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AGE DIFFERENCES ON THE PATTERN OF BLOOD PRESSURE RESPONSE TO ISOMETRIC EXERCISE IN NORMOTENSIVE MALES

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AGE DIFFERENCES ON THE PATTERN OF BLOOD PRESSURE RESPONSE TO ISOMETRIC EXERCISE IN NORMOTENSIVE MALES

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A thesis submitted to the school of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Science

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ABSTRACT

The purpose of this study was to compare the pattern of blood pressure (pressor) response to isometric handgrip contractions between older and younger males with and without blood occlusion. Ten older males (mean age 54.7 $\pm$ 3.0 yrs) and ten younger males (mean age 26.6 $\pm$ 1.9 yrs) normotensive at rest and dynamic exercise participated in this study. Following the determination of their maximum voluntary contraction (MVC), subjects performed handgrip contractions (dominant) at 20%, 40%, and 60% MVC (for 30 seconds), and at MVC (for 12 seconds), with and without blood occlusion. Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and heart rate (HR) responses were recorded at every 5 seconds for the 20%, 40%, and 60% MVC, and at every 2 seconds during the MVC test using a Finapres BP monitor. Results indicated that without blood occlusion the mean absolute SBP, DBP, and MAP to isometric handgrip contractions were significantly higher in the older males than in the younger males for all contraction intensities although the mean absolute HR at all intensities was significantly lower in the older males. With blood occlusion, the mean absolute SBP was also significantly higher for all contraction intensities in the older males; mean absolute DBP was significantly higher in the older males only at MVC and mean absolute MAP was significantly higher in the older males except at 60% MVC. Mean absolute HR was again significantly lower in the older males during blood occlusion. However, there were no significant differences with and without blood occlusion between the two age groups in the slopes of SBP, DBP, MAP, and HR responses to the isometric contractions except for DBP at 60% MVC with blood occlusion, which was higher in the younger males.

Within the younger age group, the mean absolute SBP was significantly greater with

blood occlusion than without blood occlusion at 20% and 40% MVC, but similar at 60% MVC and at MVC; mean absolute DBP and MAP were significantly higher with blood occlusion than without blood occlusion at 20%, 40%, and 60% MVC but not at MVC. Mean absolute HR was similar between blood occlusion and no blood occlusion for all contraction intensities. Within the older age group, there were no significant differences between with and without blood occlusion for mean absolute SBP, DBP, MAP, and HR responses to isometric handgrip contractions.

It is concluded that although absolute pressor response to isometric handgrip contractions is higher in older males, the pattern of pressor response to isometric handgrip contractions is similar between the two age groups. Blood occlusion during isometric handgrip contractions did not influence the pressor response of older males. However, blood occlusion increased in the pressor response of younger males at lower percentages of MVC.

INTRODUCTION

The normal human aging process is accompanied by progressive physiological changes. Which contribute to an increase in blood pressure, both at rest and during exercise (Lipsitz, 1989; Jetté et al., 1991). These include an increase in left ventricular mass (Jetté et al., 1991), a decrease in the compliance of the blood vessels (Lakatta et al., 1987), a reduction in the baroreflex-mediated heart rate response to alterations in blood pressure (Shimada et al., 1985), and a decline in the beta-adrenergic receptors mediated vasodilation (Hiremath et al., 1989).

Various investigations of the age differences on the pressor response to isometric exercise have provided conflicting results. Some investigators concluded that the pressor response to isometric exercise is significantly greater in the older males than in the younger males (Petrofsky and Lind, 1975; Van Loan et al., 1989). Other investigators have reported that the pressor response to isometric exercise is similar for both age groups (McDermott et al., 1974; Sagiv et al., 1988). Goldstraw and Warren (1985) showed that when resting blood pressure was taken into consideration, the pattern of pressor response to isometric exercise was similar in the two age groups and that the resting blood pressure differential between the older and younger males was maintained during isometric exercise. They concluded that age does not influence the pattern of pressor response to isometric exercise. However, in their study, blood pressure was recorded by a sphygmomanometer thus restricting the number of measurements to adequately assess the pattern of pressor response to isometric exercise. Although some studies have investigated the effect of age on the pressor response to isometric exercise, only a few have reported on the pressor response to isometric exercise using continuous BP measurements (Saltin et al., 1981).

Donald et al. (1967) and Coote et al. (1971) showed that the pressor response to

isometric exercise was increase when arm blood flow was occluded in young subjects. They concluded that the higher pressor response during and following forearm exercise during blood occlusion was due to the accumulation of metabolites within the contracting muscles. It appears that there are no data with regard to age differences on the pressor response to isometric exercise with blood occlusion. Therefore, the purpose of this study was to compare the response of older and younger males for the pattern of pressor response to isometric handgrip contractions measured continuously both with and without upper arm blood occlusion.

METHODOLOGY

Subjects

Ten healthy males between the ages of 20 to 29 yrs (mean age 26.6 $\pm$ 1.9 yrs) and ten between the ages of 50 to 59 yrs (mean age 54.7 $\pm$ 3.0 yrs) participated in the study. The subject was selected on the basis of the following criteria: did not suffer from a physical ailment which could affect his performance, was non-smokers, had a normal resting blood pressure (<140/90 mmHg), had a normal blood pressure response to a moderate level of dynamic exercise (stage A of Canadian Aerobic Fitness Test, 1987), provided negative answers to all questions on the modified PAR-Q (Jetté, Quenneville and Sidney, 1992), did not take any medication that could alter his cardiovascular response to isometric exercise. The subject was requested to read and sign an informed consent form prior to participation in the study. The study received the approval of the Human Research Ethics Committee of the University of Ottawa.

Materials

Resting blood pressure and heart rate were measured using a standard sphygmomanometer and stethoscope. The modified Canadian Aerobic Fitness Test (CAFT) (Jetté, Landry and Sidney, 1991) was employed to assess BP response to dynamic exercise of the subjects using a standard sphygmomanometer and stethoscope. Handgrip contraction was performed using an air pressure gauge (H.O. Trerice Co., Detroit, Michigan) connected by surgical tubing to a rubber squeeze bulb and recorded in kiloPascals. A Finapres BP monitor 2300 (Ohmeda; Louisville, CO) connected to an IBM computer (IBM 5151, IBM Co, Armonk, New York) and printer (IBM 5152, IBM Co, Armonk, New York) were used to measure continuously BP and HR during isometric handgrip contractions.

Procedure

The testing was conducted on two separate days. On Day 1, the subject was thoroughly briefed as to the nature of the testing and his involvement in the study. The subject was then requested to complete the modified PAR-Q and to read and sign the informed consent form. The subject's height, weight, and skinfolds were measured (Jetté, 1983). From these measurements body mass index (BMI) was calculated. The subject was then seated for at least 5 minutes and the resting BP was determined in duplicate (by auscultation). If the resting BP met the selection criterion (SBP<140, DBP<90), the subject then performed the modified Canadian Aerobic Fitness Test in order to screen out exercise hypertensive subjects (ie. an increase in SBP >45 mmHg from rest to stage A of the CAFT) (Jetté et al., 1991). The results of the last CAFT stage completed by the subject were used to calculate predicted VO_2max (Canadian Standardized Test of Fitness, 3rd ed, 1987). If the subject showed a normal BP response to the CAFT, he then performed, after a 10 minute rest, three maximal voluntary handgrip contractions with a one minute rest between each contraction. The mean of the best two trials was used to establish handgrip MVC.

For Day 2 of the experiment, the subject was requested not to participate in physical exercise and to consume neither caffeine nor alcohol prior to the testing. The testing session was conducted between 08:00 and 12:00 hours, at least forty-eight hours following the preliminary testing session. When the subject arrived, he was seated for ten minutes. Resting and exercise SBP, DBP, MAP, and HR were derived from the Finapres BP Monitor. Handgrip contractions were performed at different % MVC levels using the Trerice pressure gauge connected by surgical tubing to a rubber squeeze bulb. During the handgrip contractions, the subject was instructed to avoid the Valsalva manoeuvre as much as possible. All handgrip contractions were performed in the sitting position, arm extended at the side using a dominant hand. The experimental protocol was conducted as follows:

1): The subject performed handgrip contractions (dominant hand) at 20%, 40%, and 60% MVC for 30 seconds and at MVC for 12 seconds. The exercise BP and HR were recorded at every 5 seconds for the 20%, 40%, and 60% MVC and at every 2 seconds for MVC. The order of the % MVC levels was randomly presented for each subject. Each test was performed with a 5-minute rest period between each contraction.

2): After a 10-minute rest, the identical intensities were repeated after inflating to 200 mmHg a sphygmomanometer cuff placed around the upper contracting arm. A 10-minute rest period was given between each contraction.

Data Analysis

All data were analyzed on the University of Ottawa IBM computer using the SAS program (SAS Institute Inc., 1985).

T tests (compare older and younger males) were computed on mean anthropometric, resting BP and HR, and physical data collected on Day 1.

The Finapres BP and HR data of Day 2 test were collected by IBM computer and transferred to the University of Ottawa Conversational Monitor System (CMS) for analysis.

The independent variables included the two age groups, the with and without blood occlusion contractions at 20%, 40%, and 60% MVC, and at MVC. The dependent variables included SBP, DBP, MAP, and HR, at rest, and during isometric handgrip contractions. The mean values for the exercise cardiovascular responses (including 7 measurements from time 0 to the end of contractions) were analyzed by a 2x2x4 factorial design ANOVA. When a significant difference was found, Scheffé's procedure was utilized to determine between which means the difference existed. Results at the 0.05 level of probability were accepted as statistically significant.

Linear regression analysis was calculated using all the data points from time 0 to the end of contractions to determine the individual slope and intercept for BP and HR

responses over time for each percentage of MVC. The exercise slope and intercept were also entered in a 2x2x4 factorial design ANOVA.

RESULTS

The mean age and physical characteristics of the subjects are shown in Table 1. The mean age of the younger subjects was 26.6 $\pm$ 1.9 yrs, while the mean age of the older subjects was 54.7 $\pm$ 3.0 yrs. Although there were no significant differences in mean resting DBP or HR, the mean resting SBP of the older subjects was significantly higher than that of the younger subjects (126.0 $\pm$ 9.8 vs 118.0 $\pm$ 5.6 mmHg). However, none of the subjects (younger or older males) demonstrated resting or dynamic exercise hypertension. In addition to age, the older subjects had a significantly lower predicted VO_2max.

The mean absolute SBP, DBP, MAP, and HR at rest (60 seconds) and during isometric contractions at 20%, 40%, and 60% MVC (30 seconds) and at MVC (12 seconds) are shown in Table 2. The results showed that without upper arm blood occlusion mean absolute SBP, DBP, and MAP to all contraction intensities were significantly higher ($p<0.05$) in the older subjects, while mean absolute HR response was significantly lower ($p<0.05$). With blood occlusion, mean absolute SBP response was also significantly higher ($p<0.05$) in the older than in the younger males for all contraction intensities; mean absolute DBP response for the two groups was similar at 20%, 40%, and 60% MVC but significantly higher ($p<0.05$) in the older males at MVC; mean absolute MAP response was significantly higher ($p<0.05$) for all contraction intensities in the older males, except at 60% MVC; mean absolute HR response was significantly lower ($p<0.05$) in the older males during occlusion for all contraction intensities.

With respect to the younger subjects, mean absolute SBP response was significantly greater ($p<0.05$) with occlusion than without occlusion at 20% and 40% MVC but was similar at 60% MVC and at MVC; mean absolute DBP and MAP

responses were also significantly higher ($p<0.05$) with occlusion than without occlusion at 20%, 40%, and 60% MVC but similar at MVC; mean absolute HR response was similar with and without occlusion for all contractions. For the older age group, there were no significant differences in mean absolute SBP, DBP, MAP, and HR responses for all contractions with or without occlusion.

