmHg above resting for DBP and 3 to 6 mmHg for SBP (Fig 6).

Pressor response to HG combined with CPT

The mean ΔSBP, ΔDBP, ΔMAP and ΔHR responses to the first minute of HG (condition 1), the first minute of HG combined with simultaneous CPT (condition 3) and HG performed following one minute of CPT (condition 4) are shown in Table 4.

Mean ΔSBP was significantly higher when HG was performed simultaneously with CPT (condition 3) compared to HG performed alone (condition 1) or after 1 minute of CPT (condition 4) (Fig 7). Mean ΔSBP values for HG without CPT (condition 1) were also significantly higher than values for HG proceeded by 1 minute of CPT. The smallest increase in mean ΔSBP was observed during HG initialed 1 minute after CPT (condition 4). There were no significant differences for mean ΔDBP and mean ΔMAP among the three conditions (Figs 8 and 9).

The increase in mean ΔHR when HG was performed simultaneously with CPT was significantly higher than when HG was performed without CPT and following 1 minute of CPT (Fig 10). Moreover, the mean increase in ΔHR to HG per se was significantly higher than to HG performed after 1 minute of CPT.

During conditions 1, 3 and 4, all mean Δblood pressure parameters showed a linear rise, but this linear trend was not significantly different among conditions. Changes in mean ΔHR over the 1-min HG contraction period were not statistically linear for any of the three test conditions (Fig 10).

DISCUSSION

The purposes of this study were to compare the pressor response between CPT and isometric exercise at 33% MVC and examine the pressor response to isometric exercise (33% MVC) combined with CPT (immersion of the right foot in cold water) applied either at the onset or during the last minute of a 2-min CPT in normotensive males. One distinctive feature of this study was the ability to make immediate and continuous measurement of the pressor response using the Finapres BP monitor as opposed to previous studies which measured blood pressure by auscultation. The results of this study indicate that (1) the pattern of HR response between the 2-min HG and the CPT was different; (2) DBP values during the initial 30s and the last 15s of CPT were significantly lower than corresponding values during HG, while there was no significant difference between the CPT and HG with respect to SBP responses; (3) when HG and CPT were performed simultaneously, the effects on SBP and HR were additive, whereas the effects on DBP and MAP were not; (4) CPT performed for 1 minute prior to HG attenuated the SBP and HR responses to HG at 33% MVC; and (5) although both HR and BP increased in response to HG at 33% MVC, only BP increased progressively in a linear fashion when combined with CPT.

Comparison of the pressor response between HG and CPT

The results of this study indicated that the pattern of mean ΔHR response to CPT and HG was different (Fig 4). During the CPT, mean ΔHR showed an initial increase and then a prompt decrease. This is in agreement with the observations of Seals (1990) and Frey and co-workers (1980). However, mean ΔHR response to HG demonstrated averaged 3 to 8 beat/min above resting. The mean ΔHR response to the CPT during the first 15s was significantly higher than for the HG, while during the last 45s, it was significantly lower (Fig 4).

The initial increase in HR during isometric exercise is primarily the result of parasympathetic withdrawal. It is mediated by baroreflexes and is accompanied by bursts of vasoconstrictor impulses (Freyschuss, 1970; Martin et al., 1974 and Mitchell et al., 1989). Sympathetic nervous activity (SNA) then causes a further increase in HR (Martin et al., 1974 and Mark et al., 1985). During isometric exercise, HR is reported to increase promptly but MSA does not increase until the second minute of contraction (Mark et al., 1985). On the other hand, MSA has been shown to increase immediately and then decrease during a 1-min CPT and is responsible for the increase in cardiovascular response (Fagius et al., 1989). This initial increase in MSA during a CPT may in part be activated by the pain associated with the cold stimulation (LeBlanc et al., 1979 and Seals, 1990). Thus, the pattern of sympathetic nervous activity and hence the cardiovascular response differs between CPT and HG. Since HR is primarily controlled by the balance between parasympathetic and sympathetic nervous activities acting on the intrinsic pacemaker discharge frequency of the sinoatrial node, the different pattern of sympathetic nervous activity during CPT and isometric exercise could explain, at least in part, the different mean ΔHR observed in this study.

Frisk-Holmberg and co-workers (1983) reported that the MAP response to a 2-min handgrip at 33% MVC and to a 2-min cold hand immersion is similar. They did not report values for SBP and DBP. The results of the present study showed that while changes in mean ΔSBP were similar for HG and CPT (Fig 1), changes in mean ΔDBP and ΔMAP at the onset and final 30s were not (Figs 2 and 3).

The marked progressive increase in DBP during isometric exercise in this study (Fig 2) is in agreement with previous experiments (McCloskey and Mitchell, 1972; Rowell et al., 1981 and Mitchell et al., 1983). However, the reduction in mean ΔDBP at the onset of CPT differs from previous reports (LeBlanc, 1975). The observed difference in pressor response could be due to the inability of the auscultation method to detect rapid/transient pressure changes - all earlier studies (Cummings et al., 1983; LeBlanc, 1975; LeBlanc et al., 1979; Robertson et al., 1979; Seals, 1990 and Stratton et al., 1983) measured BP changes using auscultation. In the present study, BP was recorded by a Finapres monitor at 5-second intervals.

The reason for the decline in mean ΔDBP and ΔMAP at the onset of CPT despite an increase in mean ΔHR and mean ΔSBP is not clear. Petrofsky and Lind (1980), Frisk-Holmberg and co-workers (1983) and White and Carrington (1993) suggested that the magnitude of the pressor response to isometric exercise is affected by the degree of fast twitch fibre (FT) involvement. However, the percentage of FT fibre is not related to pressor response of CPT (Frisk-Holmberg et al., 1983). Thus, it might be expected that the pressor difference in response to CPT and HG could be different. In the present study, there was a decrease in mean ΔDBP at the onset of the CPT, while mean ΔDBP to the initial HG showed an increase. In marked contrast to the DBP response, changes in mean ΔSBP with HG and CPT were similar. Since the mean ΔDBP for HG was significantly higher than for CPT during the initial 30s and the last 15s (Fig 2), this could suggest a greater contribution from peripheral signals during isometric contraction. However, compared to DBP, SBP has a stronger relationship with MSA during CPT (Fagius et al., 1989). Therefore, it is possible that an increase in SBP in response to CPT is due in part to vasoconstriction resulting from the cold stimulation. However, the implication of the initial increase in mean ΔSBP and ΔHR along with the simultaneous decrease in mean ΔDBP at the onset of the CPT remains to be elucidated.

Comparison of the pressor response between post-HG and post-CPT

There were no significant differences in cardiovascular responses between CPT and HG during recovery (Fig 5). Previous studies also indicate that BP promptly decreases within 1 minute during post-HG and post-CPT. The observation that mean HR returned to resting level within 15s of cessation of HG is in agreement with Rusch and co-workers (1981). O'Leary (1993) suggested that the fall in post-exercise HR might be the result of an increase in parasympathetic nervous activity. LeBlanc (1979) reported that following immersion of the hand in cold water for 2 minute, HR was lower than initial values in female subjects, but not in male subjects. His findings on males with hand immersion differ from the present findings on males with foot immersion. The reason for the lower mean HR levels during the 30s to 60s of recovery in this study is not clear. Frey and co-workers (1980) reported that after an initial increase in HR during a 1-min CPT, the decrease in HR response during foot immersion was greater than for hand immersion due to greater sympathetic activation during the hand immersion. It may be that the effects of a cold pressor stress applied at different body sites might provoke a different HR response during post-CPT. However, further investigations are needed to compare gender differences in HR response during post-CPT produced by stimulation of various body areas.

Pressor response to HG combined with CPT

In the present study, mean ΔHR to HG combined with simultaneous CPT (condition 3) was significantly higher than that to HG performed without CPT (condition 1). This finding is in agreement with that of Peikert and Smolander (1991) who showed that HR response to a 3-min isometric exercise combined with a CPT performed during the last minute of the contraction was significantly higher than the isometric exercise without CPT. A number of studies have shown that isometric exercise initially produces a vagal dependent tachycardia (Freyschuss, 1970, Martin et al., 1974 and Mitchell et al., 1989). Other studies have also shown that an increase in HR response at the onset of CPT is caused by an increase in SNA (Fagius et al., 1989). Thus, it is possible that both the increase in SNA and the decrease in parasympathetic nervous activity contributed to the increase in HR when HG was combined with CPT. On the other hand, Peikert and Smolander (1991) reported that SBP and DBP responses to isometric exercise when combined with CPT were higher than without CPT, but only the DBP response was significantly different between the two tests. By contrast, in the present study, only mean ΔSBP to HG combined with CPT was significantly higher than to HG without CPT. The reason for these contradictory findings may be due in part to the different research designs. In the Peikert and Smolander's experiment, the BP response to combined isometric exercise and CPT was determined by adding cold hand immersion during the last minute of a 3-min leg extension contraction at 15% MVC. In the present study, isometric exercise was produced by forearm contraction at 33% MVC and cold foot immersion was superimposed at the beginning of the HG. The BP response to combined isometric exercise and CPT may be affected by whether the cold pressor stress is applied at the onset of isometric exercise or following it. Furthermore, White and Carrington (1993) suggested that the peripheral reflex input to the pressor response in arm and leg muscle may be influenced by relative fast twitch fibre area. The absence of significant increase in SBP response to combined isometric exercise and CPT in Peikert and Smolander's study could also be due to a lower exercise intensity (15% MVC). The results of the present study indicated that there were additive effects on SBP and HR response to the moderate HG combined with the simultaneous CPT. However, the response was not the algebraic sum of the separate responses to CPT and HG alone. This additive effect of isometric stress and cold stress may be due to vasoconstriction effects in the contralateral unstressed limb (Peikert and Smolander, 1991).

The present results showed that after a prompt increase at the onset of HG combined with CPT, mean ΔHR levelled off (Fig 10) while mean ΔSBP (Fig 7) and mean ΔDBP (Fig 8) continued to increase. This difference in mean ΔHR and ΔBP responses to HG combined with CPT suggests that different control mechanisms are operating. Seals (1990) reported that SBP increased progressively while HR increased slightly and remained at this level until the end of a 1.5-min CPT. LeBlanc and co-workers (1979) suggested that the increase in BP is due primarily to the peripheral vasoconstriction caused by norepinepherine during CPT. Fagius and co-workers (1989) indicated that BP elevation correlated linearly with an increase in total outflow of sympathetic activity during CPT. Therefore, during the combined isometric and cold stress, a progressive increase in BP could in part be due to peripheral vascular constriction activated by cold stimulation. On the other hand, Mark and co-workers (1985) suggested that a reduction in central command, as the duration of the isometric contraction (20 to 30% MVC) progressed for 2 minutes, is responsible for the decline in HR from its peak exercise level, although the pressor response increased due to the effects of groups III and IV afferent fibres. It may be that a dissociation between BP and HR responses in this study was due to a reduction of central command, activation of groups III and IV fibres, and high cutaneous vasoconstriction. It is reasonable to suggest that there were integrated effects of HR and BP response to HG combined with CPT. It is shown clearly in Figure 4 that mean ΔHR increased quickly at the onset and then decreased during CPT, while during HG, mean ΔHR increased gradually. By contrast, when HG and CPT were administered simultaneously, mean ΔHR rapidly increased before stability for the duration of the test (Fig 10).

No previous study has addressed the problem of whether a local cold stress prior to the administration of HG could affect the cardiovascular response to an isometric contraction. In the present study, HG performed following 1 minute of CPT produced a smaller increase in mean

ΔSBP and mean ΔHR in comparison to HG performed alone or when combined with CPT. It has been shown that vagal withdrawal occurs rapidly at the onset of isometric exercise whereas sympathetic activation occurs after 30s of effort (Maciel et al., 1987). Therefore, the initial increase in HR during isometric exercise is due to vagal withdrawal (Martin et al., 1974 and Mitchell et al., 1989). When HG is performed after 1 minute of CPT, it may be that the vagal withdrawal is masked by sympathetic nervous activity due to cold stimulation. This may be the reason why mean ΔHR response did not further increase at the onset of the HG performed following CPT in this study. These results suggest that HR response at the onset of HG performed following 1 minute of CPT is not vagal dependent. Furthermore, ΔSBP values to HG performed following 1 minute of CPT in this study were significantly lower than values obtained when HG was performed alone, or combined with CPT. As shown in Table 4, the SBP value prior to the HG when HG was performed following CPT was higher than the prior handgrip values when HG was performed without CPT or combined with CPT. This would indicate that a 1-min local cold stress has already produced an increase in SBP prior to the HG when HG was performed following CPT. In this case, an added isometric stress at 33% MVC could not cause a further increase SBP.