The mean regression values with and without occlusion for SBP, DBP, MAP, and HR responses to the isometric contractions are shown in Tables 3 and 4 and are graphically presented in Figures 1 to 4. There was an increase in pressor response and heart rate throughout the duration of contractions and particularly at the higher intensities. This was observed in younger and older groups. The results indicated that there were no significant differences between the two age groups in the slopes of SBP, DBP, MAP, and HR responses to isometric handgrip contractions with and without occlusion except for the slope of DBP response at 60% MVC with occlusion, which was significantly higher in the younger subjects. With respect to the intercept, SBP was significantly higher in older males at 20% and 40% MVC without occlusion. MAP was significantly higher in older males for all contraction intensities without occlusion. With occlusion, the intercept of SBP was significantly higher in older males in only one (60% MVC) of the contractions. Results further indicated that higher contraction intensities generally elicited greater responses of BP and HR in both age groups.

DISCUSSION

Previous studies have reported conflicting results with respect to pressor response to isometric exercise between older and younger males. Furthermore, it appears that there are no data available regarding age differences on the pattern of pressor response to isometric exercise with blood occlusion. Thus, this study involved two different age groups, two blood flow conditions, and four % MVC levels. The major focus of this study was in the pressor response to isometric handgrip contractions with and without blood occlusion between older and younger males.

The results of the present study indicate that the absolute pressor response to isometric handgrip contractions without blood occlusion was significantly higher in the older age group. These results are in contradiction to those of McDermott et al. (1974) and Sagiv et al. (1988) who reported that there was no difference in pressor response to isometric contractions between the younger and the older males. These results are, however, in agreement with those of Goldstraw and Warren (1985) and Van Loan et al. (1989) who showed that older males had a significantly higher absolute pressor response to isometric exercise than that of younger males, although mean absolute HR was significantly lower in the older males. This lower HR response to isometric exercise observed in the older males is apparently due to a decrease in sympathetic tone with age (Kino et al., 1975; Van Loan et al., 1989). A lower stroke volume response to isometric exercise in older males has also been reported (Van Loan et al., 1989). Thus, the results of this study indicate that there is a different response to isometric handgrip contractions in the two age groups. Since the two major determinants of the blood pressure response are cardiac output and total peripheral resistance (Donald et al., 1967), this would suggest that the higher pressor response in the older males is the result of a larger increase in peripheral vascular resistance

which is probably due to a decrease in the elasticity of the blood vessels (Lakatta et al., 1987) and / or a decline in the beta-adrenergic receptors mediated vasodilation (Hiremath et al., 1989) rather than an increase in cardiac output. Therefore, this study and previous studies suggest that the higher absolute pressor response in the older males probably has a functional importance to maintain the blood supply to the contracting muscles during isometric effort to compensate for a lower cardiac output response in normal aging. .

In this study, although the absolute SBP response with blood occlusion was significantly higher in the older males than in the younger males, the absolute SBP response with occlusion was significantly greater than without occlusion at submaximum isometric levels only in the younger age group. This could suggest that younger males have a better ability to increase SBP to overcome the combined effects of submaximum isometric exercise and blood occlusion and have a better ability to maintain the blood supply to the contracting muscles. The increase in DBP response to isometric exercise is a reflection of an increase in peripheral resistance (Durstine and Pate, 1988). The increase in DBP was similar for both age groups at submaximum isometric levels with occlusion indicating that peripheral resistance was similar in both age groups. With respect to the two blood flow conditions, the results showed that there were no significant differences in the pressor response at MVC between occlusion and no occlusion in both age groups. This indicate that the intramuscular pressure at MVC without occlusion is sufficient to completely restrict blood flow. An interesting finding of this study was the fact that HR response to all isometric contractions was similar between occlusion and no occlusion in both age groups. This would be in agreement with the suggestion of Bull et al. (1989) and Gandevia and Hobbs (1990) who indicated that the metabolites trapped within the contracting muscles by blood occlusion do not influence HR in young subjects. Mean absolute HR

response to isometric contractions with occlusion was also significantly lower in the older males. Thus, this study would also suggest that the lower HR response to isometric contractions in older males with occlusion is not compensated by a further increase in pressor response as was observed without occlusion.

With respect to the younger group, the results of this study are in general agreement with the study of Coote et al. (1971) which showed that the increase in pressor response was greater during isometric exercise with occlusion than that obtained at an identical intensity without occlusion. The present data showed significant differences in the SBP with and without occlusion at 20% and 40% MVC but no significant differences between the two blood flow conditions at 60% MVC in the younger males. This could indirectly indicate that at a contraction of 60% MVC, circulation in the normal human forearm muscles is nearly completely restricted. This supports Humphreys and Lind (1963) who indicated that blood flow through the forearm is completely occluded when the intensity of contraction is greater than 70% MVC.

The pressor response to isometric exercise in this study was similar to the investigation of Donald et al. (1967) which examined the pressor response at different handgrip contractions (10%, 20%, and 50% MVC) with and without blood occlusion. They showed that the difference in pressor response was most marked at the lower intensities of contractions (10% and 20% MVC) and the least at the highest intensity (50% MVC). Therefore, the higher pressor response during the combination of occlusion and isometric contraction in this study indicates that the reflex control mechanism during submaximum isometric exercise with blood occlusion can contribute to a higher pressor response in the younger males. Muscle chemoreflexes could play an important part in this control mechanism.

A somewhat unexpected finding of this study was the similar pressor response observed in the older males during the submaximum handgrip contractions both with

and without occlusion. Bull et al. (1989) and Gandevia and Hobbs (1990) indicated that the maintained elevation of blood pressure following contraction with occlusion is due to muscle chemoreflex activity. The results of this study therefore suggest that the metabolites trapped within the contracting muscles by blood occlusion at the submaximum handgrip contractions indicate that muscle chemoreflex does not play an important part in the reflex control mechanism in the older males. Mitchell et al. (1981) suggested that there is a greater input from receptors in the contracting muscle when a greater muscle mass is involved. A number of studies have indicated that normal aging is accompanied by a progressively decreasing muscle mass (Pearson et al., 1985; Kallman et al., 1990). Thus, the decrease in the contribution of muscle chemoreflex in the older males could be the result of a decrease in the reflex input mediated by the receptors (Group IV), most likely due to a decrease in muscle mass associated with age.

The results of the regression analyses showed that there were no differences in the slopes to isometric exercise without occlusion in the two age groups. These data are in agreement with those of Goldstraw and Warren (1985) who indicated that the pattern of pressor response to isometric exercise was not influenced by age. In addition, the present study showed that there were also no differences in the slopes to isometric exercise with occlusion in the two age groups except for the slope of DBP response at 60 %MVC. Thus, this would indicate that the pattern of pressor response to isometric exercise with occlusion is also not influenced by age.

In conclusion, although absolute pressor response to isometric handgrip contractions was higher in older males, the pattern of pressor response to isometric handgrip contractions (20%, 40%, and 60% MVC for 30 seconds and MVC for 12 seconds) were not influenced by age. Blood occlusion during isometric handgrip contractions did not influence the pressor response of older males. However, blood

occlusion increased the pressor response of younger males at lower percentages of MVC.

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Table 1. Mean (±SD) age, physical characteristics, and maximal handgrip contraction values for the two age groups.

Variables	Younger(n=10)	Older(n=10)
Age (years)	26.6±1.9*	54.7±3.0
Height (cm)	178.1±5.7	175.4±8.9
Weight (kg)	77.8±11.3	80.8±7.8
BMI (kg/m^2)	24.5±2.9	26.3±1.8
SBP (mmHg)	118.0±5.6*	126.0±9.8
DBP (mmHg)	72.8±6.6	79.4±7.6
HR (b/min)	68.6±6.5	66.0±11
PVo2max (ml/kg/min)	52.1±5.2*	35.9±3.3
MaxHG (kPa)	82.4±13.3	86.2±8.8
#ΔSBP (mmHg)	29.6±10.8	19.8±9.8

Note: *Significant differences found between the two age groups at the $p<0.05$ level.
#Systolic blood pressure of the first stage of dynamic exercise minus the resting systolic blood pressure.

Table 2. Mean responses of systolic (SBP), diastolic (DBP), mean arterial pressure (MAP), and heart rate (HR) at rest and during different %MVC levels with and without occlusion for the two age groups.

Blood flow conditions & Age groups	Rest	20%	40%	60%	MVC
Systolic blood pressure					
No occl.& Younger	115±9*	128±12*	134±11*	144±17*	137±16*
No occl.& Older	125±12	142±17	149±19	158±26	153±21
Occl.& Younger		#135±10*	#141±15*	149±20*	140±14*
Occl.& Older		144±17	147±20	159±22	158±18
Diastolic blood pressure					
No occl.& Younger	69±10*	74±10*	80±13*	86±13*	84±12*
No occl.& Older	75±9	80±13	86±12	91±16	95±16
Occl.& Younger		#81±8	#85±9	#93±13	87±16*
Occl.& Older		84±12	89±14	93±17	96±14
Mean blood pressure					
No occl.& Younger	82±10*	88±11*	94±13*	101±14*	98±12*
NO Occl.& Older	91±10	99±14	104±15	111±18	112±18
Occl.& Younger		#95±9*	#100±11*	#108±14	101±16*
Occl.& Older		102±13	107±16	113±19	114±16
Heart rate					
No occl.& Younger	69±10*	69±9*	75±10*	82±12*	87±12*
No occl.& Older	63±12	65±13	67±13	74±15	77±16
Occl.& Younger		69±11*	76±14*	85±15*	85±13*
Occl.& Older		60±13	65±13	69±16	75±19

Note: * Significant differences found between two age groups within the blood flow condition at the $p<0.05$ level.

Significant differences found between two blood flow conditions within the age group at the $p<0.05$ level.

Table 3. Regression slopes and intercepts of SBP and DBP responses to different %MVC with and without occlusion for the two age groups.

%MVC levels	Blood flow conditions & Age groups	Slope	Intercept
SBP 20%MVC	No occl & Younger	0.00±0.46	128.1±8.2*
	No occl & Older	-0.08±0.66	142.7±11.9
	Occl & Younger	0.05±0.38	134.5±6.9
	Occl & Older	0.11±0.64	142.0±11.6
SBP 40%MVC	No occl & Younger	0.45±0.37	127.5±6.7*
	No occl & Older	0.32±0.73	143.7±13.1
	Occl & Younger	0.51±0.52	133.0±9.3
	Occl & Older	0.67±0.71	136.8±12.8
SBP 60%MVC	No occl & Younger	0.82±0.58	131.6±10.5
	No occl & Older	0.82±0.94	145.8±16.9
	Occl & Younger	1.07±0.62	132.5±11.1*
	Occl & Older	0.79±0.81	146.6±14.7
SBP MVC	No occl & Younger	0.22±1.49	136.0±10.8
	No occl & Older	1.94±2.02	142.0±14.2
	Occl & Younger	0.73±1.38	135.4±9.6
	Occl & Older	2.04±1.65	146.2±11.9
DBP 20%MVC	No occl & Younger	0.07±0.38	73.2±6.8
	No occl & Older	-0.01±0.49	80.3±8.8
	Occl & Younger	0.17±0.30	78.6±5.5
	Occl & Older	0.10±0.44	82.7±7.9
DBP 40%MVC	No occl & Younger	0.42±0.47	74.1±8.5
	No occl & Older	0.18±0.46	83.3±8.2
	Occl & Younger	0.47±0.30	78.4±5.4
	Occl & Older	0.35±0.52	83.6±9.3
DBP 60%MVC	No occl & Younger	0.64±0.42	76.4±7.6
	No occl & Older	0.39±0.57	85.2±10.3
	Occl & Younger	0.87±0.36*	80.1±6.6
	Occl & Older	0.49±0.65	85.1±11.8
DBP MVC	No occl & Younger	1.07±1.03	77.1±7.4
	No occl & Older	1.81±1.39	84.3±9.7
	Occl & Younger	1.01±1.52	81.0±10.6
	Occl & Older	1.91±1.20	84.6±8.6

Note: * Significant differences found between two age groups within blood flow condition at the $p<0.05$ level.

Table 4. Regression slopes and intercepts of MAP and HR responses to different %MVC with and without occlusion for the two age groups.

%MVC levels	Blood flow conditions & Age groups	Slope	Intercept
MAP 20%MVC	No occl & Younger	0.07±0.41	86.8±7.4*
	No occl & Older	-0.02±0.53	99.2±9.6
	Occl & Younger	0.18±0.33	92.3±6.1
	Occl & Older	0.10±0.48	100.7±8.7
MAP 40%MVC	No occl & Younger	0.42±0.46	87.5±8.2*
	No occl & Older	0.22±0.56	101.1±10.1
	Occl & Younger	0.53±0.36	92.1±6.6
	Occl & Older	0.45±0.58	100.2±10.4
MAP 60%MVC	No occl & Younger	0.75±0.46	89.6±8.4*
	No occl & Older	0.47±0.67	103.6±12.1
	Occl & Younger	0.93±0.41	94.1±7.5
	Occl & Older	0.60±0.68	103.6±12.4
MAP MVC	No occl & Younger	1.07±1.08	91.6±7.8*
	No occl & Older	1.74±1.63	102.1±11.4
	Occl & Younger	1.11±1.48	94.5±10.3
	Occl & Older	1.94±1.45	102.8±10.5
HR 20%MVC	No occl & Younger	0.02±0.35	68.4±6.4
	No occl & Older	0.06±0.51	63.9±9.1
	Occl & Younger	-0.02±0.42	68.8±7.6
	Occl & Older	0.04±0.50	59.8±9.0
HR 40%MVC	No occl & Younger	0.20±0.36	71.9±6.5
	No occl & Older	0.18±0.48	64.6±8.6
	Occl & Younger	0.23±0.52	72.2±9.4
	Occl & Older	0.14±0.48	62.9±8.7
HR 60%MVC	No occl & Younger	0.62±0.38	72.2±6.9
	No occl & Older	0.36±0.53	68.2±9.6
	Occl & Younger	0.65±0.51	74.8±9.2
	Occl & Older	0.31±0.59	64.1±10.7
HR MVC	No occl & Younger	2.13±0.77	73.9±5.6
	No occl & Older	2.02±1.37	65.0±9.6
	Occl & Younger	2.44±0.92	70.7±6.4
	Occl & Older	2.30±1.61	61.0±11.6

Note:*Significant differences found between two age groups within blood flow condition at the $P<0.05$ level.