The results of this study show that HG at 33% MVC combined with cold foot immersion causes additive SBP and HR responses, and that cold foot immersion prior to HG attenuates SBP and HR responses. The results also indicate that the HR response to 2-min HG at 33% MVC differs from that observed with a 2-min cold foot immersion, possibly a result of a difference in SNA response to isometric and cold pressor stressors. However, the initial and transient decreases in DBP and MAP at the onset of the CPT require further study.

TABLE 1:

Mean (±SD) age and physical characteristics of the subjects

N 10	Age (yrs)	Height (cm)	Weight (Kg)	HR (beat/min)	SBP (mmHg)	DBP (mmHg)	MVC (kPa)
$\bar{x}$	26.7	177.30	74.0	70.8	113.7	63.0	86.2
±SD	3.69	7.24	6.65	9.31	9.31	8.96	11.59

Note: HR=Resting heart rate

SBP=Resting systolic blood pressure

DBP=Resting diastolic blood pressure

MVC=Maximal voluntary contraction

SD=Standard deviation

TABLE 2:

Cardiovascular responses during HG and CPT

		R	15s T1(Δ)	30s T2(Δ)	45s T3(Δ)	60s T4(Δ)	75s T5(Δ)	90s T6(Δ)	105s T7(Δ)	120s T8(Δ)
SBP (mmHg)										
HG	x̄	108.7	10.0	14.0	14.8	16.3	17.0	21.5	27.6	30.5
	±SD	11.4	7.5	10.1	9.0	7.2	7.3	8.5	9.4	14.6
CPT	x̄	113.5	7.8	15.1	20.5	22.7	22.6	23.0	24.8	21.1
	±SD	13.5	9.0	9.5	9.5	8.3	8.2	7.7	7.4	6.3
DBP (mmHg)										
HG	x̄	54.1	5.1*	8.7*	10.1	12.1	12.4	15.4	19.4	22.3*
	±SD	7.0	2.9	3.7	3.8	4.3	5.3	5.5	7.0	8.9
CPT	x̄	57.4	-3.6*	0.6*	5.3	8.1	9.9	10.0	12.3	9.5*
	±SD	9.4	5.2	8.4	8.5	7.6	6.7	7.3	7.8	7.1
MAP (mmHg)										
HG	x̄	68.7	5.9*	9.4*	10.8	12.8	13.7	17.2	21.4*	24.6*
	±SD	6.1	3.6	5.0	4.8	4.7	5.3	5.6	8.1	9.9
CPT	x̄	72.6	-2.6*	1.9*	6.6	9.6	11.2	11.5	13.6*	11.4*
	±SD	8.7	5.4	9.0	9.5	9.2	7.0	8.1	6.7	7.3
HR (beat/minute)										
HG	x̄	69.0	2.7*	3.3	3.8*	4.5	3.6	4.2*	6.7*	8.3*
	±SD	10.9	3.6	6.6	3.8	4.1	4.7	5.1	6.7	8.3
CPT	x̄	67.5	9.0*	1.9	0.0*	2.4	3.0	0.7*	1.1*	0.8*
	±SD	9.5	5.4	5.0	3.4	4.1	3.9	3.1	2.9	3.3

Notes: *: Significant differences between CPT and HG at $P<.05$

Δ = Values are mean ±SD expressed as difference scores related to resting values.

CPT = Cold pressor test HG = 33% MVC handgrip

R: Average value for 15 seconds period prior to CPT/HG

T: Value averages during each 15s period.

TABLE 3:

Cardiovascular responses following CPT and HG

		R	15s T1(Δ)	30s T2(Δ)	45s T3(Δ)	60s T4(Δ)	120s T5(Δ)	180s T6(Δ)	240s T7(Δ)
SBP (mmHg)									
HG	$\bar{x}$	108.7	14.1	5.3	4.4	2.9	5.1	6.6	6.1
	±SD	11.4	9.5	7.8	11.0	7.5	7.1	7.6	7.3
CPT	$\bar{x}$	113.5	14.7	13.0	5.4	6.2	6.2	4.7	5.0
	±SD	13.5	16.2	11.9	7.8	12.8	9.8	10.2	8.2
DBP (mmHg)									
HG	$\bar{x}$	54.1	5.0	0.7	1.5	1.7	2.0	4.1	4.1
	±SD	7.0	4.7	3.7	5.7	4.8	5.3	5.2	5.5
CPT	$\bar{x}$	57.4	4.5	2.2	1.6	2.0	2.5	2.0	2.0
	±SD	9.4	8.3	5.6	5.3	7.8	5.3	3.6	3.9
MAP (mmHg)									
HG	$\bar{x}$	68.7	7.4	1.5	1.6	1.9	2.6	4.6	4.3
	±SD	6.1	5.4	3.6	6.0	4.4	4.1	4.6	4.8
CPT	$\bar{x}$	72.6	7.0	4.1	2.1	2.7	3.6	3.0	2.8
	±SD	8.7	8.5	6.4	4.9	7.6	5.6	4.4	3.5
HR (beat/minute)									
HG	$\bar{x}$	69.0	0.3	-0.8	-0.5	-0.7	0.0	0.3	-1.6
	±SD	10.9	5.6	4.8	6.9	6.6	6.2	4.8	4.8
CPT	$\bar{x}$	67.5	3.4	-1.5	-3.4	-1.4	-0.1	1.4	0.6
	±SD	9.5	4.0	4.2	5.2	7.0	6.0	5.0	5.0

Notes: CPT = Cold pressor test HG = 33% MVC handgrip

Δ = Values are mean ±SD expressed as different score between the pre-test value and the response value

R: Average value for 15 second prior to tests

P1-P4: Average value for 15s periods during the first minute of the post-tests

P5-P7: Value averaged over 1-min period from the second minute to the fourth minute of the post-tests

TABLE 4:

Cardiovascular responses to HG performed alone (condition 1), simultaneously with CPT (condition 3) and preceded by 1 minute of CPT (condition 4)

		R	15s	30s	45s	60s
			T1(Δ)	T2(Δ)	T3(Δ)	T4(Δ)
SBP* (mmHg)						
Condition 1	$\bar{x}\pm$SD	108.7±11.4	10.0±7.5	14.0±10.1	14.7±9.0	16.3±7.2
Condition 3	$\bar{x}\pm$SD	119.1±13.3	17.0±7.4	18.6±11.8	23.4±12.6	28.3±9.6
Condition 4	$\bar{x}\pm$SD	132.9±16.9	5.9±11.4	9.5±15.9	7.4±12.8	7.8±14.5
DBP (mmHg)						
Condition 1	$\bar{x}\pm$SD	54.1±7.0	5.2±2.9	8.9±3.5	10.0±3.8	12.2±4.3
Condition 3	$\bar{x}\pm$SD	57.3±9.7	5.0±4.2	6.0±6.6	10.8±8.3	14.7±6.8
Condition 4	$\bar{x}\pm$SD	64.5±5.9	4.0±5.0	7.2±5.1	8.7±5.4	10.6±7.9
MAP (mmHg)						
Condition 1	$\bar{x}\pm$SD	68.7±6.1	5.9±3.6	9.4±5.0	10.8±4.8	12.8±4.7
Condition 3	$\bar{x}\pm$SD	72.9±8.6	7.6±3.7	8.4±8.1	13.4±8.8	17.6±6.7
Condition 4	$\bar{x}\pm$SD	81.8±7.5	4.9±5.7	8.0±6.7	9.1±6.5	10.5±8.9
HR* (beat/min)						
Condition 1	$\bar{x}\pm$SD	69.0±10.9	2.7±3.6	3.3±6.6	3.8±3.8	4.5±4.1
Condition 3	$\bar{x}\pm$SD	72.5±13.4	11.9±5.3	10.2±9.2	9.9±8.3	9.3±8.4
Condition 4	$\bar{x}\pm$SD	74.6±8.8	0.6±5.2	0.8±4.9	1.8±4.9	3.5±6.3

Note: R: Average value for 15s period prior to HG

T: Average value for 15s period during the first minute of HG

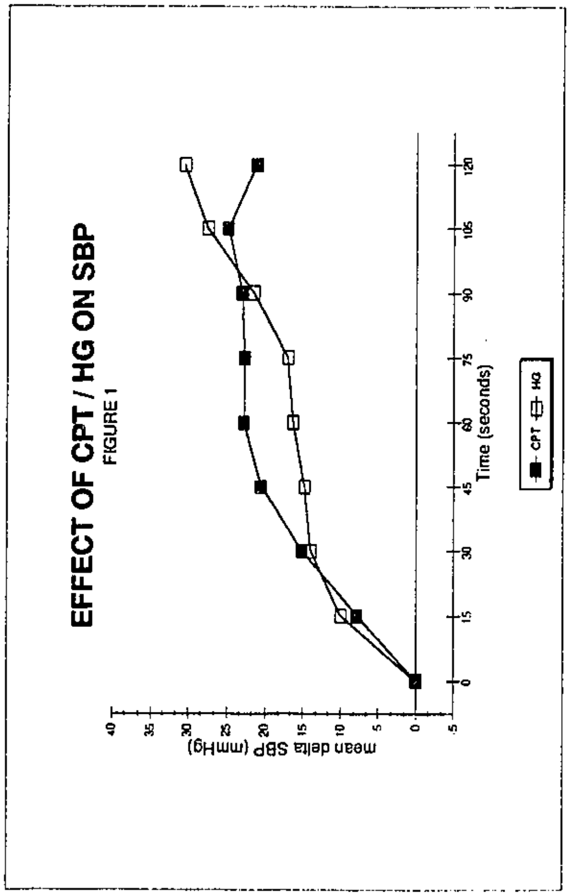
Δ: Values are mean ±SD expressed as difference score related to pre-exercise values.

*: Overall significant differences in variables between condition 3 vs condition 1, condition 3 vs condition 4, and condition 1 vs condition 4 at $p<.05$

Condition 1 = HG performed without CPT

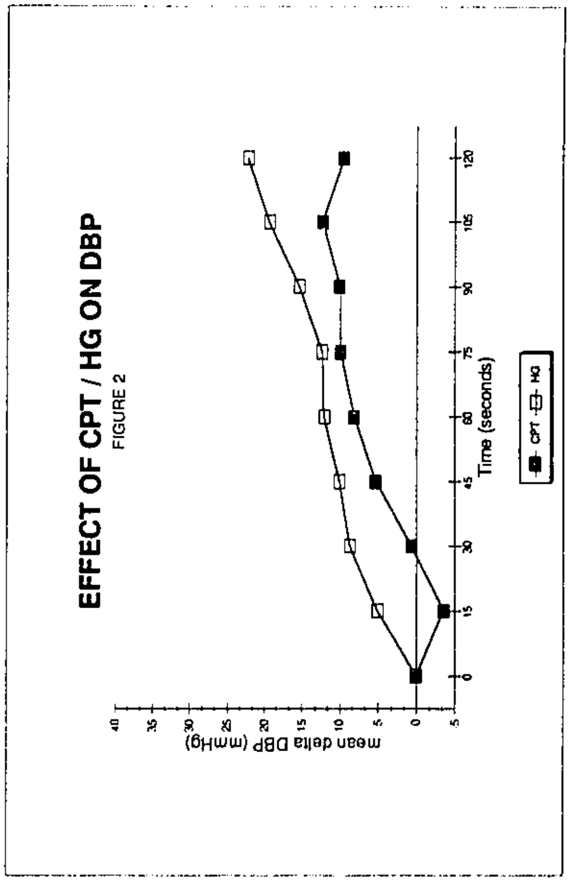
Condition 3 = HG with simultaneous CPT

Condition 4 = HG performed during the last minute of a 2-min CPT



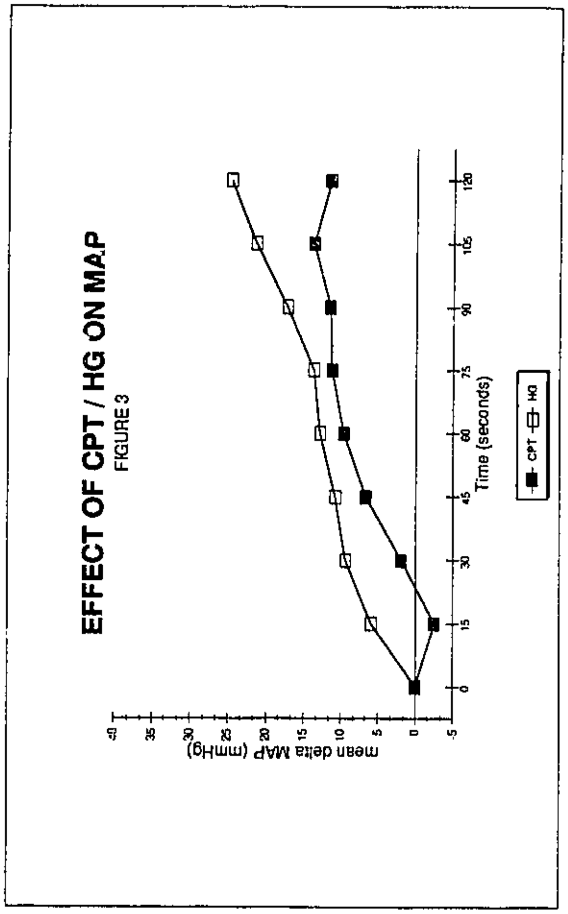
EFFECT OF CPT / HG ON SBP

FIGURE 1



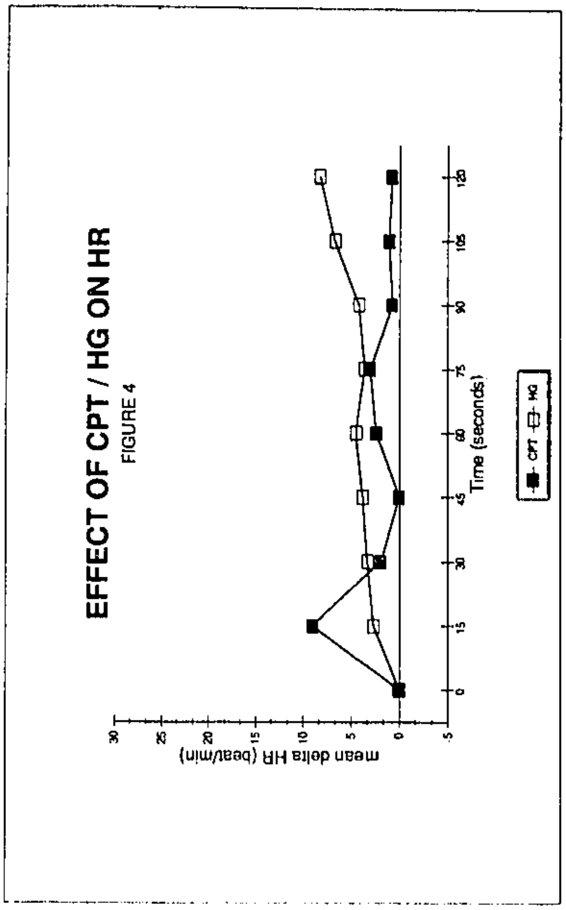
EFFECT OF CPT / HG ON DBP

FIGURE 2



EFFECT OF CPT / HG ON MAP

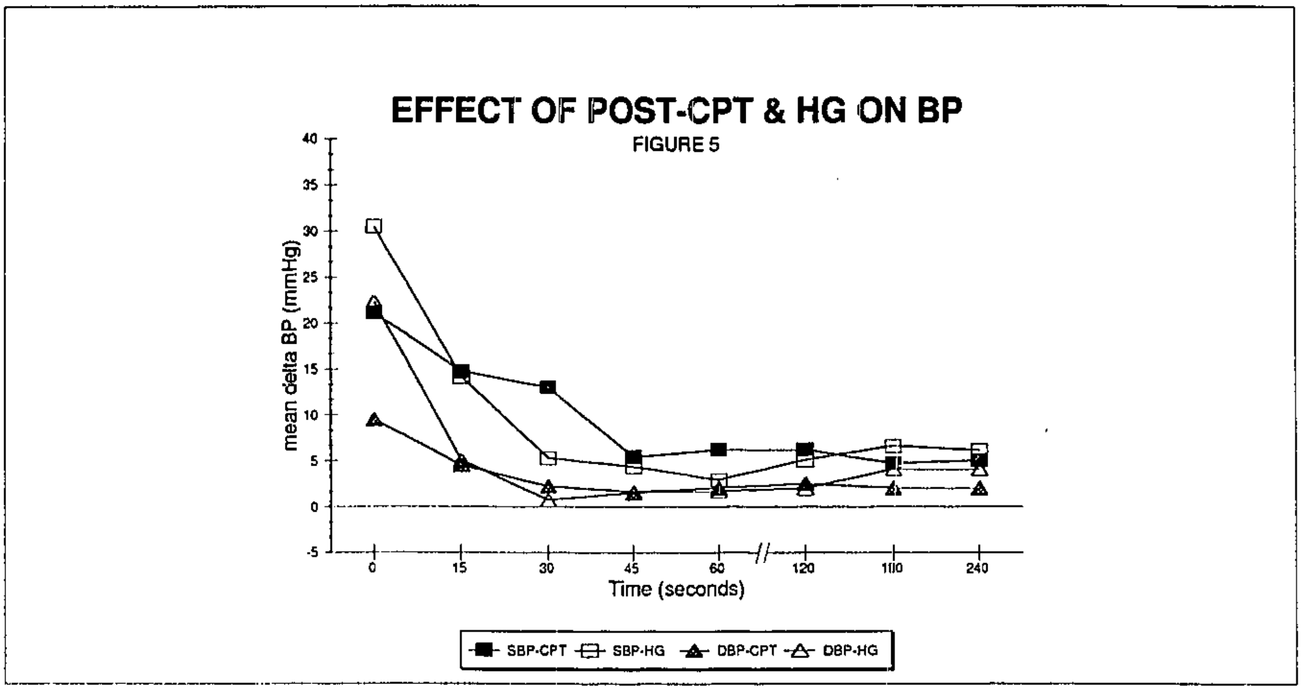
FIGURE 3



EFFECT OF CPT / HG ON HR

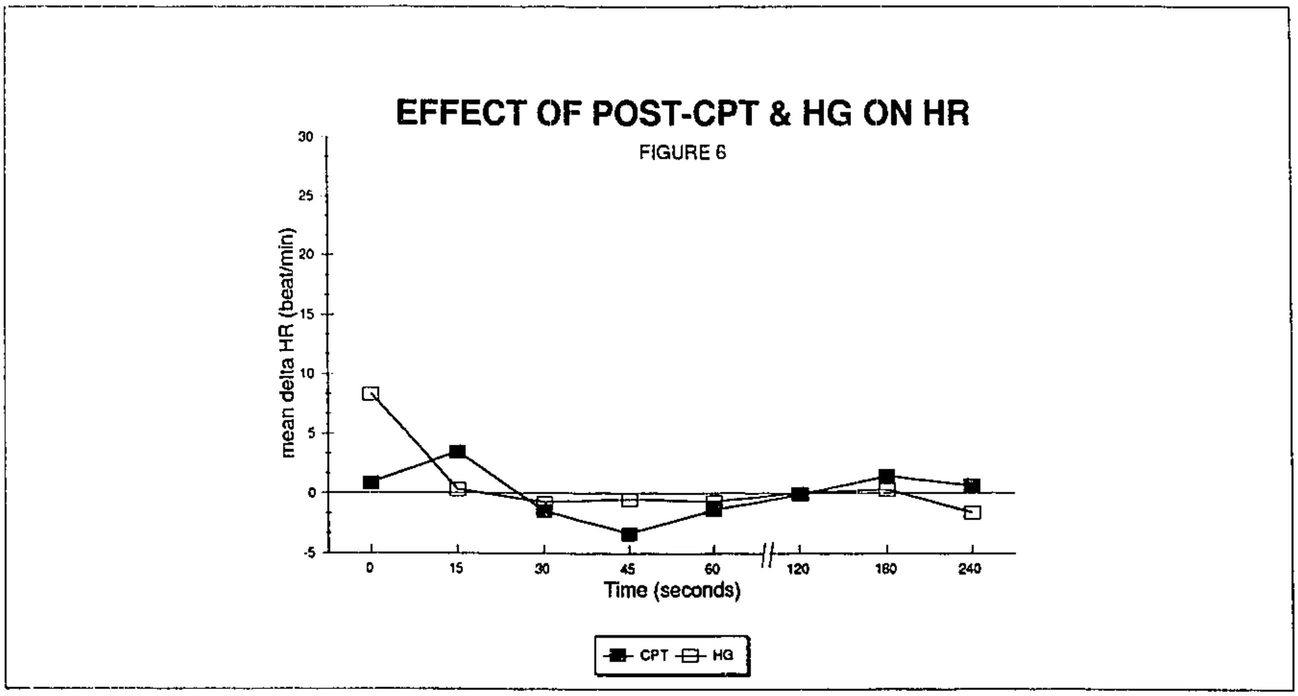
FIGURE 4

Figs 1-4



EFFECT OF POST-CPT & HG ON BP

FIGURE 5

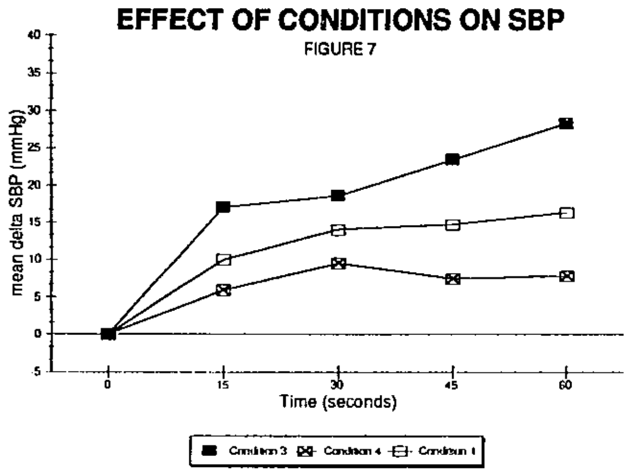


EFFECT OF POST-CPT & HG ON HR

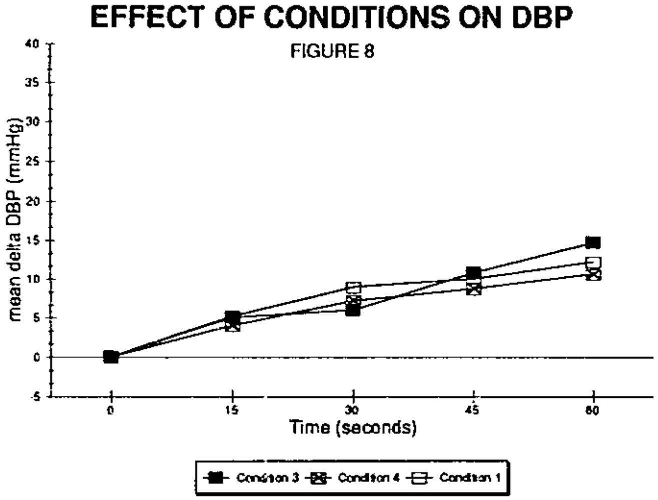
FIGURE 6

Figs 5-6

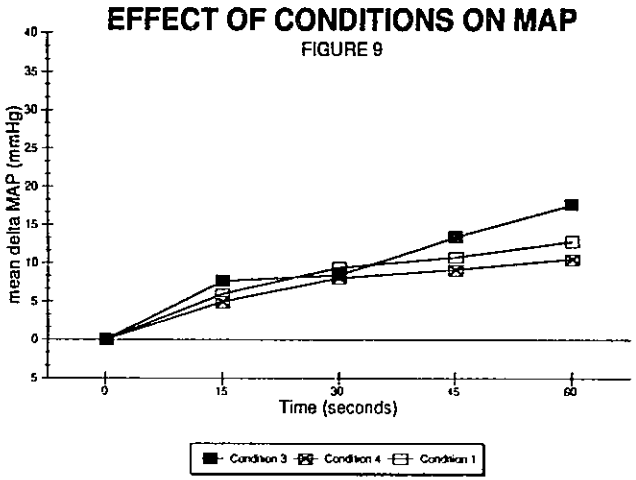
Figs 7-10



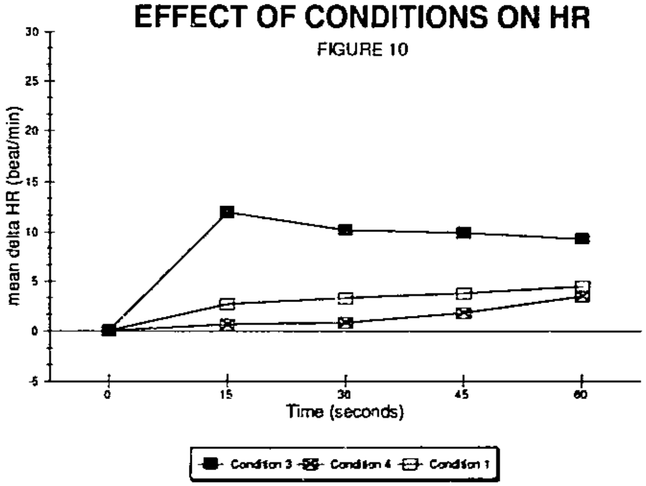
EFFECT OF CONDITIONS ON SBP
FIGURE 7



EFFECT OF CONDITIONS ON DBP
FIGURE 8



EFFECT OF CONDITIONS ON MAP
FIGURE 9



EFFECT OF CONDITIONS ON HR
FIGURE 10

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CHAPTER I

INTRODUCTION

1.1 Introduction

The pressor response to isometric exercise (Lind et al., 1964; Michael et al., 1979; Asmussen, 1981; Mitchell et al., 1983) is the result of an increase in cardiac output with little or no change in total peripheral vascular resistance (Shepherd et al., 1981 and Laird et al., 1979). The increase in cardiac output during isometric exercise is due to an increase in heart rate (HR) (Keul et al., 1981). The initial increase in HR is caused by vagal withdrawal (Freyschuss, 1970), whereas sympathetic nervous activity (SNA) causes a further increase in HR (Martin et al., 1974 and Mark et al., 1985). During isometric exercise at a tension of about 60% to 70% of maximal voluntary contraction (MVC), blood flow is occluded through the contracting muscle because of higher intramuscular pressure (Humphreys et al., 1963 and Byrd et al., 1971). The muscle contraction produces metabolites which may activate group III and IV afferents (Kaufman et al., 1984) and induce a reflex pressor response to isometric exercise (McCloskey and Mitchell, 1972; Mitchell et al., 1983; Waldrop et al., 1984). It has been shown that the pressor response to isometric exercise is the results of both central control (Mitchell, 1985) and peripheral adaptation (Mitchell et al., 1983). Following an isometric contraction, blood pressure (BP) rapidly returns to resting level (Frisk-Holmberg et al., 1983 and Peikert and Smolander, 1991).

Several studies have shown that the increase in HR as well as systolic blood pressure (SBP) and diastolic blood pressure (DBP) during the cold pressor test (CPT) (Folisis et al., 1978, pp 267-277; LeBlanc et al., 1978; LeBlanc et al., 1988; Frey et al., 1980; Seals, 1989) is due to an increase in SNA (LeBlanc et al., 1978; Fagius et al., 1989). During local cooling, the cutaneous vasoconstriction may increase peripheral vascular resistance and afterload of the heart (LeBlanc et al., 1978; Shephard, 1987). Therefore, during cold exposure, systemic blood pressure (BP) increases in part because of an increase in cardiac output and in part due to high cutaneous vasoconstriction. Also, the cold stimulation of thermal receptors and nociceptors causes a reflex increase in BP (Lloyd, 1986, p81; Lovallo, 1975a).

Thus, the BP response to CPT and to isometric exercise, when applied separately, has been shown to be similar, although the mechanisms resulting in the pressor response are different (Frisk-Holmberg et al., 1983). Yet, only a few studies have evaluated the effects of isometric exercise combined with the cold pressor stress on cardiovascular function (Humphreys et al., 1963; Petrofsky et al., 1981b; Peikert and Smolander 1991). Humphreys and coworkers (1963) reported that BP response to sustained contraction to the point of fatigue at 30% to 70% MVC was independent of temperature when the hand and forearm were immersed in a water bath for 30 minutes at temperatures of 10 to 42°C. Petrofsky and coworkers (1981b) observed that an increase in BP response to sustained contraction (40% MVC) after a 30-min hand immersion was almost constant at temperatures of 22 to 40°C, but that there was an increase and then a reduction in the magnitude of both SBP and DBP for contractions exerted at colder temperature (3°C and 10°C). More recently, Peikert and Smolander (1991) investigated the influence of the CPT (immersion of left hand in ice-water) during the last minute of a 3-min isometric knee extension exercise (15% MVC) on HR and BP. Their data indicated that SBP, DBP, HR, and pressure rate product were higher when the isometric exercise was combined with the CPT in comparison to the isometric exercise performed without immersion.

According to these studies, an application of a local cold stress will potentiate the BP response to isometric exercise. However, most of the previous studies were either conducted in water temperature above 10°C or employed a low level of MVC so that little is known concerning BP and HR responses to isometric exercise when combined with CPT applied either at the onset or during immersion. It may be that the pressor response depends, in part, on whether the isometric exercise is performed at the onset of cold application or once the response to the cold application has stabilized. Furthermore, the blood pressure in these studies was measured by auscultation, such that an immediate and continuous measurement of the pressor response was not obtained.

1.2 Statement of the problem

The purposes of this study were to (1) compare the pressor response between isometric exercise and a CPT and (2) examine the pressor response to isometric exercise (33% MVC) combined with a CPT (cold foot immersion in ice-water) applied either at the onset or during the last minute of a 2-min CPT (2° C to 4° C).

1.3 Hypothesis

1. HG at 33% MVC combined with simultaneous cold foot immersion would cause an additive pressor response.
2. A 1-min cold foot immersion prior to HG would attenuate the pressor response.

1.4 Scope of the study