FIG1.SBP RESPONSES TO DIFFERENT %MVC WITH & WITHOUT OCCLUSION

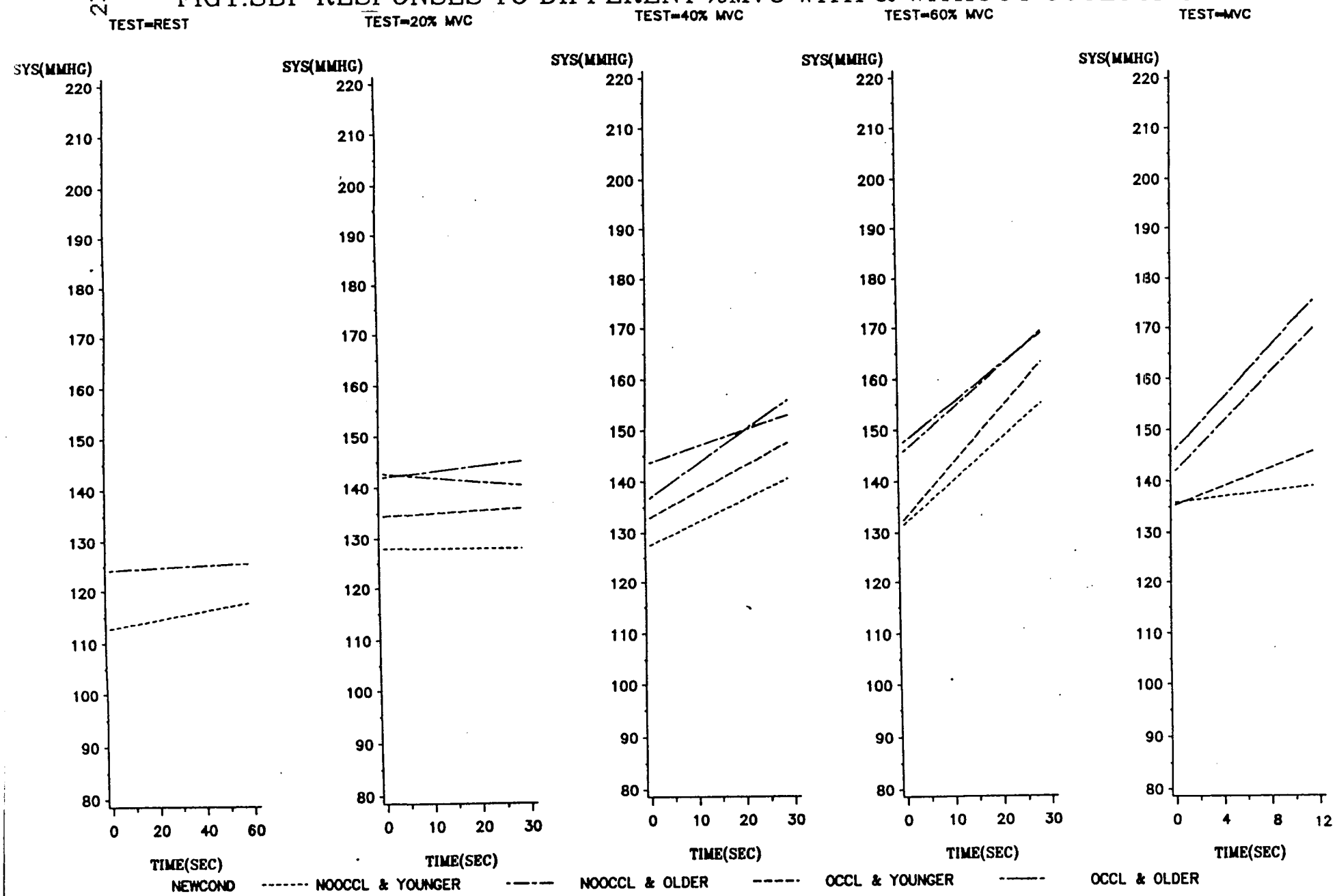


FIG2.DBP RESPONSES TO DIFFERENT %MVC WITH & WITHOUT OCCLUSION

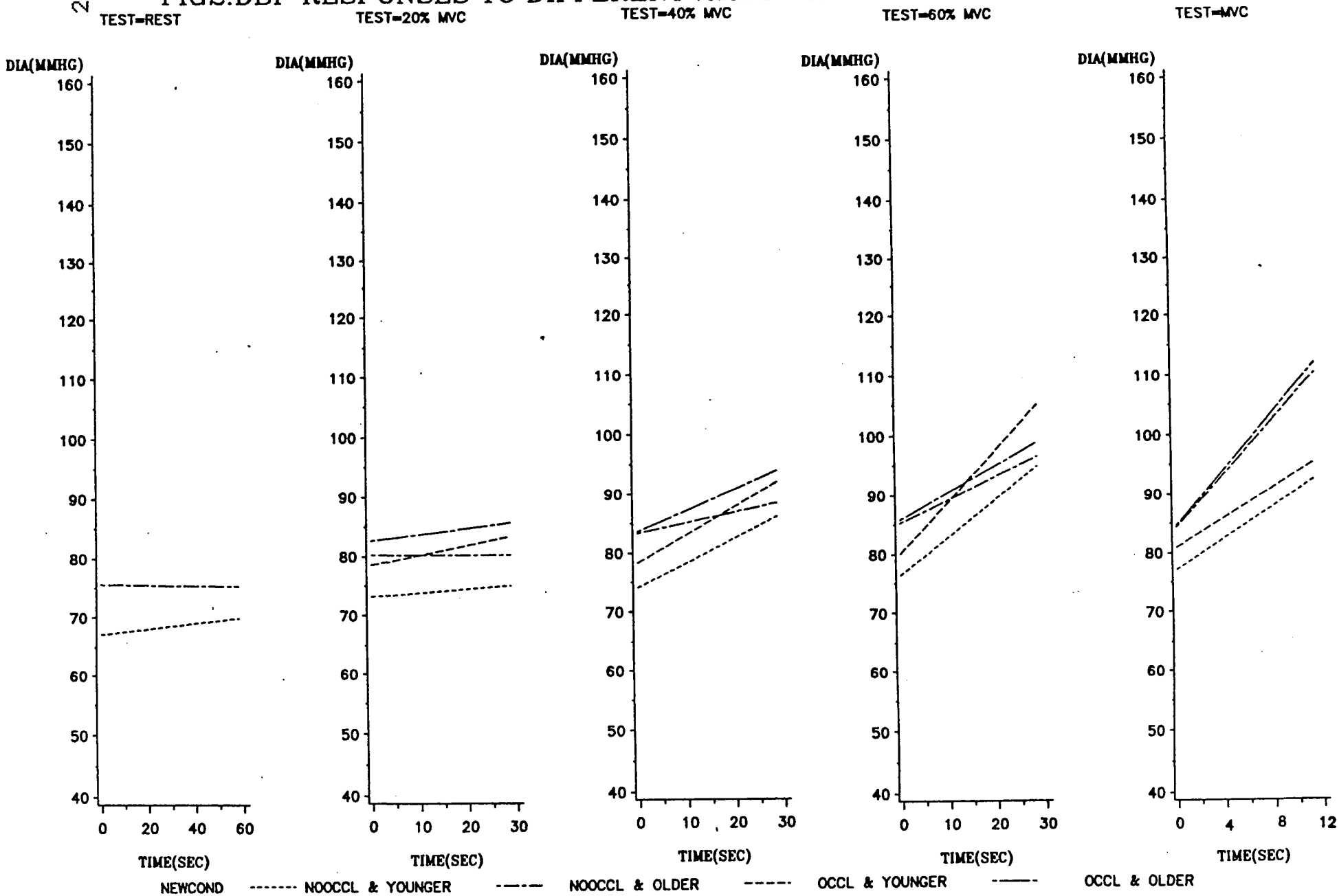


FIG3.MAP RESPONSES TO DIFFERENT %MVC WITH & WITHOUT OCCLUSION

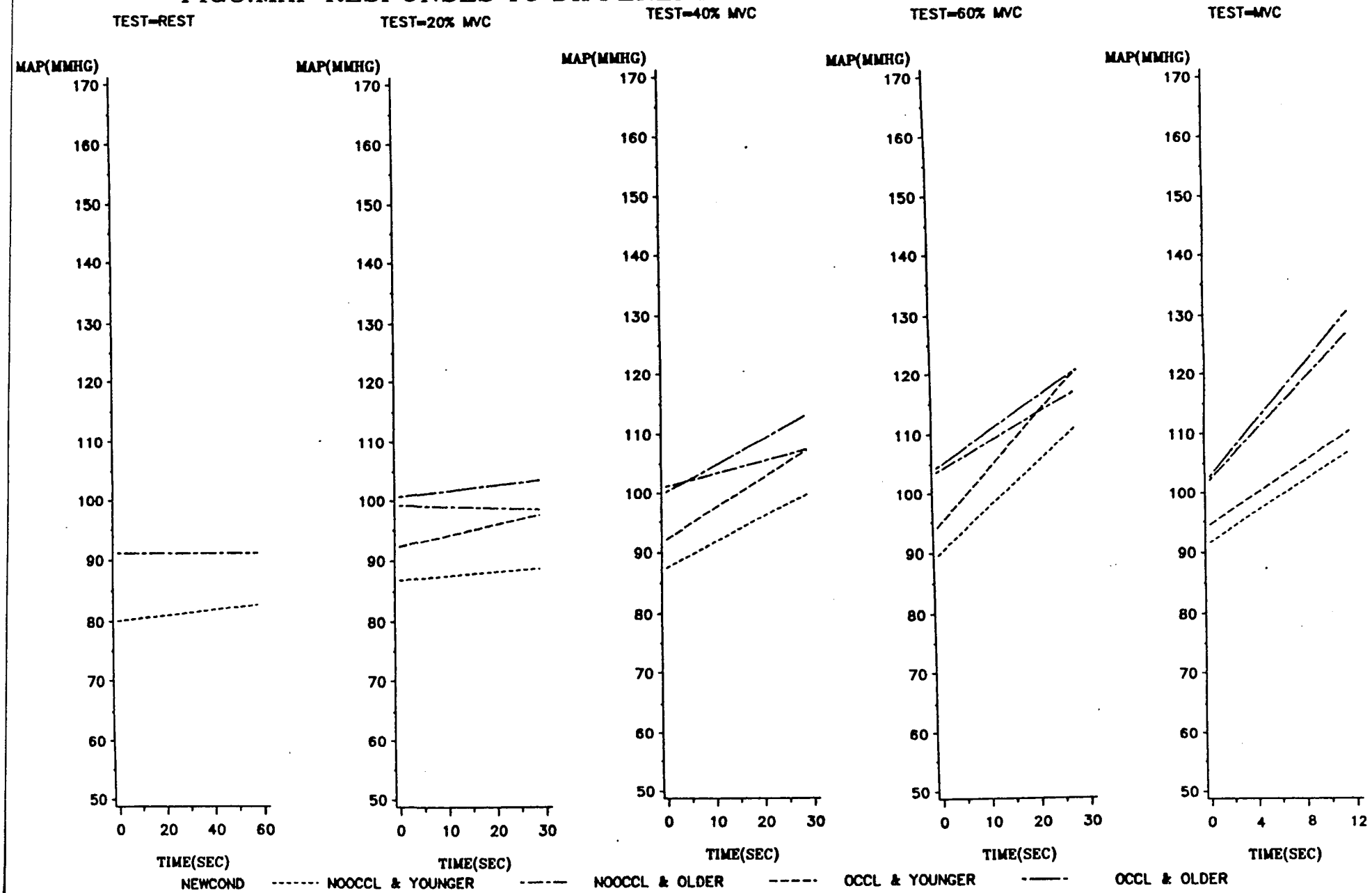
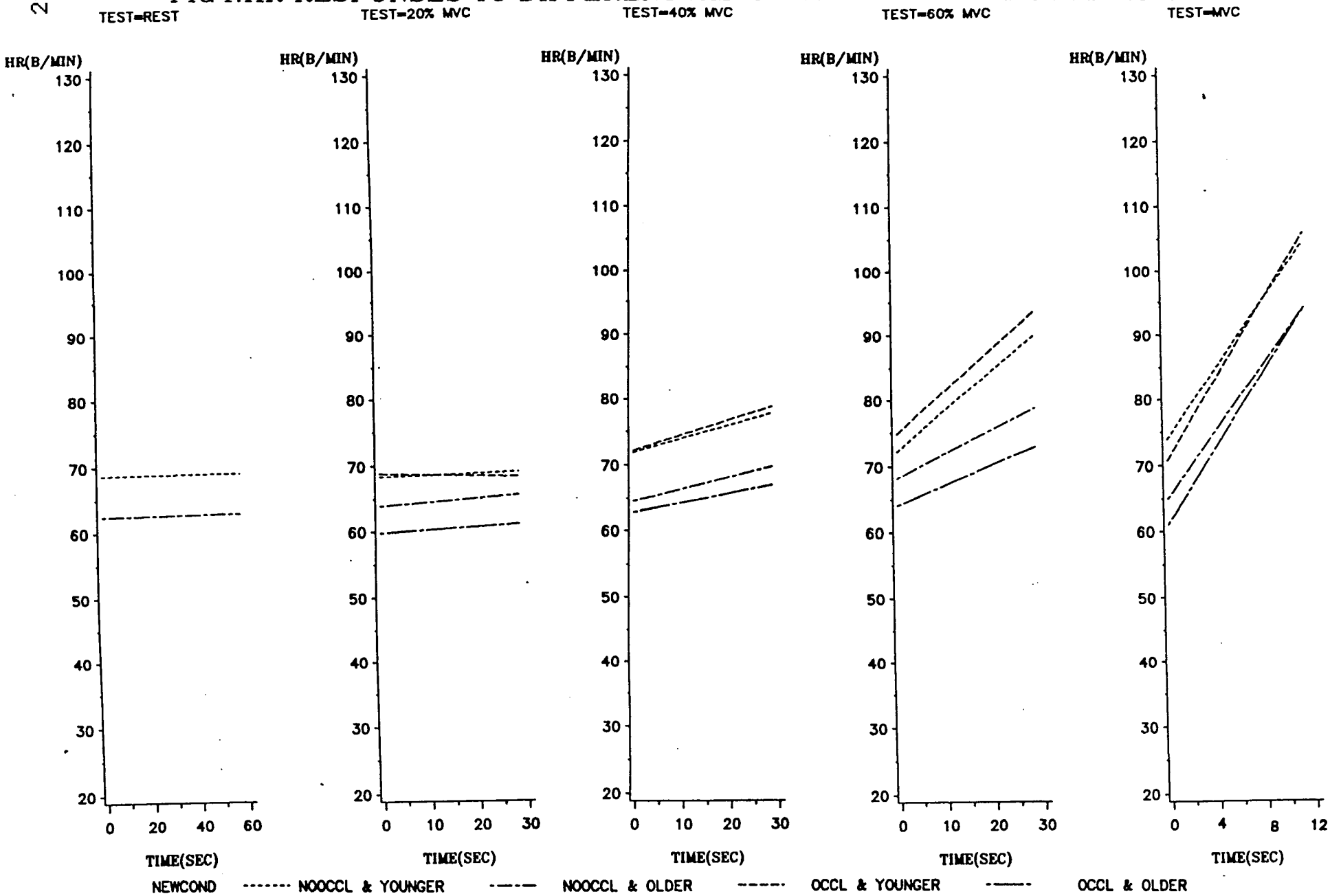


FIG4.HR RESPONSES TO DIFFERENT %MVC WITH & WITHOUT OCCLUSION



I

INTRODUCTION

1.1 Introduction

The normal human aging process is accompanied by progressive physiological changes which contribute to the elevation of blood pressure both at rest and during exercise (Shimada et al., 1985; Pan et al., 1986; Lakatta et al., 1987; Lipsitz, 1989; Jetté et al., 1991). Recent studies suggest that the increase in the left ventricular mass (Weber, 1988; Jetté et al., 1991) and the decrease in the compliance of the blood vessels (Lakatta et al., 1987) associated with aging indeed influence blood pressure regulation. Several studies have also indicated a progressive age-related reduction in the baroreflex-mediated heart rate response to alterations in blood pressure (Shimada et al., 1985; Hainsworth & Al-shamma, 1988). In addition, other researchers demonstrated that beta-adrenergic receptors mediated vasodilation is reduced with aging (Vestal et al., 1979; Pan et al., 1986; Hiremath et al., 1989).

It is also well recognized that aging is associated with a progressive decline in muscle strength (Larsson et al., 1979) and mass (Pearson et al., 1985). This decline accelerates at about the age of 50 (Larsson et al., 1979; Kallman et al., 1990). In fact, both muscle strength (Lind and McNicol, 1967) and mass (Mitchell et al., 1980) are said to play a significant role in blood pressure (pressor) response to an isometric exercise.

Various investigations of age differences on the blood pressure response to isometric exercise have led to different results. Petrofsky and Lind (1975) reported that systolic blood pressure is significantly greater in older males than in younger males at rest and at 40% maximum voluntary isometric handgrip contraction (MVC), whereas there is no significant difference in diastolic blood pressure. They indicated that the

greater systolic blood pressure response in older males is due to an increase in the stiffness of the blood vessels. Baldwa et al. (1983) showed that both systolic and diastolic blood pressures are higher in older males than in younger males both at rest and during isometric handgrip contraction at 30% MVC level. They suggested that the higher pressor response in older males is due to an increase in peripheral vascular resistance. Van Loan et al. (1989) showed a greater pressor response to isometric exercise performed at various % MVC levels (15, 30, 45, and 60% MVC) in older males. They indicated that the differences between the two age groups are due to changes in the vascular system associated with aging. In contrast, McDermott et al. (1974) reported that the blood pressure response is similar at 33% MVC for both young and old males, although a hyperadrenergic state induced by isometric exercise is associated with aging. Sagiv et al. (1988) reported a similar blood pressure response to 30% isometric contraction for handgrip and deadlift in young and old males. They indicated that there is no significant age effect associated with left ventricular function and hemodynamic responses during isometric exercise. Ordway and Wekstein (1979) however, showed that diastolic blood pressure is lower during different % MVC handgrip levels (15 and 30% MVC) in older males. They suggested that the reduced diastolic blood pressure response in older males may be due to a decrease in their heart rate response. However, Goldstraw and Warren (1985) indicated that increasing age does not change the pattern of the blood pressure response to different % MVC levels (10, 20 and 30% MVC), although the resting blood pressure increases with age.

A rise in blood pressure is maintained at a high level by blood occlusion after cessation of isometric exercise due to the metabolites trapped within the active muscles (Donald et al., 1967; Freund et al., 1978; Mitchell et al., 1981). The classic work of Alam and Smirk (1937) indicated that the rise in blood pressure is much

greater during forearm exercise with blood occlusion than that obtained at an identical intensity without blood occlusion. However, few studies compared the pressor response to isometric exercise between older and younger males in a continuous manner. In addition, it appears that there are no data with regard to age differences on the pressor response to isometric exercise with blood occlusion. Therefore, further investigation is required to compare age differences on the pressor response to isometric handgrip contractions at different % MVC levels with and without upper arm blood occlusion.

1.2 Statement of The Problem

The purpose of this study was to compare the pattern of pressor response between older and younger males measured continuously with and without blood occlusion during isometric handgrip contractions at different % MVC levels.

1.3 Hypothesis

The pattern of pressor response to isometric handgrip contractions at different % MVC levels with and without blood occlusion will be affected by age differences.

1.4 Scope of The Study

Ten males between the ages of 20-29 years and ten between the ages of 50-59 years volunteered to participate for the study. Prior to isometric testing, all subjects performed the CAFT to ensure that they did not show an exaggerated blood pressure response to dynamic exercise. The subjects performed dominant handgrip contractions at 20%, 40% and 60% MVC levels (independent variables) for a period of 30 seconds, at MVC for a 12 second period with and without blood occlusion. The systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR) (dependent variables) were measured using a Finapres 2300 BP Monitor. A 2x2x4 factorial design ANOVA with repeated measures was conducted on the mean values for cardiovascular responses to determine if there were

any significant differences in BP and HR with respect to the two age groups with and without blood occlusion at four different % MVC levels of isometric exercises. The slope of cardiovascular responses to the various conditions was calculated and the results applied in a 2x2x4 factorial ANOVA design with repeated measures on these factors to determine if there were any significant differences among the means.

1.4 Definition of Terms And Abbreviations

Aging:

A progressive loss of physiological adaptability (impaired homeostasis) to the environment.

Maximum voluntary contraction(MVC):

The maximum force of a single or group of muscles exerted by a single maximal contraction.

Pressor response:

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

II

REVIEW OF LITERATURE

2.1 Introduction

Investigations of age differences on the pressor response to isometric exercise have led to conflicting results. Several studies have showed that the blood pressure is higher in older males than younger males both at rest and during different % MVC levels. Conversely, other studies have demonstrated a similar blood pressure response to isometric exercise in different age groups. In addition, the study by Goldstraw and Warren (1985) indicated that aging does not change the pattern of the blood pressure response at different % MVC levels, even though the resting blood pressure is higher with aging. However, their study was limited because the blood pressure was recorded only by a sphygmomanometer and stethoscope, thus restricting the number of measurements that can be taken during the relatively short contraction time. In addition, it appears that no study compared pressor response between older and younger males at different % MVC levels with blood occlusion. Therefore, the purpose of this study was to compare with continuous BP measurement, the pressor response to different % MVC levels during handgrip contractions with and without blood occlusion between older and younger males.

2.2 Mechanisms for the cardiovascular response to isometric exercise

There are two separate mechanisms of neural control for the cardiovascular response to isometric exercise: the reflex control mechanism and the central control mechanism (Mitchell, 1985).

2.3 Reflex Control Mechanism

The reflex control mechanism was first observed by Alam and Smirk (1937) in that the rise in blood pressure is reflexly induced by the accumulation of metabolites in the

contracting muscles. Lind and McNicol (1967) supported the reflex control mechanism indicating that the activity of metabolites reflexly governs the cardiovascular responses to isometric exercise. Further evidence by Coote et al. (1971) showed that the reflex response is abolished by sectioning the dorsal root afferents from the active muscles. Mitchell et al. (1977), in agreement with Coote et al. (1971), indicated that electrically stimulating the peripheral ends of sectioned spinal ventral roots still cause an increase in the cardiovascular responses during induced isometric exercise. Thus, the isometric muscle contraction reflexly causes cardiovascular response due to afferent impulses being conducted in the corresponding dorsal roots. Hultman and Sjoholm (1982) reported that the stimulated afferents within the active muscles have the full ability to regulate the cardiovascular responses to static exercise. This reflex control mechanism, which was termed the " exercise pressor reflex " by Mitchell et al. (1983), would serve as a feedback control for the cardiovascular responses during isometric exercise. Mitchell et al. (1989b) further studied the effect of epidural anesthesia (epidural anesthesia not only reduces muscle strength by motoneuron blockade but also blocks sensory feedback from the contracting muscles) on the cardiovascular responses to isometric exercise in human subjects. The experiments with epidural anesthesia showed that an increase in central command is related to the actual force developed but is not associated with the cardiovascular responses. The results of their study indicated that the reflex control mechanism rather than the central command is important in determining the magnitude of the cardiovascular responses to isometric exercise. Additionally, Mitchell (1985) stated that the reflex cardiovascular response is due to afferent impulses being conducted by the Group III and IV afferents during isometric exercise.

2.3.1 Group III and IV afferents

The afferents from skeletal muscles have been divided into four groups based on

both anatomical and electrophysiological measurements (Mitchell, 1985). Andres et al. (1985) indicated that the endings of Group III afferents are associated with collagen structures in the skeletal muscle and the endings of Group IV afferents are associated with blood vessels. Kniffki et al. (1981) indicated that the majority of afferents in mammalian skeletal muscles are of the Group III (thin myelinated and slow conduction velocity) and the Group IV (unmyelinated and the slowest conduction velocity) type afferents. The studies of McCloskey and Mitchell (1972) showed that blockade of the large myelinated afferent nerves (Group I & II) in the spinal dorsal roots had no effect on the cardiovascular responses to induced muscle contraction. They also showed that blockade of the fine myelinated afferent nerves (Group III & IV) in the spinal dorsal roots abolished the cardiovascular responses to induced muscle contraction. In addition, McCloskey, Matthews & Mitchell (1972) observed that the electrical and mechanical activation of Group I and II muscle afferents show little or no cardiovascular response (Group I and II innervate muscle spindles and tendon organs). Waldrop et al. (1984) also reported that the chemical stimulation of Group I and II muscle afferents again show no cardiovascular response. Conversely, Mitchell et al. (1983) and Kaufman et al. (1984) indicated that the Group III and IV muscle afferents when electrically or chemically activated show cardiovascular responses to the induced contractions. According to Mitchell et al. (1983), the Group III and IV muscle afferents can be divided into two groups, ergoreceptors (exercise pressor reflex) and nociceptors (pain receptors); however most of the Group III and IV muscle afferents have combined functions. Kniffki et al. (1981) suggested that most of the Group III are ergoreceptors (low threshold), whereas most of the Group IV are nociceptors (high threshold). Kaufman et al. (1983) and Mitchell et al. (1983) also provided further evidence indicating that the Group III and IV muscle afferents contribute to the reflex control mechanism, which is related to both mechanical and

metabolic activity in the active skeletal muscles. Neural discharges related to the mechanical activity may be sent to the medullary cardiovascular centre primarily by Group III muscle afferents, whereas neural discharges related to the metabolic activity may be sent to the medullary cardiovascular centre primarily by Group IV muscle afferents. Kaufman et al. (1984) concluded that the enhanced cardiovascular responses are more common in Group IV afferents than in Group III afferents during ischemic contraction because of the fact that Group IV afferents are more sensitive to the metabolic products of ischemic contraction than are Group III afferents. However, the actual reflex control pathways are unknown (Shepherd et al., 1981). The precise metabolites activating Group III and IV afferents during isometric exercise are also not clearly defined.

2.3.2 Metabolites

The cardiovascular responses to the intensity of exercise serve to direct a major function of circulatory delivery of oxygen to active skeletal muscles. Kaufman et al. (1984) suggested that the resultant mismatch between blood supply and blood demand generates metabolites within muscles which is sensed by Group III and IV afferents during ischemic contractions. Alam and Smirk (1937) hypothesized that the accumulation of metabolites from contracting muscles may cause a rise in blood pressure. One test for this hypothesis was to continue occlusion after cessation of exercise. The results indicated that the metabolites trapped within the muscles, as a constant or increasing pressure-raising stimulus, do cause the increase in blood pressure. By comparison, Kaufman et al. (1984) demonstrated that circulatory occlusion in resting skeletal muscles causes no cardiovascular responses due to very low metabolite rate within inactive muscles. The results of this study also showed that the Group III and IV afferents are stimulated more by ischemic contractions (more metabolites) than by nonischemic contractions. Therefore, the results suggested that

the exercise pressor reflex may not be fully activated during mild (nonischemic) contractions but the increased cardiovascular responses to ischemic contractions is modulated by metabolic factor stimulating Group III and IV afferents. Mitchell (1985) also indicated, in agreement with Kaufman et al. (1984) that the metabolic factor may not be activated during light-intensity exercise and the metabolic factor may act as a contributing factor in the reflex cardiovascular responses during isometric contraction at a level that restricts blood flow. Thus, the increase in cardiovascular responses to isometric exercise is generally considered to be a reflex drive, originating in the contracting muscles, and probably initiated by potassium.

2.3.3 Potassium Ion

A number of studies have suggested that the possible metabolic product to activate Group III and IV afferents may be potassium (Saltin et al., 1981; Rybicki et al., 1984; Kaufman et al., 1984). Wildenthal et al. (1968) indicated that potassium released from contracting muscle is well known. They also found increased cardiovascular responses during potassium infusion in dogs. The data indicated that potassium efflux from the contracting muscle induces cardiovascular response to isometric exercise. Saltin et al. (1981) concluded that the change in potassium concentration in the femoral venous blood rather than lactate concentration, closely matches the blood pressure response during and after the isometric exercise in human subjects. Rybicki et al. (1984) suggested that the rise in interstitial potassium concentration may be a contributing factor in the exercise pressor reflex in the active muscle during isometric exercise. The results showed the enhanced cardiovascular responses following change in potassium concentration (KCl solution injection) in dogs. Donald et al. (1967) showed that the efflux of potassium is more closely parallelled to the pressor response than the efflux of lactate and hydrogen ion during and after isometric exercise. Shepherd et al. (1981) indicated that the potassium ion efflux from the

contracting muscle is likely correlated with the rise in blood pressure.

Therefore, the exercise pressor reflex would serve as a feedback control in the increase in cardiovascular responses originating in the contracting muscles themselves (Figure 5). This process which is related to both mechanical and metabolic activity in the active muscles would monitor the effectiveness of the blood flow to meet the increased metabolic needs in the active muscles. Group III afferents are more sensitive to the mechanical activity in the contracting muscles, whereas Group IV afferents are more sensitive to the metabolic activity in the contracting muscles. However most of the Group III and IV muscle afferents have combined functions.

2.4 Central Control Mechanism

The central control mechanism of pressor response to isometric exercise was first expressed in 1894 by Johansson (cited in Mitchell et al., 1981). It suggests that the stimulus descends from the higher motor centres and is responsible for the cardiovascular responses to isometric exercise. Goodwin, McCloskey & Mitchell (1972) examined the central control mechanism during isometric exercise with vibration and without vibration stimulus of the biceps in human. The results showed that the changes in blood pressure are associated with the changes in central command. These authors indicated that there is central modification to the magnitude of the cardiovascular responses which is dependent on the descending central command during isometric exercise. Since vibration is known to be a powerful stimulus to the primary afferents of muscle spindles (these receptors are not involved in reflex cardiovascular response), the less central command is required to maintain the same force due to an involuntary increase in motor activation of the muscle (Mitchell et al., 1981). In addition, Waldrop et al. (1986) investigated the cardiovascular responses to electrical stimulation of the subthalamic locomotor region (STLR) in anesthetized cats. The results showed that stimulation in the STLR elicited immediate

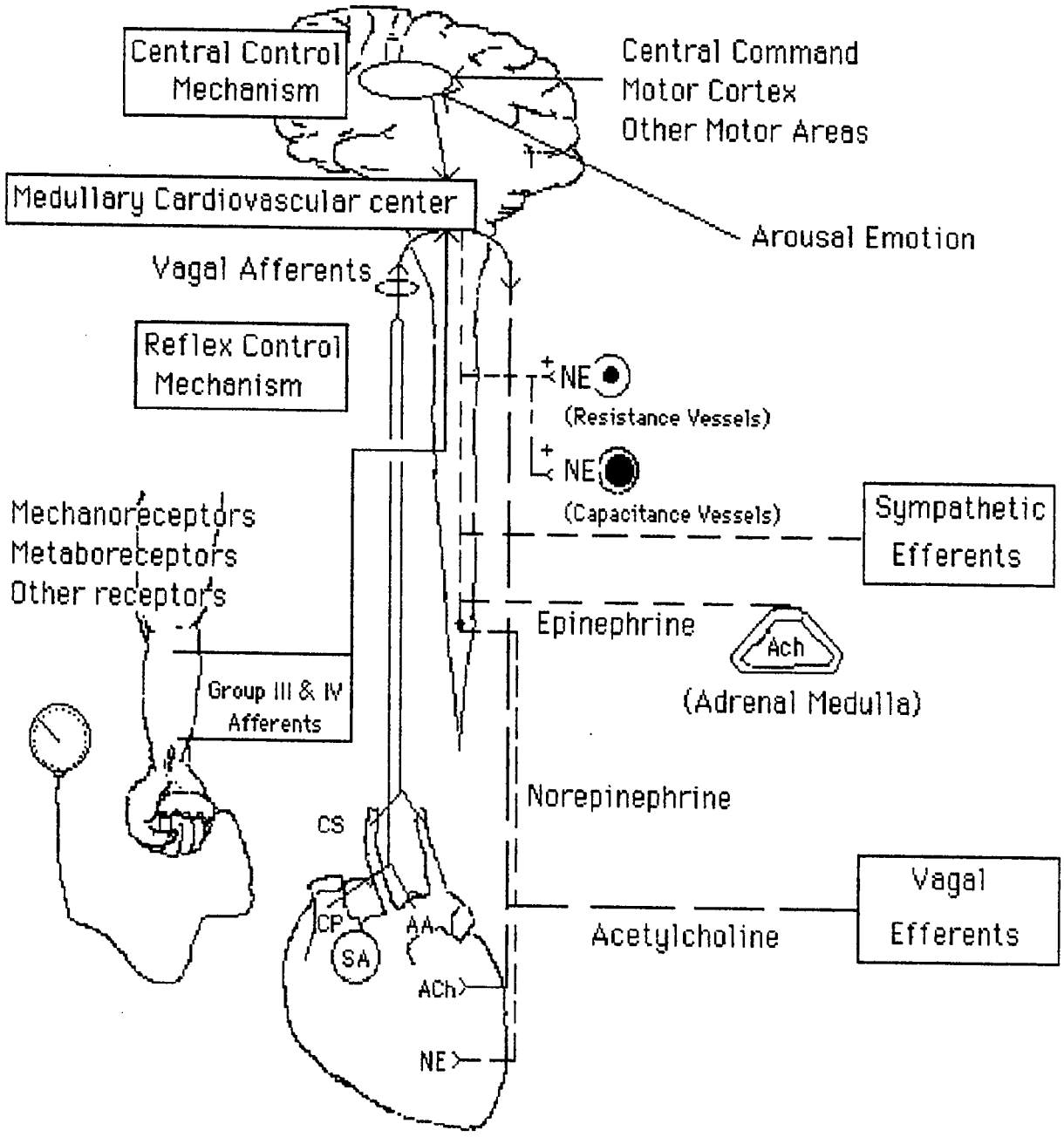


Figure 5. Neural control mechanisms of cardiovascular responses to isometric exercise. (adapt from Shepherd et al., 1981)

increases in BP and HR. When the STLR stimulation was stopped, the cardiovascular responses returned to control values. They indicated that the important region of mediating central command, STLR, causes the cardiovascular responses even when the cat is paralyzed. Mitchell (1985) stated that central control mechanism is related to the central activity for the recruitment of motor units and determines the level of the autonomic nervous system activity to the heart and blood vessels. Additionally, in two similar studies, Leonard et al. (1985) and Mitchell et al. (1989a) investigated the effect of partial neuromuscular blockade (tubocurarine) on the cardiovascular responses to isometric exercise in human subjects. The experiments with tubocurarine during isometric voluntary exercise made it possible to induce a disproportion between an increase in central command and a constant or decreasing signal from receptors responding to muscle tension and metabolism. The results of these studies indicated that the signals arising from higher motor centres determine the magnitude of the cardiovascular responses to isometric exercise. Thus, the cardiovascular responses are determined by a central control mechanism served as a feedforward control. Central command and muscle fibre recruitment both contribute to the central control mechanism.