Ten male subjects between the ages of 20 and 35 years was recruited for the study. MVC of the dominant hand was determined using the average of three maximal handgrip contractions. CPT was applied by immersing the right foot in an ice water bath (2 to 4° C). Following an introductory session, the following testing sessions were administered randomly: (1) HG (2 minutes) and CPT (2 minutes) performed separately, (2) HG in combination with CPT (2 minutes), and (3) HG performed during the last minute of a 2-min CPT. SBP, DBP, HR, and mean arterial pressure (MAP) prior to, during and following each test were measured using a Finapres 2300 BP Monitor.

Data was reported as mean values and standard deviation (SD). A student's t-test was used to test the difference between CPT and HG performed alone on blood pressure and heart rate responses. A factorial design analysis of variance (ANOVA) with repeated measurements and trend analysis design was used to determine if there are any statistical significant differences in SBP, DBP, MAP, and HR responses to HG performed either at the onset, during, or without

CPT. If there was a statistical significant F-ratio, a post-hoc analysis would be used in order to localize significant differences among means. For all statistical analyses, the significant level would be set at $P<0.05$.

1.5 Definition of terms and abbreviations

Isometric Contraction

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

Maximal Voluntary Contraction (MVC)

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

Pressor Response

The abrupt increase in blood pressure following isometric exercise or cold pressor test

Cold Exposure

Any environmental condition where the potential exists to lose a significant amount of heat from a region of the body

Cold pressor test

An immersion of an extremity in ice water for a specific period of time.

CHAPTER II

REVIEW OF LITERATURE

2.1 Introduction

The purposes of this study were to (1) compare the pressor response between isometric exercise and a CPT and (2) examine the pressor response to isometric exercise (33% MVC) combined with a CPT (cold foot immersion in ice-water) applied either at the onset or during the last minute of a 2-min CPT (2°C to 4°C). The literature review would examine the mechanisms and the factors which elicit the cardiovascular response to isometric exercise and to a CPT.

2.2 Cardiovascular response to isometric exercise

Isometric exercise primarily involves a change of muscle tension with no change in muscle length. In our daily life, many activities are related to isometric exertion which induce cardiovascular responses. The mechanisms of the cardiovascular response to isometric contraction are associated with local and systemic physiological changes.

2.2.1 Hemodynamic response

During isometric exercise, even when a small muscle mass is involved, SBP, DBP and MAP increase dramatically. Yet, HR and cardiac output increase relatively little, while stroke volume and peripheral vascular resistance remain essentially unchanged (Pollock and Schmidt, 1979). The change in BP and HR result from a direct action of a central command from the cardiovascular centre and a peripheral component from active muscle.

Heart rate: Muscle sympathetic nervous activity (MSNA) is not increased at the onset of isometric exercise (Mark et al., 1985). The initial increase in HR is due to vagal withdrawal

(Freyschuss, 1970; Maciel, et al. 1987). The loss of baroreflex sensitivity is also in part associated with the increase in HR (Cunningham et al., 1972; McCloskey and Streateild, 1975). The sympathetic effector response appears later to further cause an increase in HR (Martin et al., 1974).

Cardiac output: During isometric exercise, stroke volume has been shown to remain unchanged or to increase slightly (Laird et al., 1979; Keul et al., 1981), because there is no change in total peripheral vascular resistance and the amount of venous blood returned to the heart. Therefore, an increase in cardiac output during isometric exercise is mainly due to the increase in HR (Keul et al., 1981 and Shepherd et al. 1981). During maximal isometric exercise, the increase in cardiac output can be maintained for a short time only, because decreased venous return will limit the pumping capacity of the heart (Keul et al., 1981).

Peripheral vascular resistance: In a human experiment of static handgrip at a tension of 30% MVC for 2 minutes, Saito and coworkers (1990) found that calf vascular resistance (CVR) in the resting limbs increased within 1.5 minutes. Thereafter, it did not increase further, while the MSNA was still increasing. However, the total peripheral vascular resistance remains unchanged (Laird et al., 1979; Pollock and Schmidt, 1979) in spite of the increase in BP during isometric exercise. This may be due to a reflex vasodilatation from baroreceptor stimulation (Martin et al., 1974).

Blood pressure: BP is the product of cardiac output and total peripheral resistance (Guyton, 1971, p219). Any factor which influence either cardiac output or peripheral resistance will cause a change in BP. Therefore, an increase in BP during isometric exercise is due to an increase in cardiac output with little or no change in total peripheral vascular resistance (Laird et al., 1979). Vasoconstriction due to sympathetic stimulation is also a mechanism for increasing blood pressure (Martin et al., 1974). Moreover, muscle contraction in part induces a rise in BP to overcome the intramuscular pressure for perfusion of the active muscle (Shephard, 1987, p77). Intramuscular fluid pressure increased linearly with force up to MVC (Sejersted et al., 1984). However, at the end of fatiguing contractions at tension above 15% MVC, the magnitude of the pressor response is independent of the tension exerted by the muscle (Petrofsky et al., 1981a).

This may be due to a recruitment of all motor units (Petrofsky et al., 1981a). During post-isometric exercise, BP drops rapidly (Mark et al., 1985). This is caused by reduced baroreceptor activity and post-exercise hyperaemia of the exercising muscle (Morales et al., 1991; Taylor et al., 1988).