2.4.1 Central Command

The rapid change in cardiovascular responses to isometric exercise is thought to be caused by signals originating from the cerebral cortex to the cardiovascular centres in the medulla (Mitchell et al., 1981; Shepherd et al., 1981). Goodwin et al. (1972) was the first to name the signal from the higher motor centres to the medullary cardiovascular centre as " central command ". However, the exact location of the higher motor centres is not clear. Shepherd et al (1981) suggested that the central command impulses may arise in the motor cortex, other motor areas, and / or non-motor areas (arousal or emotion) as shown in Figure 5. Rusch et al. (1981)

investigated the influence of the central nervous system on blood pressure and heart rate responses. The results showed that the central command is important for the rise in blood pressure and heart rate. Funderburk et al. (1974) suggested that the heart rate response may reflect the combined effect of an early central command setting the initial rapid increase, and mechanical and metabolic factors related to contracting muscle causing the delayed, slower increase. Also, Kino et al. (1975) indicated that the rise in heart rate during isometric exercise is influenced by central command and reflex control, but the rise in blood pressure is considered to result from a reflex control. Conversely, Mitchell et al. (1981) suggested that the blood pressure response to isometric exercise represents the combined action of central command and reflex drive. However, the central command and reflex drive of the cardiovascular responses during isometric exercise could be varied more or less independently (Goodwin et al., 1972). Victor et al. (1989) examined the effect of partial neuromuscular blockade (curare) on sympathetic nerve responses to isometric exercise in human subjects. The results showed that subjects used near-maximal motor effort to attempt a sustained handgrip contraction, but they generated almost no force and much smaller increase in blood pressure response during curare. They indicated that the central command is responsible for the increase in cardiovascular responses during isometric exercise. Mitchell (1985) stated that central command is related to the central activity for muscle fibre recruitment. Therefore, this central command is responsible for the initial rise in cardiovascular responses to isometric exercise and is responsible for the muscle fibre recruitment.

2.4.2 Muscle Fibre Recruitment

The circulatory requirements of the active skeletal muscles would be directly related to the number of muscle motor units activated or to the discharge frequencies in recruited motor units. Therefore, cardiovascular responses to the intensity of exercise

would be some function of motor unit recruitment. Mitchell et al. (1980) concluded that more motor units are required during a larger muscle fibre recruitment for a given contraction intensity; thus, both central command and reflex drive may be also increased. Mitchell et al. (1981) examined the blood pressure response to isometric contractions between a large, strong leg and a small, weak leg in young men. The results indicated that a greater increase in systolic blood pressure and electromyographic activity (EMG) is observed in the stronger leg when each leg contracts at the same % MVC. These authors suggested that if a greater skeletal muscle mass were contracted, there would be a greater number of centrally activated motor units (increased central command). Also with the greater mass of skeletal muscle being involved, there would be a greater input from receptors in the skeletal muscle. Asmussen (1981) also suggested that more signals are sent to the medullary cardiovascular centre during a larger muscle fibre recruitment.

Thus, the central command would serve as a feedforward control in the rise in cardiovascular responses to isometric exercise, which is responsible for the recruitment of motor units and determines the immediate change in the level of the efferent nerve activity of the sympathetic and parasympathetic nervous systems to the cardiovascular system. 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.

2.5 Blood Flow to Contracting Muscle

Muscular exercise is associated with the cardiovascular responses which lead to an increase in blood flow to the active muscles (Perez-Gonzalez, 1981). Shepherd et al. (1981) indicated that the blood flow to contracting muscle is directly related to perfusion pressure and local metabolites as well as intramuscular pressure during isometric exercise. An increase in perfusion pressure results in a proportional

increase in the blood flow to contracting muscles (Lind & McNicol, 1967). A large increase in intramuscular pressure lowers the effective perfusion pressure and limits blood flow to contracting muscles during heavy isometric exercise (Asmussen, 1981; Sejersted et al., 1984). By comparison, Saltin et al. (1981) indicated that a mismatch between intramuscular pressure and blood flow also exists at lower levels of isometric exercise which generates the local metabolites. Consequently, the increased local metabolites cause relaxation of the resistance blood vessels which also increase the blood flow to contracting muscles (Shepherd et al., 1981). In the contracting muscles, the change in blood flow represents a compromise between metabolically induced vasodilation and compression of the blood vessels by the contracting muscles (Shepherd et al., 1981). The compression of the blood vessels is caused by the transferred intramuscular pressure during isometric exercise (Asmussen, 1981).

2.5.1 Intramuscular Pressure

Asmussen (1981) concluded that the progressive rise in intramuscular pressure in the active muscle causes a mechanical hindrance on local blood vessels during isometric exercise. The degree of hindrance depends on the contracting muscle mass and the relation between intramuscular pressure and blood pressure (Shepherd et al., 1981). Humphreys and Lind (1963) investigated the mechanical compression of the blood vessels through the active muscles during sustained contractions. The results indicated that the blood flow to contracting forearm muscles is not completely blocked until the intensity of isometric exercise exceeded 70% MVC. They concluded that the progressive reduction in blood flow through the contracting muscles is due to a progressive rise in intramuscular pressure. By comparison, Shepherd et al. (1981) suggested that the blood flow in leg muscle may block when the intensity of the isometric exercise exceeds 20% MVC. Sejersted et al. (1984) examined the intramuscular pressure at different locations within active muscles. They confirmed that

the intramuscular pressure is linearly related to the tension developed and that lower intramuscular pressure maintains better blood flow through the contracting muscles. Saltin et al. (1981) indicated a great variation in the extent of the intramuscular pressure at various locations within the active muscles. These authors also stated that local metabolites are released from the active muscles as a result of increased intramuscular pressure.

2.5.2 Local Metabolites

Some afferent nerves within the muscle that would detect the small change of the local metabolic activity serve to monitor the adequacy of muscle perfusion. One of the earlier observations by Alam and Smirk (1937) suggested that the resultant mismatch between blood supply and blood demand could generate more metabolites within active muscles following a progressive rise in the intensity of exercise. Subsequently, the metabolites released from the contracting muscles could cause the increased blood pressure. Lind and McNicol (1967) also suggested that isometric exercise is associated with a rise in blood pressure response which is related to the local metabolite concentration. Mitchell et al. (1980) stated that the rapid accumulation of metabolites from the contracting muscles can activate the reflex drive. Saltin et al. (1981) suggested that potassium is important for the regulation of the cardiovascular responses to isometric exercise, because the changes in potassium concentration of femoral venous blood closely follow the blood pressure response during and after the contraction. In addition, Kaufman et al (1984) indicated that the concentration of potassium rather than the concentration of lactate or phosphate stimulates exercise pressor reflex during ischemic isometric exercise. However, the accumulation of metabolites is caused by the hindrance of blood flow due to intramuscular pressure exceeding perfusion pressure during isometric exercise.

2.5.3 Perfusion Pressure

It is well known that an increase in perfusion pressure results in a proportional increase in blood flow. Lind et al. (1964) suggested that the rise in blood pressure by elevating the perfusion pressure may be of functional importance in maintaining the blood flow relatively constant through the contracting muscles by overcoming the mechanical hindrance on the local vessels. Shepherd et al. (1981) suggested, in agreement with Lind et al. (1964) that the enhanced blood pressure overcomes the mechanical obstruction to blood flow through the contracting muscles. Perez-Gonzalez (1981) reported that two circulatory mechanisms are triggered to increase blood flow to active muscle; a local response which causes vasodilation and a systemic response which increases perfusion pressure. Lind and McNicol (1967) indicated that the blood flow through the contracting muscles at the lower comparative % MVC is increased due to the raised perfusion pressure. By comparison, Saltin et al. (1981) indicated that the intramuscular pressure exceeds perfusion pressure at high levels of tension developed and thus increases vascular resistance and limits circulatory delivery of oxygen. Taken together, the increase in perfusion pressure is an important factor counteracting the increased intramuscular pressure resistance to blood flow in contracting muscles during isometric exercise.

2.5.4 Blood Occlusion of Arterial Supply

The pressor response to isometric exercise is higher during circulatory arrest than under normal conditions of blood flow due to the trapping of metabolites within the muscles. In Alam and Smirk's experiments (1937), limbs were occluded throughout exercise as well as recovery. They indicated that the rise in blood pressure is much greater during forearm exercise with blood occlusion than that obtained at the same intensity of the same forearm with the circulation free. Mitchell et al. (1981) studied the pressor response when the arterial supply was occluded immediately just before the end of exercise with the arrest maintained after the exercise. They indicated that the

increase in blood pressure after the contraction with blood occlusion is related to the muscle mass. Bull et al. (1989) investigated that the cardiovascular response with blood occlusion during and following voluntary contraction at 30% MVC in young subjects. The results showed that HR fell to control levels even though blood occlusion was maintained following exercise while BP remained at a significantly elevated level with blood occlusion following exercise. They concluded that maintained blood pressure elevation during blood occlusion may be due to trapped metabolites.

Therefore, the blood flow to contracting muscles during isometric exercise is a compromise between intramuscular pressure, local metabolites and perfusion pressure. The increased intramuscular pressure within the contracting muscles causes a mechanical resistance on the local blood vessels and limits blood flow to contracting muscles during isometric exercise. Consequently, the resultant mismatch between blood supply and blood demand generates metabolites from contracting muscles. The vasodilation due to local response and the rise in perfusion pressure due to systemic response maintain the blood flow relatively constant at low level % MVC through the contracting muscles by overcoming the increased resistance to blood flow due to intramuscular pressure during isometric exercise. The pressor response to isometric exercise is higher when the blood supply is occluded than under normal conditions of blood flow, due to the trapping of metabolites.

2.6 Tension Developed (% MVC) and Muscle Mass

Since the cardiovascular responses to isometric exercise are due to both central and reflex drives, several studies have been devised to investigate whether the reflex from the contracting muscles is related to the relative tension developed or to the muscle mass (Alam & Smirk, 1937; Lind & McNicol, 1967; Donald et al., 1967; Mitchell et al., 1980; Seals et al., 1983; Misner et al., 1990). Lind and McNicol (1967) indicated that the increased cardiovascular responses are related to the percentage of maximal

tension developed due to an increased reflex stimulus. In contrast, Mitchell et al. (1980) stated that the enhanced cardiovascular responses are related to the mass of the contracting muscle due to more activated motor units. Therefore, it appears that both the tension developed and the muscle mass influence the cardiovascular responses to isometric exercise.

2.6.1 Tension Developed (% MVC)

Alam and Smirk (1937) examined the systolic blood pressure response to exercise at different intensities. The results showed that the magnitude of systolic blood pressure response is related to the tension developed. Also, the level of systolic blood pressure following the contraction, when the circulation was arrested, was also related to the relative tension developed. Lind & McNicol (1967) investigated the cardiovascular responses to more than one muscle group contracted at the same intensity and then contracted at different intensities. The results provided evidence to warrant the conclusion that the cardiovascular responses to isometric exercise are dependent on the tension developed and are independent of the muscle mass. Donald et al. (1967) indicated that the magnitude of cardiovascular responses depends on both the duration of contraction and the tension developed. Also, Funderburk et al. (1974) examined the effect of cardiovascular responses to handgrip contractions at 20, 40, and 60% MVC to the point of fatigue. Their results showed that the mean blood pressure response to isometric exercise is not related to the tension developed, but that heart rate response to isometric exercise is related to the tension developed. In contrast, Davies & Starkie (1985) stated that the blood pressure response to static contraction, unlike heart rate, is proportional to the tension developed but independent of the muscle mass. Rusch et al. (1981) suggested that the additional muscle masses may not elicit proportional increase in heart rate response. Thus, different control mechanisms would contribute to the mediation of blood pressure and heart rate

(Asmussen, 1981; Misner et al., 1990).

2.6.2 Muscle Mass

Mitchell et al. (1981) investigated the cardiovascular responses to 40% MVC sustained static contractions with four different muscle movements (finger abduction, handgrip, leg extension and combination of handgrip and leg extension) in young men. Evidence presented by Mitchell et al. (1981) indicated that the cardiovascular responses to the isometric exercise at the same % MVC level are muscle mass dependent due to more activated motor units. Also, the magnitude of blood pressure following the isometric exercise, during the circulatory arrest, was directly related to the muscle mass. Seals et al. (1983) examined the cardiovascular responses to 30% MVC of three different muscle movements (handgrip, both leg extension and "dead lift") in young males. The results indicated that the cardiovascular responses to the isometric exercise at the same % MVC level are directly dependent on the active muscle mass. Lewis et al. (1985) reported, in agreement with Mitchell et al. (1980) that the cardiovascular responses to a static exercise are dependent on the active muscle mass (hand finger flexors and both leg extensors). Misner et al. (1990) studied the cardiovascular responses to maximal voluntary sustained isometric contraction using three different muscle groups (right hand finger flexors, right leg extensors and both leg extensors) in young men. The results showed that the cardiovascular responses to isometric exercise were related to the muscle mass.

2.6.3 Muscle Fibre Type

Since most muscles contain both slow and fast twitch fibres, the sustained contraction is partially dependent on the muscle fibre type within the muscle. Edgerton et al. (1979) concluded that there is a progressive predominance from slow to fast twitch fibre recruitment during force increases. Asmussen (1981) indicated that the critical value of % MVC, below which sustained contraction can be maintained, varies

between 10 and 25% MVC. As an example, the tension limit lies at about 10% MVC in the elbow extensor consisting mainly of fast twitch fibres (fatiguable, glycolytic). But, in the elbow flexor where there are very few fast twitch fibres, the tension limit is much greater at 25% MVC. He concluded that during a sustained contraction, fast twitch fibres are vulnerable to fatigue as compared to slow twitch fibres (less fatiguable, oxidative). Petrofsky et al. (1981) examined the pressor response during isometric contraction from 10 to 100% MVC in cats. The results of study showed that the initial recruitment of slow twitch fibres showed a low pressor response in comparison to the initial recruitment of fast twitch fibres. They concluded that muscle fibre type is important in determining the pressor response at low levels of isometric contractions. Mitchell et al. (1983) indicated that a pressor response is observed in fast twitch but not in slow twitch fibres. They suggested that the difference in BP response to induced sustained contractions of slow and fast twitch fibres may be due to differences in blood flow, metabolism, or the distribution and sensitivity of Group III and IV afferent nerve endings between the slow and fast twitch fibres.

2.6.4 Arm versus Leg during Different Types of Exercise

Miles et al. (1989) indicated that the blood pressure is higher during arm-crank exercise than observed during leg-cycle exercise for a given oxygen uptake. Since the cardiac output was similar for arm and leg exercises for a given oxygen uptake, the greater total peripheral resistance is a possible explanation for the higher blood pressure response observed during arm-crank exercise (Astrand et al., 1965; Miles et al., 1989). On the basis of the experiments of Blomqvist et al. (1981), Lewis et al. (1985), and Miles et al. (1989) indicated that there is a gradual transition from a " dynamic " to a " static " hemodynamic response pattern as the size of the active muscle mass decreases. The results indicated that, in human, the size of the active muscle mass rather than the mode of contraction (static versus dynamic) is the primary

determinant of the hemodynamic responses. Conversely, Perez-Gonzalez (1981) suggested that the mode of exercise may be the primary determinant of the hemodynamic response. Asmussen (1981) concluded that the blood pressure is higher during static exercise than that during dynamic exercise for the same oxygen uptake which is probably due to different local, chemical, thermal, and mechanical factors. Shepherd et al. (1981) summarized whether the same local, chemical, thermal, and mechanical factors are operative, but at different levels. Thus, these two types of exercise lead to different hemodynamic responses. However, the higher pressor response associated with static exercise has been identified as the major hemodynamic distinction between static and dynamic exercise (Mitchell & Blomqvist, 1979). In the case of isometric exercise, Mitchell et al. (1980) found that blood pressure is higher during leg extension than that during handgrip at a given % MVC level which is due to a greater number of motor units being activated during leg extension. They suggested that both central and reflex drives may be increased when more motor units are activated. In contrast, Lind and McNicol (1967) found similar blood pressure responses during leg extension and handgrip at a given % MVC level.

2.6.5 Age-Related Decline in Muscle Strength

Normal aging is accompanied by a decline in muscle strength, but the cause of this decline has yet to fully elucidated. It has been attributed to several factors including declining muscle mass (Pearson et al., 1985), increasing muscular fibrous tissue (MacLennan et al., 1980), changing muscle fibre type (Larsson et al., 1979), and loss of motor units (Grimby & Saltin, 1983). Pearson et al. (1985) found that the muscle strength is significantly negatively correlated with age and the muscle mass is also significantly negatively correlated with age. They indicated that the decline in muscle strength is due to the loss of muscle mass with aging. In contrast, MacLennan et al. (1980) suggested that a decrease in muscle strength may not be related only to the

loss of muscle mass but would be related to increasing muscular fibrous tissue. Larsson et al. (1979) examined histochemical changes with age in human muscle tissue. The results indicated that the decline in muscle strength with aging is not only accompanied by the loss of muscle mass, but also accompanied by a decline in type II (fast-twitch) muscle fibres with a corresponding increase in type I (slow-twitch) muscle fibres. However, the changes in muscle fibre type with aging is still controversial. These authors stated that a net loss of muscle mass is replaced by fat and connective tissue. Recent experiments using human skeletal muscle have shown that muscle weakness associated with aging is not only due to muscle atrophy but also due to a reduction in the force per cross-sectional area (Bruce et al., 1989). Grimby and Saltin (1983) and Kallman et al. (1990) similarly suggested that the decline in muscle mass is associated with the loss of motor units, which is also related to the decline in muscle strength. Kallman et al. (1990) showed by regression analysis that the decline in muscle strength accelerates after the age of 40. However, Larsson et al. (1979) indicated that strength loss is primarily observed after the age of 50. Petrofsky and Lind (1975) reported that the decline in muscle strength accelerates after the age of 60. However, there is much individual variations with respect to the decline in strength observed with age (Kallman et al.,1990). It thus appears that age-related decline in muscle strength is mainly explained by a reduction in muscle mass.

2.6.6 Age-Related Decline in Muscle Mass

Numerous studies showed that normal aging is accompanied by progressively decreasing muscle mass (Grimby and Saltin, 1983; Pearson et al., 1985; Kallman et al., 1990). There is no direct measurement for estimating muscle mass (Fleg & Lakatta, 1988). Even though the cause of the muscle atrophy associated with aging is also unclear, it has been attributed to a decrease in the number of muscle fibres (Grimby and Saltin, 1983) and a decrease in fibre size (Essén-Gustavsson and Borges, 1986).

Grimby and Saltin (1983) concluded that the decline in muscle mass with aging is closely related to a decreased number of muscle fibres without a change in muscle fibre size. By comparison, Essén-Gustavsson and Borges (1986) demonstrated that a reduction in fibre size is the most noticeable change with aging. The results indicated that the reduced fibre size with aging may in part explain the decline in muscle mass and muscle strength. In addition, Bruce et al. (1989) suggested that the decline in muscle mass is more likely to affect quadriceps than other muscle groups. Pearson et al. (1985) examined the muscle strength in different muscle groups. They indicated that the changes in quadriceps mass is more closely associated with strength loss than other muscles. By comparison, Kallman et al. (1990) indicated that decreasing forearm muscle mass is highly correlated with declining handgrip strength. However, although fat-free mass is reduced by aging (MacLennan et al., 1980), due to either the decline in the number of muscle fibres or in fibre size, the external changes in muscle mass may not be reflected due to replacement by fat and connective tissue or fibrous tissue, for which there is also wide individual variation.

Therefore, both the tension developed and the muscle mass could make a significant contribution to the exercise pressor reflex. Studies confirm that they can both influence blood pressure and heart rate responses to isometric exercise, even though the mechanisms involved in blood pressure and heart rate responses to isometric exercise are different.

2.7 Baroreflex Modulation

This baroreflex involves impulses arising from baroreceptors in the carotid sinus and aortic arch areas (Greene and Bachand, 1971) or cardiopulmonary baroreceptors (Abboud et al., 1981). Shepherd et al. (1981) stated that the baroreflex plays an important role in the control of blood pressure by regulating input to the autonomic nervous system. Abboud et al. (1981) indicated that the regulation is not simply an

additive effect but rather an interaction leading to a change in the gain of Group III and IV afferents by the input from arterial baroreceptors during isometric exercise. Thus, as concluded by Shepherd et al. (1981), the increased activity of baroreflex inhibits cardiovascular reflex elicited by the stimulation of Group III and IV afferents from contracting muscles during isometric exercise. In addition, it inhibits sympathetic outflow when stimulated by increases in systemic arterial pressure. Asmussen (1981) showed that the baroreflex causes a sudden lowering of heart rate when the blood pressure rises suddenly. In contrast, Freund et al. (1978) suggested that the changes in heart rate may not be taken as the only index of baroreflex activity, particularly as inhibiting cardiovascular reflex drive. However, studies assessing baroreflex modulation of blood pressure during isometric exercise have presented conflicting results indicating normal (Greene and Bachand, 1971; Eckberg and Wallin, 1987) versus inhibited response (Cunningham et al., 1972) during isometric exercise.

2.7.1 Normal Versus Inhibited Response

Several investigators have demonstrated that the ability of the baroreflex to modulate blood pressure response is not inhibited by isometric exercise. Conversely, Cunningham et al. (1972) showed that the effectiveness of the baroreflex to modulate blood pressure response is diminished by isometric exercise. Greene and Bachand (1971) indicated that the elevation of blood pressure is normally associated with the baroreflex slowing of the heart rate, at least in part, by increasing vagal tone, which inevitably reduces blood pressure. Abboud et al. (1981) investigated the effect of baroreflex to modulate blood pressure and heart rate responses during handgrip at 10 and 20 %MVC. The results of their study indicated that heart rate and vasoconstriction is decreased by baroreflex to depress the activation of Group III and IV afferents during isometric exercise. They also showed increased activation of Group III and IV afferents during low carotid sinus pressure, after vagotomy (which removed the input from

cardiopulmonary baroreceptors), and during lower body negative pressure (LBNP is thought to reduce mainly the central sympathoinhibitory influence of cardiopulmonary baroreceptors). This was attributed primarily to a decreased baroreflex activity during isometric exercise. In contrast, Cunningham et al. (1972) examined the baroreceptor sensitivity to three different types of exercise in young subjects. They indicated that the capability of the baroreflex modulation is reduced during sustained isometric exercise, due to a decrease in baroreceptor sensitivity. Thus, it appears that there is a decrease in the baroreflex buffering of muscle sympathetic nerve activity (MSNA) during isometric exercise. However, further research by Eckberg and Wallin (1987) investigated the effect of isometric exercise on MSNA to alterations of baroreflex input. The results showed that handgrip significantly shortened the average R-R intervals but did not significantly change the average MSNA. They indicated that at the onset of isometric handgrip contraction rather small but directionally opposite effects on carotid baroreflex control of HR and MSNA and that the baroreflex to modulate blood pressure response is preserved during isometric exercise.