Blood flow: Intravascular pressure can restrict blood flow to the exercising forearm muscles (Kilbom and Persson, 1981). Blood flow during both low- and high-intensity isometric contractions is influenced not only by intramuscular pressure, but also by vasodilating substances (Byström and Kilbom, 1990). A large increase in intramuscular pressure during heavy static exercise lowers effective perfusion pressure and limits muscle blood flow (Lewis et al., 1985). Occlusion of blood flow through the contracting muscle occurs at about 60 to 70% MVC (Humphreys et al., 1963; Byrd et al., 1971). Taylor and coworkers (1988) reported that the level of post-exercise hyperaemia is directly related to HR, SBP and exercise intensities. The authors suggested that the level of hyperaemia may indicate the level of O_2 deficit, which consequently induces release of vasodilator metabolites that produces a stimulus for muscle chemoreflex activation. Saito and coworkers (1990) suggested that during post-handgrip (30% MVC), the local effect of vaso-active metabolites around the vessels might cause a remarkable increase in blood flow in the resting leg. In a study on physiological response to continuous and intermittent HG with 10%, 25% and 40% MVC, Byström and Kilbom (1990) found that blood flow through the active forearm decreased markedly as the contraction intensity increased. The greatest difference was seen between 10% and 25% MVC. However, maximal post-exercise blood flow showed no significant difference between 25% and 40% MVC, because vasodilating metabolites have already reached their maximal effect at 25% MVC, and the sympathetic vasoconstriction interferes with maximal vasodilation.

To summarize, isometric exercise is characterized by a decrease in vagal outflow at the onset of exercise and an increase in sympathetic stimulation as the exercise progresses. As a result, even without an increase in total peripheral vascular resistance, cardiac output, HR, MAP, SBP and DBP are all increased. During isometric exercise, occlusion of blood flow through the contracting muscle occurs at tensions above 60% MVC. Post-exercise hyperaemia in exercising muscle is related to vaso-active metabolites. Those changes in cardiovascular response during isometric exercise are related to central and peripheral reflex control mechanisms.

2.2.2 Central control mechanism

In his review of 1985, Mitchell stated that central control mechanism is related to the recruitment of motor units (central command). The level of central command can be measured by electromyographic (EMG) activity (Taylor et al., 1988).

Muscle fibre recruitment: Mitchell and coworkers (1979) determined the role of muscle mass recruitment on cardiovascular response to static exercise in healthy males. Their study indicated that the number of motor neurons activated to produce a given force with arm muscles may be larger than when using the leg muscles, because the number of muscle fibres in a motor unit is less in the arms than in the legs. It was suggested that activation of greater motor units generated a larger contribution from both central and peripheral components and, as a result, increased BP and HR. Furthermore, Mitchell and coworkers (1981) examined the BP response to isometric contractions of leg muscles in previously injured soccer players. The normal and larger leg was determined to be 50 kg and that for the previously injured and smaller leg was 30 kg. Their results showed that a greater increase in SBP and electromyographic activity (EMG) was in the larger leg when each leg was contracted at the same tension of MVC. It was suggested that if a greater skeletal muscle mass was contracted, there would be a greater input from receptors in the skeletal muscle, and a greater number of centrally activated motor units (the increased central command). Thus, the increase in EMG activity during isometric exercise is caused by an increased central command. Similarly, according to the Asmussen's study (1981), there were more signals sent to the medullary cardiovascular centre during a larger muscle fibre recruitment. Therefore, it could be said that the muscle fibre recruitment is matched by the central activity.

Central command: Goodwin and coworkers (1972) investigated the central control mechanism with vibration and without vibration stimulus of the biceps during isometric exercise in human subjects. The results indicated that when a similar force but less central command was incorporated, BP and HR were lower than when similar force but greater central command were utilized. It was suggested that there is central modification to the magnitude of cardiovascular response which is dependent on the descending central command during isometric exercise. Funderburk and coworkers (1974) suggested that during isometric exercise, HR response may reflect the combined effect of an early central command, and mechanical and metabolic factors. In the 1980's, the studies concerning the influence of the central nervous system on BP and HR responses during isometric exercise have agreed with that the central command in part plays a role in the rise of BP and HR (Mitchell et al., 1981; Rusch et al., 1981; Victor et al., 1989). In addition, Shepherd and coworkers (1981) suggested that the central command impulses may arise in the motor cortex, other motor areas, and/or nonmotor areas (arousal or emotion).

In brief, central command involves the recruitment of motor units. Increased central drive would elicit a greater number of motor units, while a larger muscle mass would evoke more reflex contribution from active muscle nerve endings. The central control is partially responsible for cardiovascular response to isometric exercise.

2.2.3 Reflex control mechanism

Muscle contraction which stimulates afferent endings within the skeletal muscle can produce a reflex cardiovascular change during isometric exercise (Mitchell et al., 1983).

Muscle fast twitch fibres: Petrofsky and Lind (1980) suggested that the magnitude of the pressor response is affected by the degree of muscle fast twitch (FT) fibre involvement. Frisk-Holmberg and coworkers (1983) also reported that the individual with a higher percentage of fast twitch fibres had a higher BP response to isometric exercise. It was suggested that this may be due to the BP response having triggered by the stimulation of locally formed metabolites and mediated via the sympathetic nervous system. More recently, White and Carrington (1993) concluded that the peripheral input to the pressor response is linked to FT fibre activation during isometric exercise.

Spinal dorsal roots: Coote and coworkers (1971) demonstrated that the reflex response is abolished by sectioning the dorsal root afferent from the active muscles in cardiovascular response to isometric exercise. In a cat experiment using nerve-blocking techniques, McCloskey and Mitchell (1972) showed that during the anodal dorsal roots block, the muscular contraction of the cat limb produced the same reflex pressor response as the control experiment and that the pressor responses were abolished by cutting the dorsal roots. It was suggested that reflex cardiovascular response is initiated within the muscle. Similarly, Mitchell and coworkers (1977) demonstrated that electrically stimulating the peripheral ends of sectioned spinal ventral roots still causes an increase in cardiovascular response during the induced isometric exercise. The section of the corresponding dorsal roots indicated that the cardiovascular changes reflectively originate from contracting skeletal muscle. Thus, during isometric exercise, peripheral reflex originates from contracting muscle and transmits from peripheral to centre through a neuronal pathway located in the dorsal root.

Group III and IV afferents: The afferents from skeletal muscles have been divided into four groups (group I, II, III, and IV) based on both anatomical and electrophysiological measurements (Mitchell,1985). McCloskey and Mitchell (1972) examined the influence of the myelinated and unmyelinated afferent in the cardiovascular response during isometric exercise by using nerve-blocking techniques. The findings indicated that group I and group II fibres were not involved in reflex cardiovascular response during isometric exercise, but the fibres in either or both group III (small myelinated fibres) and group IV (unmyelinated C-fibres) are responsible for the cardiovascular response. In a cat experiment using nerve techniques and succinylcholine injection, Waldrop and coworkers (1984) also demonstrated that group I and II afferent fibres cannot affect BP and HR responses to static contractions. Yet, the muscular contraction would cause increased firing of group III and IV afferents. It was concluded that only group III and IV are involved in the reflex cardiovascular response during isometric exercise. Furthermore, muscle metabolism is linked with afferent nerve activity through the thermosensitivity of group III and IV, and an increase in muscle temperature may elevate firing rate in more than 50% of the group III and IV afferents (Rowell, 1986, p291-292). In their review of 1983, Mitchell and coworkers suggested that the Group III muscle afferents are very mechanosensitive as ergoreceptors, and group IV muscle afferents may function as both ergoreceptors and nociceptors, which are responding to mechanical or metabolic factors. Therefore, it seems clear that during isometric exercise, the reflex cardiovascular changes are due to afferent impulses being conducted by the Group III and IV afferent as ergoreceptors, which respond to mechanical events. Metabolites due to ischemia may also stimulate these afferents.

Metabolites: Kaufman and coworkers (1984) found that a population of group III and IV muscle afferents were stimulated more by static contraction with occlusion of blood flow to contracting muscle than by normal contraction when tension developed by both contraction was the same. It was suggested that ischemic contraction produces more metabolites. Mitchell and coworkers (1983) found that there is a different BP response between induced sustained contractions of slow and fast twitch fibres, because of difference in blood flow, metabolism, and sensitivity of group III and IV afferent nerve endings between slow and fast twitch fibres. Petrofsky and coworkers (1981c) suggested that if the magnitude of the pressor response is related to the release of a metabolite such as K^+, it is possible that the proportion of slow-twitch motor units may modify the release of the metabolites. In his review of 1985, Mitchell concluded that the metabolic factor may contribute to the reflex cardiovascular responses during isometric contraction at a level of exercise intensity which restricts blood flow. Moreover, potassium as a metabolic product may activate Group III and IV afferents (Kaufman et al. 1984). Shepherd and coworkers (1981) suggested that potassium ion efflux from contracting muscle is likely correlated with a rise in BP. Byström and coworkers (1991) found that in the continuous handgrip, intermittent handgrip, and intermittent handgrip with electrical stimulation at 25% MVC in human, potassium ion increased within 3 minute of the exercises to a steady-state level of about 5.0-5.4 $mmol.l^{-1}$ (The rest level was 4.2 $mmol.l^{-1}$.). Therefore, the cardiovascular response to isometric exercise is in part induced by the stimulation of Group III and IV afferent fibres due to increased production of metabolites, especially potassium ion.

Therefore, isometric contraction at a tension which restricts blood flow or with occlusion of blood flow through active muscle could produce metabolites, which may stimulate group III and IV afferents. In this way, an isometric muscle contraction involves a peripheral feedback reflex mechanism for the control of the cardiovascular response to isometric exercise.

2.2.4 Autonomic nervous system control

The autonomic nervous system is responsible for the regulation of sympathetic and parasympathetic nerve activity to the heart and blood vessels in order to match the hemodynamic response to the intensity of the isometric exercise (Mitchell, 1985).

Sympathetic and parasympathetic nervous activities: Freyschuss (1970) investigated the sympathetic and parasympathetic nervous outflows adjusting the initial cardiovascular response to isometric contraction in humans by using atropine and phentolamine. The marked inhibition of the BP response under the influence of both atropine and phentolamine indicated that a rise in BP is mediated by alpha-adrenergic receptor, and that the HR modulation during the initial part of the contraction is elicited by a withdrawal of vagal tone. Similarly, Martin and coworkers (1974) found that the initial increase in HR during isometric exercise is absent with the administration of atropine, even though the later rise in HR still occurs. But, the typical HR response to isometric exercise is lower with the administration of popranolol. After combined atropine-popranolol is administered, the HR response is completely inhibited throughout the entire period of exercise. It was concluded that the vagal withdrawal is the first mechanism in heart rate response to HG and that the sympathetic outflow corresponds to the later HR response. More recently, Mitchell amd coworkers (1989) have also suggested that the early HR response to isometric exercise is related to vagal withdrawal. Therefore, both sympathetic and parasympathetic activities modulate the cardiovascular response to isometric exercise.

Baroreflex modulation: Baroreflex activity plays an important role in BP response to exercise (Rowell, 1986, p247-249). During isometric exercise, a decrease in systemic vascular resistance could be in part due to reflex vasodilatation from baroreceptor stimulation (Martin, 1974). An increase in HR as well as BP is associated with a lower baroreflex sensitivity, while a reduction in HR is related to an increase in baroreflex sensitivity (Cunningham, et al., 1972).

McCloskey and coworkers (1972) suggested that the sensitivity of the baroreceptor reflex is reduced during voluntary exercise. Similarly, Cunningham and coworkers (1972) found that reflex sensitivity was decreased by rhythmic grip, and total circulatory occlusion to the exercising arm did not change the reflex response during handgrip. It was concluded that metabolic rate and baroreflex activity have various relationships during the different types of exercise. Abboud and coworkers (1981) suggested that HR and vasoconstriction were decreased by baroreflex to depress the activation of Group III and IV afferents during isometric exercise. Ebert (1986) examined carotid baroreflex function during HG in conscious humans. A five second neck suction of 30 mmHg was applied immediately prior to the beginning of HG at 20% MVC. This 30 mmHg neck suction response was termed as "anticipatory" to the onset of handgrip. The results showed that the R-R interval and the MAP responses to similar carotid sinus distending pressure were decreased progressively from control to anticipation of exercise onset and to HG. It implicated an important influence of central command on baroreflex function during anticipation of voluntary muscular exertion. More recently, Eckberg and Wallin (1987) examined the baroreceptor activities during HG with and without neck pressure. Their findings indicated that HG without neck pressure significantly shortened the average R-R intervals but did not significantly change average MSNA. However, during HG with neck pressure, the average R-R intervals were reduced slightly, but MSNA increased significantly. It was suggested that sympathetic responses to change of baroreceptor input are modified by isometric exercise. Thus, handgrip exercise may affect human carotid baroreflex control of BP and HR (Ebert, 1986). The vagal-cardiac control mechanism which is related to baroreflex response is in part responsible for cardiovascular response to isometric exercise.

Thus, an increase in sympathetic efferent nerve activity and a decrease in parasympathetic efferent nerve activity during isometric exercise have shown the role of the autonomic nervous control in the modulation of BP response to isometric exercise by baroreflex activity.

2.2.5 Muscle mass

Active muscle size is an important factor in the regulation of sympathetic nervous system discharge and cardiovascular function during isometric exercise in human (Seals, 1993). Mitchell and coworkers (1979) investigated the BP and HR responses to a sustained static contraction at 40% MVC. Different muscle mass were represented by finger abduction, handgrip, leg extension and a combination of handgrip and leg extension. The results indicated that the HR and BP increased with the increase in muscle mass utilized. It was suggested that intramuscular pressure may be different with different muscle mass involved at the same intensity. The large muscle mass utilized could activate more motor units through central and peripheral drives. Similarly, Seals and coworkers (1983) examined the cardiovascular responses to 30% MVC of three different muscle movements (handgrip, leg extension and dead lift) in young men. The results showed that the cardiovascular response to the isometric exercise at the same intensity level is directly dependent on the active muscle mass. The same results have been reported by Lewis and coworkers (1985), Mitchell and coworkers (1981), Nagle and coworkers (1988) and Misner and coworkers (1990). It was suggested that peripheral receptors may be activated by metabolites which are associated with muscle mass involved and not with the tension developed (Mitchell et al., 1981). Therefore, during isometric exercise, the amount of muscle mass utilized can affect the magnitude of the cardiovascular response, because different muscle mass involved at the same intensity may have different intramuscular pressure and different number of activated motor units.

2.2.6 Intensity

There are controversial findings about whether the static tension exerted could influence the magnitude of HR and pressor responses to isometric exercise. Lind and coworkers (1964) reported that magnitude and rate of increase of cardiovascular response are related to the tension exerted. Similarly, Quarry and coworkers (1974) found that the absolute increase in SBP and DBP are directly related to the degree of exertion. Riendl and coworkers (1977) also suggested that although the size of muscle mass influences the DBP and HR responses, the relative muscle tension is the major factor determining the hemodynamic responses to isometric exercise. Moreover, McCloskey and Streateild (1975) suggested that the pressor response is related to the tension achieved, not muscle mass utilized. However, Funderburk and coworkers (1974) demonstrated that during isometric exercise, although the magnitude of HR was directly related to the increase in tension, both the pattern of pressor response and the level of BP reached at the point of fatigue were the same at tensions of 20, 40, 60% MVC. It was concluded the BP response is unaffected by the tension exerted. Mitchell and coworkers (1981) found that the pressor response during sustained contraction at 40% MVC elicited progressive elevations when the muscle mass was increased. Following occlusion of the muscle after isometric contraction, the BP also increased with a larger contribution of muscle mass. In agreement with their studies, in the later 1980's, a number of studies have suggested that the magnitude of cardiovascular response to isometric exercise is positively related to muscle mass but not tension (Lewis et al., 1985; Mitchell et al., 1981; Nagle et al., 1988 and Misner et al., 1990).

2.2.7 Other factors

Besides muscle mass and intensity utilized, previous studies have showed that there are several factors which might or might not influence the cardiovascular response to isometric exercise. Quarry and coworkers (1974) demonstrated that there was no posture influence in heart rate at the end of maximal voluntary contraction, and also no significant influence of sitting and supine position in BP response to isometric exercise. Iellamo and coworkers (1993) confirmed that the pressor response to isometric exericse was not significantly influenced by change in posture, either in magnitude or in time course. Baldwa and coworkers (1983) showed that HR and BP rise significantly higher in smoking subjects when compared to non-smokers of the same age. It was suggested that this is due to reduction in the oxygen transporting capacity of the blood associated with a corresponding reduction of working capacity. Grucza and coworkers (1991) found that increases in cardiac output and SBP did not differ substantially between trained subjects and untrained subjects. However, in trained subjects, the increased level of cardiac output was an effect of greater stroke volume and lower HR than that in untrained subjects. Aging and gender also play important roles in cardiovascular response to isometric exercise.

Petrofsky and coworkers (1975) reported that isometric strength was greater in men than in women. Similarly, Davies and coworkers (1988) investigated the gender difference in the relationship of performance in the HG to lean limb volume in young adults. For a given lean volume, the HG performance of men (467N) was significantly superior to that of the women (298N). Sanchez and coworkers (1980) indicated that during sustained isometric exercise plasma epinephrine and norepinephrine level rose rapidly for men but remained unchanged for women. However, there are contradictory findings on cardiovascular response to isometric exercise based on gender difference. Petrofsky and coworkers (1975) and Sanchez and coworkers (1980) suggested that increases in HR and BP were similar for women and men in handgrip contraction. But Rogowsky and coworkers (1978) found that during sustained isometric exercise the increase in HR was more intense in women, whereas the elevation in BP was more striking in men. The authors suggested that such results could reflect sex differences in adrenergic reactivity (cited Sanchez et al., 1980).

Age might influence cardiovascular response to isometric exercise. Petrofsky and coworkers (1975) examined the influence of age on sustained isometric exercise in men and women. The results showed that at the end of a 40% MVC isometric exercise, older subjects had a smaller increase in exercising HR, compared to younger subjects. The SBP at the end of the 40% MVC was affected by both the resting SBP and age. It was suggested that this was the result of aging which is accompanied by a decrease in the flexibility of the arteries as well as a decrease in aortic compliance. These authors also showed that strength decreased and endurance increased with age. It was suggested that this reflects the lower absolute work load during isometric contraction in older people. Similarly, Baldwa and coworkers (1983) found that isometric strength decreased with age.

To summarize, isometric contraction induces an increase in HR, SBP and DBP due to the central command, peripheral adaptation, and autonomic nervous activity. The change in cardiovascular responses to isometric exercise is influenced by many factors, such as muscle mass, age and sex.

2.3 Cardiovascular response to cold pressor test (CPT)

The CPT produced by immersion of an extremity in ice water can induce an initial cutaneous vasoconstriction, and increases in BP and HR. During the CPT, most subjects experience pain in the immersed limb (Zbrozyna and Krebbel, 1985). This cold-induced discomfort may be the result of the vasoconstriction (Lloyd, 1986, p81).

2.3.1 Cold pressor pain

Pain is a subjective experience and can be a kind of unpleasant sensations due to thermal discomfort. Cold pressor pain (ice water immersion) might be increased rapidly within the first 20 to 40 seconds after immersion (Woolf and Hardy, 1941). During the course of repeated cold stimulation, the intensity of the subjective pain sensation changes very little (Zbrozyna and Westwood, 1990). There are a number of studies concerning perceived intensity and affect of cold pressor pain.

Cold stimulation pain may attenuate pain (Nielsen and Gotliebsen, 1992). Blitz and Dinnerstein (1968) found that the attenuation of pain could be achieved even when the source of pain was cold stimulation and even when this stimulation was from a contralateral stimulus. Similarly, Le Bars and coworkers (1979) and Willer and coworkers (1989) suggested that the pain inhibition correlates with the pain intensity of the counterirritation. Furthermore, Talbot and coworkers (1987) examined the perception of painful heat stimuli on the face before, during and after the subject submerged a hand in cold water (5°C) for 5 minutes (cold pressor pain). Their results showed that the subjects' ratings of the heat stimuli were significantly reduced by the cold pressor pain and the pain threshold was significantly increased from 45.7°C to 47.3°C while the hand was in cold water. More recently, Nielsen and Gotliebsen (1992) found that cold pressor pain decreased a laser-induced pain intensity. It was suggested that pain can be effectively inhibited by cold induced pain due to Ipsilateral Aδ- and C-fibre interaction and/or Ipsilateral large fibre pain inhibition. Wahren and coworkers (1989) also suggested that pain from cold stimulation may be suppressed by activation of cold sensitive receptors with large myelinated fibres.

It was also suggested that the nature of pain threshold does not vary between males and females, whereas pain tolerance is greater in males (Hall and Davies, 1991). Hall and Davies (1991) investigated gender differences between athletes and nonathletes on measures of perceived pain intensity and pain affect. The cold pressor pain was induced by cold water (1 to 2°C) immersion for maximum of 5 minutes. The subjects completed two visual analysis scales, one to rate the pain sensation intensity (Perception of the strength of the pain stimulus) and one to rate the perceive magnitude of the induced pain (How the pain feels.). The findings showed that the female nonathletic group showed significantly higher responses on pain affect than athletes, but they were not significantly different from the male nonathletes. Moreover, the male nonathletes were significantly higher than male athletes on affect of pain. Both nonathletic groups perceived affect of pain greater than intensity of pain. In contrast, the athletes perceived intensity of pain greater than perceived affect. The authors suggested that pain tolerance may be socially learned, depending upon the type and frequency of pain experiences the individual has encountered. Moreover, Goolkasian (1985) suggested that women had lower pain thresholds due to menstrual cycle effects. Hapidou and coworkers (1988) also found that dysmenorrheic women reported less pain than did non-dysmenorrheic women.

Therefore, cold stimulation could be used for the relief of pain or the cold pressor pain itself. A stimulus which is painful in one situation may not be in another and thermal pain tolerance varies among people.

2.3.2 Blood flow

Although the intensity of subjective sensation of pain is not reflected to the changes in the local vascular response (Westwood et al. 1989), the local application of cold may cause a reduction in muscle blood flow when muscle temperature and rectal temperature are normal or low, but if the muscle temperature is elevated, muscle blood flow is not reduced during local cooling (Pendergast, 1988). Moreover, cold stimulation induces an increase in vascular resistance, causing a reduction in blood flow to the skin (Horvath, 1981). However, during long immersions of the hand, the capillary beds initially constrict and then periodically dilate for brief periods (Lovallo, 1975a). The cold-induced vasodilation (CIVD) is due to an increased blood flow resulting from the sudden opening of the arteriovenous anastomoses (LeBlanc, 1975b). Also, the CIVD indicates a periodic weakening of sympathetic tone (Lovallo, 1975a). By measuring and evaluating pulse-wave amplitude in the finger, Lovallo and Zeiner (1975b) investigated the effect of initial level of vasomotor tonus on the cutaneous blood-volume response to the foot cold pressor test. In order to establish the different level of sympathetic tonus to the cutaneous blood vessels, subjects rested for 20 minutes with temperature set at either 32° C, 22° C, or 12° C. Then, the subjects placed his left foot into the ankle-deep ice water for 1 minute. The results showed that when the blood vessels of the skin of the hand are dilated prior to CPT (resting in 22 or 32° C air temperature), the stress response is vasoconstriction. When the vascular state prior to CPT is vasoconstriction (resting in 12° C air temperature), the response is either no change or vasodilation to the stressor. It was suggested that the magnitude and direction of change in cutaneous vascular systems are dependent on their prestimulus tonus. In a study about CIVD response during CPT, Livingstone (1976) found that after an exposure to Arctic conditions for 2 weeks, there was an increased vasoconstrictor tone when the subjects placed their finger in ice water for 30 minutes. More recently, Zbrozyna and Westwood (1990) investigated the effect of vascular changes in the calf and forearm during repeated immersions of foot in water at 4° C. The results indicated that during the CPT, the vasodilatation that was elicited in the forearm and calf was habituated on repeated stimulation. The authors suggested that selective inhibition of vasodilatation in the calf or forearm is controlled through the spinal level because the sympathetic outflow to the extremity vascular beds is located at different segmental spinal levels. Leftheriotis and coworkers (1990) found that during cold exposure maximal metabolic vasodilatation is decreased after local cold-acclimation, and that the increase in thermal capabilities after cold-acclimation could be the results of changes in skin and/or underlying tissue blood flow. Similarly, Savourey and coworkers (1992) demonstrated that repeated 5°C cold water immersion of the right hand and forearm developed a local cold adaptation of the extremities. They suggested that the reason for the warmer temperatures of the extremities after adaptation would be an increased peripheral cutaneous blood flow, but not increased local heat production in muscle.

Therefore, the degree of vasoconstriction or vasodilatation during local cooling depends on the initial level of vasomotor tone. Local cold acclimation increases CIVD response to CPT.

2.3.3 Heart rate and pressor response

The CPT has long been used to evaluate the response of the cardiovascular system to stress (Lovallo and Zeiner, 1975b). The CPT produces increases in HR and BP. The cardiovascular response to cold pressor stress reaches its peak at the end of 60s of immersion or during the following 10-20s (Zbrozyna and Krebbel, 1985). Godden and coworkers (1955) found SBP response reached maximum level between 40 and 70 seconds of a 2-minute immersion. Frey and Kenney (1979) reported with immersion in water at 10°C, arterial pressures were approximately the same at 2 and 3 minute. Frey and coworkers (1980) further indicated that arterial pressure was rising monotonically from 30-60s during a one-minute CPT and by contrast, HR response during CPT showed a return toward control levels after an initinal increase and the decrease in HR in foot immersion was greater than in hand immersion due to greater sympathetic activation with the hand immersion. The authors suggested that this decrease in HR response is due to partly the results of a baroreceptor reflex stimulated by the increased arterial pressure during CPT.

Increase in BP and HR during local cooling result from an increase in plasma catecholamine. LeBlanc and coworkers (1979) found that BP increased during a 2-min CPT. The increased BP returned to its initial level within about one minute after the test. HR changes were preceded by an increase in plasma epinephrine whereas norepinephrine changes were seen after the effect on BP had taken place. The authors suggested that the increased BP is due primarily to the vasoconstriction caused by norepinephrine and the increase in HR due to epinephrine. Fagius and coworkers (1989) demonstrated that MSNA initially increased and then decreased during a 1-min CPT. The BP elevation correlated linearly with an increase in total outflow of sympathetic activity. The relationship was stronger for SBP than for DBP although individual variations were observed. The authors suggested that the increase in MSNA is responsible for the increase in BP and with longer duration of immersion the pressor response is attenuated. Similarly, Seals (1990) reported that during a 1.5-min CPT, SBP increased progressively with time; HR increased slightly and remained at this level until the end of the test. MSNA increased much greater during two hand immersion than in one hand immersion. However, the magnitude in SBP and HR response was no different between one and two hands immersion. It was indicated that increase in MSNA, but not SBP response, is influenced by the size of the tissue area exposed to the stimulus. This is because the changes in cardiac output are not similarly augmented due to vasoconstrictor tone in vascular beds other than muscle.

Therefore, the pressor response to CPT is caused by an increase in SNA. The size of the area over which the cold stimulus is applied may influence the magnitude of MSNA but not SBP and HR responses. Pressor response to CPT is time dependent.

2.3.4 Adaptation

Cutaneous receptors are linked to a temperature regulating centre located in the hypothalamus (LeBlanc, 1975b). Cold stimulation of the cutaneous receptors may induce cutaneous vasoconstriction to increase insulative level in order to maintain core temperature. This is an important defence reaction which is controlled by the hypothalamus. The cutaneous vasoconstriction response to CPT can be adapted in humans. Local cold adaptation is characterized by warmer local skin temperatures, less pain, and an earlier CIVD at higher skin temperature (Leblanc, 1975a, p195).

LeBlanc (1962) reported that the reduction of finger temperature during hand immersion in ice-water was less in Gaspe fishermen than in warm-acclimated subjects. On the other hand, Paik and coworkers (1972) suggested that the degree of vasoconstriction of finger blood vessels in response to immersion of a hand in water at 6°C is significantly greater in divers than in warm-acclimated subjects. This finding was different than the LeBlanc's studies of fishermen (LeBlanc, 1962). The reduction of finger temperature during hand immersion was less in Gaspe fishermen than in the diver, because the type of adaptation to local exposure to cold is basically different from that of whole-body exposure (Paik et al., 1972). LeBlanc and coworkers (1975b) studied the autonomic nervous system and adaptation to hand and face immersion into cold water in men. The data showed that BP and HR responses to the hand cold pressor test were weaker in the Eskimos than in white subjects, but the bradycardia which characterizes the cold face test in Eskimos was not different than in white subjects. The mailmen who worked outdoor for an average of 6 hours a day in cold weather, showed a reduction of the blood pressure response to cold hand test at the end of the winter. It was suggested that adaptation to cold is characterized by a decreased sympathetic response and an increased vagal activation. Van Someren and coworkers (1982) suggested that cold-acclimatized people tend to vasoconstrict and shiver less in the cold and therefore they increase their metabolic rate less than warm-acclimatized people. The reason for this includes the establishment of conditioned reflexes. Leftheriotis and coworkers (1990) examined the finger and forearm vasodilatatory changes after local cold acclimation. The subjects were cold-acclimated by daily immersion of the right hand and forearm in a 5°C water bath for 20 minutes on 30-successive days. At the end of a 5-min CPT, the decrease in finger and forearm skin temperature after 30 days was significantly less than before. During recovery of the CPT, the decrease in finger and forearm skin temperature was significantly greater in the non-acclimated subjects than in cold-acclimated subjects. It was suggested that during cold exposure, maximal metabolic vasodilatation is decreased after local cold-acclimation. Westwood and coworkers (1989) also indicated that repeated exposure of one foot to ice-water could lead to habituation of the vasodilatation in calf and forearm and the changes in the vascular response in the calf and forearm to the stimulus were independent and were not reflected in the intensity of subjective sensation of pain.

2.3.5 Other factors

Several factors may influence the cardiovascular response to local cooling. LeBlanc and coworkers (1978) examined the effect of cold hand and face test on the cardiovascular response of subjects aged 53-60 and 20-47 years. The findings indicated that heart rate decreased with aging but that blood pressure response was not significantly different between the two groups. It was suggested that dissociation of BP and HR responses may be due to decreased sensitivity of beta-receptor with increasing age.

Sexual dimorphism in body structure implies gender-specific physiological response to body cooling (Graham, 1988). LeBlanc and coworkers (1978) reported that at the end of a 2-min cold hand pressor test, the change in BP was significantly lower in female subjects, and in 2 minutes after the test, HR decreased lower than initial values in female subjects. The authors concluded that in women, cardiovascular function is depressed during the period following the cold hand or cold wind test. Frey and coworkers (1980) found that female subjects showed a greater increase in HR than male in cold hand or foot test (4° C water, for 1 minute). Although there were no significant difference between men and women in resting BP, SBP during seated hand immersion and both SBP and DBP during supine foot immersion were higher in male subjects.

To summarize, CPT induces an increase in SNA, and as a result, increases in HR, SBP and DBP, and decreases of blood flow in the extremities. There are gender differences in cardiovascular responses to the CPT due to the differences in morphology and physiology, and this is also associated with age. Cardiovascular responses to local cooling can be habituated.

2.4 Physiological response to combined isometric and cold stress

Frisk-Holmberg and coworkers (1983) examined the relationships of two cardiovascular stresses of different origin: isometric exercise and CPT. The isometric stress was produced by HG at 33% MVC for 2 minutes; the CPT was induced by left hand immersion in ice-water for 2 minutes. The results indicated that BP response in isometric exercise and CPT is similar. However, the mechanism eliciting the pressor response is different. Yet, only a few studies have evaluated the effects of isometric exercise combined with the cold pressor stress on cardiovascular function.

2.4.1 Temperature and blood flow

Humphreys and coworkers (1963) examined the relationship between blood flow and different intensities of isometric exercise. Following forearm immersion in water at 18, 26, 34 or 42°C for 30 minutes, the trained subjects performed HG at various intensities of MVC until fatigue. The results showed that blood flow through active muscle increased during the exercise at a given temperature of water immersion. It was suggested that the change of temperature in active muscle can be due to an increased blood flow. Also, Leftheriotis and coworkers (1990) found that during HG at tension of 10% MVC with the cooled arm, increase in forearm blood flow is predominantly due to increased muscle blood flow. Petrofsky and coworkers (1981b) found that following 30 minutes of forearm immersion in a water bath at 3 to 40°C, the deep muscle temperature of overweight subjects remained closer to core temperature than did that of thin subjects. Therefore, in general, HG at lower intensity performed in water at different temperature induces an increase in blood flow through the active forearm muscles.

2.4.2 Blood pressure and heart rate

Coppin and coworkers (1978) examined the effects of the forearm immersion in 10°C water bath for 30 minutes on HG strength. The findings showed that DBP and SBP increased initially during the immersion and returned to normal while the arm remained in the bath. Petrofsky and coworkers (1981b) investigated the influence of deep muscle temperature on the cardiovascular responses to HG in eight male subjects who were trained for 4-6 weeks. The water bath temperature were 3, 10, 15, 17.5, 22, 25, 30, and 40° C. After the 30 minutes of each water immersion, while the subject's arm was still immersed, the subject performed a fatiguing HG at a tension of 40% MVC. The findings indicated that the increase in BP during the contractions was almost constant for contractions exerted in water between 22° C to 40° C, but there was an increase and then a reduction in the magnitude of both the SBP and DBP for contractions exerted in the two coldest water (3° C and 10° C). Frisk-Holmberg and coworkers (1983) examined the relationship during cardiovascular stress originating from different receptors (isometric exercise and CPT). The results indicated that the blood pressure responses to CPT and isometric exercise were similar. The authors indicate that the cardiovascular response to the CPT involved neither the same receptors nor the same afferent neural pathways as the skeletal cardiovascular reflex. The MSNA is responsible for the increased level of BP not only in response to isometric muscle contraction (Mark et al. 1985) but also to the CPT (Fagius et al. 1989). More recently, Peikert and Smolander (1991) investigated the combined effect of CPT and isometric exercise on HR and BP. The SBP, DBP, HR, and pressure rate product increased higher with the CPT in combination with isometric exercise than that of the isometric exercise alone. The authors suggested that vasoconstriction is responsible for the elevation of BP during the CPT.

Therefore, the pressor response to isometric exercise during local cooling is influenced by the temperature of water. MSNA is involved in the cardiovascular response to both isometric exercise and CPT.

2.4.3 Isometric strength and endurance

There is a neural and mechanical influence of temperature on muscular contractions (Barnes and Larson 1985). According to the traditional concept, temperature increases in mechano-elastic systems result in lower internal viscosity and less resistance to changes in length. Temperature decreases produce just the opposite effect. However, there are conflicting findings about the effects of local cooling on isometric strength and endurance.

Edwards and coworkers (1972) found during isometric knee extension at a tension of 67% MVC in a series of seven contractions at intervals of 20 seconds, both pre-heating and pre-cooling of the quadriceps muscle resulted in a shortening of the endurance time for a single contraction following immersion of the test leg in water at 12, 26 or 44° C for 45 minutes. It was suggested that the fatigue may be due to depletion of local energy resources in muscle which is caused by reduction in the rate of regeneration of ATP from anaerobic glycolysis below that needed to maintain the contraction force. Barnes and Larson (1985) examined the effect of cooling and warming the arm with water during maximal handgrip contraction. The experiment involved maximal handgrip contractions at every 2 minute for 30 minutes during immersion in water at 40° C or 10° C. The results showed that in cold water, maximal strength increased significantly at the onset, but after 6 minute of exposure to the cold water, strength decreased progressively. The authors suggested that the strength reduction may be due to reflex vasoconstriction within the muscle, and stimulation of cutaneous receptors may provide excitatory and/or inhibitory input to the discharging alpha-motoneuron through an undetermined synaptic connection. In the experiment of forearm immersion at 10° C to 15° C water for 30 minutes, Johnson and Leider (1977) found that the 80-min post-treatment handgrip strength was significantly greater than any previous measure and also greater than any measure recorded during the control session. This increased grip strength remained for a 3-hour recovery period. It was suggested that an increased arterial blood flow might cause the rise in strength.

In contrast, Coppin and coworkers (1978) argued that the vascular response in deep muscle may not have the same degree of vasodilation than in the cutaneous vessels. Their experiment showed that grip strength significantly decreased as a consequence of immersion of the forearm at 10° C water for 30 minutes. The strength recovery to normal values took place within 40 minutes and no increases in post-immersion strength were observed. Bergh and Ekblm (1979) found that maximal dynamic strength was positively related to muscle temperature, but maximal isometric strength is affected little by changes in muscle temperature. Similarly, Petrofsky and coworkers (1981b) demonstrated that in trained subjects, the MVC was reduced markedly and progressively after forearm immersion in water bath below 22° C for 30 minutes.

Similarly, Bundschuh and Clarke (1982) suggested that maintaining lowered local environmental temperature does not affect strength and recovery, but enhances muscular endurance. Savourey and coworkers (1992) found that there was no significant difference in maximal handgrip strength before and after cold-acclimation (arm immersion in water at 5° C for 5 minutes, 5 days a week for 2 months). In his review of 1991, Doubt (1991) concluded that cooling of skeletal muscle will reduce contractile force and increase the rate of fatigue.

Therefore, during local cold water immersion, isometric strength increases initially and then decreases. This may be due to reflex vascular changes. Moreover, cold stress can produce a more marked cardiovascular response when combined with an isometric exercise (Peikert and Slomlander, 1991).

2.5 Summary

In summary, isometric contraction induces an increase in HR, SBP and DBP due to central command, reflex adaptation, and autonomic nervous activity. The initial increase in HR during isometric exercise is due to vagal withdrawal, whereas the later sympathetic effector response causes a further increase in HR. During a CPT, an increase in BP is due to an increase in SNA. However, although the pressor response to an isometric exercise (33% MVC) and the CPT is said to be similar, the mechanisms causing the pressor response are different.

CHAPTER III

METHODOLOGY

3.1 Introduction

The purposes of this study were to (1) compare the pressor response between isometric exercise and a CPT and (2) examine the pressor response to isometric exercise (33% MVC) combined with a CPT applied either at the onset or during the last minute of a 2-min CPT.

3.2 Subjects

Ten male subjects between the ages of 20 and 35 years were recruited for the study. The following criteria for the selections was applied: non-smoker, non-cold-acclimated, normotensive (<140/90 mm Hg), and not take medication that could alter their cardiovascular response to the testing. All subjects read and signed an informed consent form approved by the University of Ottawa Human Research Ethics Committee.

3.3 Testing protocol

The testing protocol was consist of a preliminary and three experimental sessions.

3.3.1 Preliminary session

At a preliminary testing session, subjects were thoroughly informed of the nature of the study and their involvement. They were required to complete a Modified Physical Activity Readiness Questionnaire (PAR-Q) (Jetté et al., 1992). If subjects answered yes to any of the question on the PAR-Q, they were not allowed to participate in the study.

After a 5-min sitting rest, resting HR and BP values were measured by auscultation to ensure that the subject was normotensive at rest. Height and weight were then measured.

MVC of the dominant hand were determined by squeezing a rubber bulb connected to an air pressure gauge (H. O. Trerice Co., Detroit, Michigan) by plastic tubing. The average of three maximal isometric handgrip contractions maintained for 3 seconds was used as MVC. There was a 5-min rest between each contraction. Subjects were instructed to keep normal breathing during the isometric exercise so as not to perform a Valsalva manoeuvre. During these contractions the subject were seated with his elbow bent at an angle of 90°.

Subjects were initiated to the Finapres 2300 BP Monitor cuff (Ohmeda Louisville, Co.). They would then be instructed on holding a 33% MVC HG.

Subjects would then be introduced to the CPT. The CPT would be administered using a container filled with ice water (2 to 4° C). The subject's right foot would be immersed into the water up to the ankle during a CPT. Subjects would be required to practice HG both at the onset and during the foot immersion.

3.3.2 Experimental session

Prior to the experimental sessions, subjects was advised not to participate in any form of physical activity or to drink caffeine-containing beverages prior to testing. Upon arrival in the laboratory (22-24° C), the subject was seated for 20 minutes prior to the beginning of testing. The Finapres cuff was then applied on the middle finger of the non-dominant hand. All tests was performed in the sitting position. Resting heart rate and blood pressure was then recorded for a period of 1 minute at every 5 second intervals prior to each test. The HR, SBP, DBP, and MAP were recorded every 5 seconds during actual testing as well as for a period of 4 minutes following the test.

The three experimental tests were presented in a random manner over three days. All tests were conducted at the same time of day.

<u>Experimental session one</u>: Following a 20-min rest, the subject performed the HG at 33%

MVC for 2 minutes. After a 15-min rest, subject was required to place his right foot in ice water up to the ankle for a period of two minutes.

Experimental session two: Following a 20-min rest, subjects performed HG at 33% MVC combined with simultaneous CPT for 2 minutes.

Experimental session three: Following a 20-minute rest, subjects performed the CPT for 2 minutes during which the HG at 33% MVC was administered during the last one minute.

3.4 Statistical analysis

Independent variables in this study included one intensity of HG (33% MVC), a two-minute cold foot immersion, and three different testing conditions for HG (HG without the CPT, HG combined with the CPT, and HG performed during the last minute of the CPT). Dependent variables, SBP, DBP, MAP and HR, were represented as the mean difference between the pre-test value and response value (ΔSBP, ΔDBP, ΔMAP, and ΔHR).

Data was reported as mean values and standard deviation. Pre-exercise resting values were the average of 15s measurements prior to the experimental tests.

A student paired t-test was used to determine whether significant difference existed between CPT and HG. A two-way repeated measures ANOVA with trend analysis design was used to examine the difference between the CPT and HG as well as post-CPT and post-HG on dependent variables.

A two-way repeated measures ANOVA with trend analysis design was also used to determine if there was any significant difference among different testing conditions on cardiovascular responses to HG.

Data were analyzed on a mainframe computer using SPSSX with one between-subjects factors and one within-time factor. If an overall difference was found concerning on the cardiovascular responses with respect to time course and testing conditions, Tukey post hoc procedure was administered to localize significant difference between means.

P values smaller than 0.05 were considered to be statistically significant.

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APPENDIX A

MODIFIED PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)

MODIFIED PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)[1]

Family Name: ____________________ Date: ________________________
Given Name: ____________________ Age: ________________________

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

If yes, please specify:

Reason: ______________________________
Name: ______________________________
Dosage: ______________________________

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

If yes, please specify:

Reason: ______________________________
Name: ______________________________
Dosage: ______________________________

Date: ________________________
Signature: ____________________

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[1] Jetté, M., Quenneville, & J., Sidney, K. (1992) Fitness testing and Counselling in Health Promotion. <u>Canadian Journal of Sports Sciences</u>, 17: 194-198.

APPENDIX B

CONSENT FORM FOR HANDGRIP AND COLD PRESSOR TEST

CONSENT FORM FOR HANDGRIP AND COLD PRESSOR TEST

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CHAIR OF FHS-HREC: Dr. Marie-des-Anges Loyer
Faculty of Health Sciences
Human Research Ethics Committee
Room 2009, 451 Smyth Road
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Date: __________ Name of volunteer: _______________

The title of this study is the "Pressor response to isometric handgrip combined with foot immersion in cold water". The purpose of this study is to compare the blood pressure response to handgrip at a tension of 33 % of maximal voluntary contraction combined with a cold foot immersion applied either at the onset or following immersion.

I understand that I will be asked to participate in a preliminary and three experimental sessions lasting from 30 minute to one hour. The study involves performing a handgrip test at 33% of maximal voluntary contraction, and immersion of the right foot in cold water (2° C-4° C), singly and combined. I may experience some discomfort such as a prickling or a tingling during immersion of the right foot in cold water. There are no known or reported risks associated with these tests as utilized in this study. It is understood that I can withdraw my foot from the water bath at any time I wish.

The confidentiality of my data will be maintained at all times. Only the principal investigator 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.

<u>I AM FREE TO WITHDRAW</u> this consent and to discontinue my participation at any time without penalty or discrimination.

Signature of volunteer and date: ______________________

Signature of witness: ______________________________

APPENDIX C

LETTER OF INFORMATION

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This project is submitted in partial fulfilment of the degree of master of science in movement studies.

The purpose of the study is to compare, in normotensive persons, the blood pressure response to handgrip at a tension of 33% of maximal voluntary contraction (MVC) combined with immersion of the right foot in cold water, applied either at the onset or following immersion (2 to 4°C).

The study consists of a preliminary and three experimental sessions conducted over four separate days. At the preliminary session, you will be briefed in detail as to the nature of the study and of your involvement. You will then be asked to complete the Physical Activity Readiness Questionnaire (PAR-Q). Your resting blood pressure will be measured to ensure that you are normotensive at rest (<140/90 mm Hg) so that you may be included in the study. Your resting heart rate, height and weight will be then measured. You will perform three maximal handgrip contractions by squeezing a rubber bulb as strongly as possible using your dominant hand for a 3-second duration. The duration of this preliminary session will be approximately one hour.

Prior to the experimental sessions, you will be asked not to participate in any form of physical activity or to drink caffeine-containing beverages for 3 hours prior to testing. At the first experimental session, following a 20-minute rest, you will perform a handgrip contraction at a tension of 33% of maximal voluntary contraction combined with simultaneous immersion of the right foot in cold water for two minutes. Your blood pressure and heart rate will be measured continuously prior to, during, and after the test using a Finapres Blood Pressure Monitor. The duration of this session will be approximately of 30 minutes.

At the second experimental session, following a 20-minute rest, you will be administered the cold foot immersion for two minutes during which you will perform, during the last minute, a handgrip at a tension of 33% of maximal voluntary contraction. Your blood pressure and heart rate will be measured continuously prior to, during, and after the test using a Finapres Blood Pressure monitor. The total duration of this session will be approximately 30 minutes.

At the third testing session, following a 20-minute rest, you will perform a handgrip at a tension of 33% maximal voluntary contraction for two minutes. Following a 15-minute rest, you will be administered the cold foot immersion for two minutes. Your blood pressure and heart rate will be measured continuously prior to, during, and after each test using the Finapres Blood Pressure Monitor. The total duration of this session will be 50 minutes.

Every effort will be made to conduct the tests in such a way as to minimize discomfort and risk. You may experience some discomfort such as a prickling or a tingling sensation during the foot immersion test. It is agreed that you may withdraw from the test whenever you feel the procedure to be uncomfortable.

This study is designed to enhance our knowledge with respect to the mechanism(s) of the blood pressure response to the combined effects of isometric handgrip and a cold pressor test.

Your privacy and anonymity will be protected in the following manner: All data sheets collected on you will not identity you by name but a coded number. All data file will be destroyed upon completion of the study. Only myself and my advisor will have access to data.

I understand that I can withdraw/refuse to participate in the study without fear of reprisals.

APPENDIX D

PRE-TESTING AND TESTING SHEETS

Isometric exercise and cold pressor test

(Pre-testing Sheet)

Code Number of subject:____________________

Date: ____________________________

Age: ______________ Sex: ______________

Weight: ____________ (KG) Height: ____________ (CM)

Smoker: Yes ___ No ___ Ex smoker: Yes ___ No ___

Cold-acclimated: Yes ___ No ___

Taken medication: Yes ___ No ___

If yes, please give the detail: ________________

____

____

Statement about participation in physical activities:

1. No regular participation in any form of exercise
2. Occasional exercise (2 or 3 times per month)
3. Regular exercise (once or twice a week)

Resting HR: ____________ (beat/min) Resting BP: ____________ (mmHg)

MVC: 1. ________ (kPa) 2. __________ 3. __________

Mean: __________ 33% MVC: __________

Investigator:________________

Isometric exercise and cold pressor test

(Testing Sheet)

Date: ____________________ Testing time: ________________

Testing session: _____________ Room Temperature: ______________

Water Temperature: __________ Code Number: ________________

Drinking any coffin-container beverage 3 hours prior to the test: Yes _____ No _______

If yes, indicate: Name _________________________

Amount _________________________

When _________________________

Participate any form exercise 3 hours prior to the test: Yes _____ No _______

If yes, described in details: ______________________

____

____

Resting HR: _________________________ (beat/min)

Resting BP: _________________________ (mmHg)

MVC: ___________________ (kPa)

33% MVC: _________________

Comment: __________________________________

____

____

Investigator ___________________