2.7.2 Age-Related Changes in Baroreflex

Numerous studies have clearly showed a progressive age-related reduction in baroreflex-mediated heart rate response to physiological stress (Shimada et al., 1985; Hainsworth and Al-shamma,1988). Shimada et al. (1985) investigated baroreceptor sensitivity as measured by the change in R-R intervals per unit change in systolic blood pressure during the Valsalva maneuver. They found that the baroreceptor sensitivity is negatively related to age and suggested that normal aging may be associated with progressive physiological changes in the control of blood pressure. Hainsworth and Al-shamma (1988) examined the cardiovascular responses to stimulation of carotid baroreceptors (neck suction) in four different age groups (G1: 19-35yrs; G2: 36-50yrs; G3: 51-66yrs; G4: 66-80yrs). The results showed that a reduction

in the response to stimulation occurred mainly between G1, G2, and G3, whereas the major change in blood pressure occurred only in G4 associated with little further decrease in the response. They suggested that abnormal baroreflex function is associated with normal aging and that the relationship between a decrease in the baroreceptor sensitivity and an increase in blood pressure is complex.

Therefore, these studies have provided evidence that the baroreflex activity is not inhibited and has a modifying effect on blood pressure response to isometric exercise. The progressive physiological reduction in the ability of baroreflex activity to modulate blood pressure response to isometric exercise is associated with normal aging, which in turn, affects the modifying effect of blood pressure response to isometric exercise.

2.8 Hemodynamic Response

Shepherd et al. (1981) concluded that isometric exercise causes an increase in the activity of the sympathetic nervous system and a decrease in the activity of the parasympathetic (vagal tone) nervous system. During isometric contraction, a marked increase occurs in diastolic and systolic blood pressures with a small increases in cardiac output, heart rate, and / or no change in total peripheral resistance and stroke volume (Lind et al., 1964; Asmussen, 1981; Mitchell et al., 1983). Donald et al. (1967) indicated that the rise in blood pressure is proportional to the product of the cardiac output and the total peripheral resistance. Thus, the exercise-induced elevation in blood pressure during isometric exercise reflects an increase in cardiac output and total peripheral resistance (Shepherd et al., 1981). Asmussen (1981) indicated that the increased heart rate and the increased blood pressure have different behaviours during isometric exercise. The heart rate increases immediately at the start of exercise followed by a slightly further increase throughout the whole exercise period, whereas the blood pressure increases gradually from the start throughout the whole exercise period. In contrast, Saltin et al. (1981) indicated that the pattern for the heart rate and

blood pressure elevation during isometric exercise is similar. However, isometric exercise is thought of as primarily producing a pressure load on the cardiovascular system (Mitchell and Blomqvist, 1979). This pressure load is more obvious in older subjects than in younger subjects at rest and during isometric exercise. In fact, a lower resting heart rate and a limited cardiac acceleration are observed for the older subjects at the same % MVC, which would be due to differences in vagal or sympathetic tone with aging (Van Loan et al., 1989). Thus, it appears that the higher BP response in older subjects during isometric exercise has a functional role increasing blood flow to the contracting muscles.

2.8.1 Autonomic Nervous System Control

The autonomic nervous system is responsible for the regulation of sympathetic and parasympathetic efferent nerve activity to the heart and blood vessels in order to match the hemodynamic response to the intensity of the isometric exercise (Mitchell, 1985). Goldstraw & Warren (1985) examined the function of the autonomic nervous system using different intensities of isometric exercise. They indicated that the exercise pressor reflex originates in the active muscles which is then transmitted by the muscle afferents to the autonomic nervous system and the efferent system is controlled by the activity of sympathetic and parasympathetic efferent nerve fibres. Martin et al. (1974) investigated autonomic nervous system control in the hemodynamic response to sustained isometric exercise by using selective autonomic blockade with intravenous propranolol (sympathetic blockade), atropine (parasympathetic blockade), and combined atropine-propranolol in young men. The results indicated that the initial increase in heart rate is absent with the administration of atropine, even though the later rise in heart rate still occurs. But, the typical heart rate response to isometric exercise is lower with the administration of propranolol. After combined atropine-propranolol is administered, the heart rate response to isometric exercise is

completely inhibited throughout the entire period of exercise. They concluded that the vagal withdrawal sets the initial pattern of autonomic activation to the heart and the sympathetic outflow modulates the following pattern of autonomic activation to the heart during sustained isometric exercise. Likewise, Freyschuss (1970) examined the autonomic nervous system in cardiovascular responses to static exercise by using selected autonomic blockade with atropine or phentolamine (alpha-receptor blockade) in young males. The results showed that heart rate acceleration is inhibited by atropinization whereas blood pressure elevation is maintained, and with phentolamine heart rate rise is maintained however the blood pressure increase is reduced. Both heart rate and blood pressure are markedly diminished by the combined dose. He concluded that initial heart rate acceleration to static exercise is due to vagal withdrawal while the marked inhibition of the blood pressure response would be attributed both to a heart rate acceleration and to an effect on peripheral vascular resistance mediated by the sympathetic nervous system. Conversely, Victor et al. (1989) investigated effects of partial neuromuscular blockade (curare) on autonomic nervous responses to static exercise and autonomic blockade (propranolol, atropine and combined atropine-propranolol) to evaluate the central command governing autonomic nervous control. The results indicated that the heart rate response to static exercise during curare is greatly decreased by atropine but is not significantly influenced by propranolol. They suggested that the central command governs vagally mediated rise in heart rate at all levels of static exercise. Mitchell et al. (1989a) examined autonomic blockade and cardiovascular responses to static exercise in partially curarized man. The results showed that the BP response to exercise with curarine was unaffected by atropine but the HR increase was reduced. However, propranolol reduced resting HR with curarine but with no effect on BP. In contrast, phentolamine reduced resting BP with curarine without affecting HR. They suggested

that the centrally generated HR response is in part caused by vagal withdrawal. However, the BP response cannot be attenuated by the sole use of alpha- or beta-receptor adrenergic blockade or combinations of these with atropine due to the redundancy in the autonomic control of BP. In addition, Perez-Gonzalez et al. (1981) examined the autonomic nervous system control in hemodynamic response to sustained isometric exercise with administration of propranolol. The results indicated that heart rate is deprived of an increase but blood pressure increase is maintained during isometric exercise at 30% MVC for three minutes following administration of propranolol. These authors concluded that the deprivation of the heart rate responses to isometric exercise is because it is mediated by beta-adrenergic stimulation and the preservation of the pressor response is maintained because it is mediated by alpha-adrenergic stimulation.

2.8.2 Influence of Age on Blood Pressure Control

The evaluation of the effect of age on beta- and alpha-adrenergic receptor responsiveness was studied by the infusion of adrenergic agonist or antagonist agents. Several studies have demonstrated a progressive decline in beta-adrenergic receptors mediated vasodilation and preservation of alpha-adrenergic receptors mediated vasoconstriction with aging (Van et al., 1981; Pan et al., 1986). Van et al. (1981) stated that the beta-adrenergic receptor is an important element in the autonomic control of the cardiovascular system. Thus stimulation of cardiac beta-adrenergic receptors increases heart rate and myocardial contractility and stimulation of vascular beta-adrenergic receptors gives rise to vasodilation. Pan et al. (1986) investigated alpha- and beta-adrenergic receptor mediated vascular responsiveness in males by using phenylephrine (alpha-selective agonist) and isoproterenol (beta-adrenergic agonist). The results indicated no differences in response to phenylephrine with increasing age but significant differences for isoproterenol. They stated that aging

is associated with blunting of beta-adrenergic receptor mediated vasodilation but preservation of alpha-adrenergic receptor mediated vasoconstriction. A similar study also compared age-related changes in beta-adrenergic sensitivity by using isoproterenol (Hiremath et al., 1989). The results confirmed earlier investigation that normal human aging is associated with a decrease in beta-adrenergic sensitivity. Vestal et al. (1979) reported that aging is accompanied by a decrease in the sensitivity of the beta-adrenergic receptor for both agonist and antagonist. Further evidence is provided by Feldman et al. (1984) who investigated lymphocyte beta-receptor affinity for agonists in two different age groups. The results showed that lymphocyte beta-receptor affinity for agonist is negatively correlated with age but beta-receptor density does not change with age. They suggested that reduced beta-receptor affinity for agonist may explain the reduced beta-adrenergic sensitivity in old subjects. Mader and Davis (1989) examined the effect of age on acute regulation of beta-adrenergic response in mononuclear leukocytes. The results indicated that beta-adrenergic receptor density rises significantly in younger subjects but not in older subjects after standing, although both younger and older subjects have similar beta-adrenergic receptor density at rest. They stated that acute regulation of adrenergic sensitivity is impaired by increasing age but resting function is preserved. Lakatta et al. (1987) indicated that an increase in the stiffness of blood vessels is another major change in aging which also influences blood pressure response to stress. This change is due to a reduction in elastic tissue and increased amount of collagen, which probably result in increased blood pressure. Recent studies suggested that the gradual change in the left ventricular morphology (Weber, 1988; Jetté et al., 1991) associated with aging process also influence blood pressure regulation. Weber (1988) and Jetté et al. (1991) similarly indicated that the gradual anatomical restructuring occurring in the resistance blood vessels and in the left ventricular mass of older subjects is associated with an

increase in blood pressure. Therefore, the normal aging process is associated with progressive physiological changes in blood pressure regulation due to a decrease in the beta-adrenergic sensitivity and the compliance of vascular and left ventricular morphology.

2.8.3 Age-Related Changes in Sympathetic Outflow

Several studies have suggested that the autonomic drive is probably intact in normal aging, but beta-adrenergic sensitivity is impaired (Hiremath et al., 1989) which would result in a hyperadrenergic state by sympathetic compensatory mechanisms. Numerous studies have showed that plasma norepinephrine (NE) is a reliable index of sympathetic efferent nerve activity. However, the effects of aging on the sympathetic nervous system have so far been studied mainly by determining the plasma norepinephrine level. Ziegler et al. (1976) indicated that plasma norepinephrine is significantly correlated with age. They suggested that the higher increase in plasma norepinephrine levels in response to stress is probably due to increased secretion or decreased catabolism. Mader and Davis (1989) confirmed Ziegler's observation, that plasma norepinephrine levels are higher in the older versus younger groups. Veith et al. (1986) suggested that the age-related rise in plasma norepinephrine may reflect an increase in sympathetic efferent nerve activity, because plasma norepinephrine parallells sympathetic efferent nerve activity under a variety of hemodynamic stresses such as postural change, isometric exercise, and the cold pressor test. The results also supported Ziegler's suggestion, that plasma norepinephrine appearance is greater and plasma norepinephrine clearance is lower in the older subjects. These authors concluded that the higher plasma norepinephrine levels of older subjects are due primarily to an increase in appearance rate and secondarily to a decrease in clearance. They also suggested that the reduced plasma NE clearance rate with increasing age may impair beta-adrenergic receptor function.

Thus, the hemodynamic response to stressor is influenced by the autonomic nervous system which causes an increase in sympathetic and a decrease in parasympathetic efferent nerve activity. However, although sympathetic outflow is increased with aging, a decreased sympathetic sensitivity is associated with aging, particularly acute regulation of beta-adrenergic responsiveness. In addition, the compliance of the left ventricle and blood vessels is also reduced in normal aging. Therefore, aging is accompanied by progressive physiological changes in the control of the hemodynamic responses.

2.9 Compensatory Mechanisms in the Control of Blood Pressure

Several studies have investigated cardiac disease and hypertensive subjects (Kino et al., 1975; Jetté et al., 1990; Julius, 1990) and normal old subjects (Kino et al., 1975; Julius, 1990) as well as exercise trained subjects (Longhurst et al., 1981; Keul et al., 1981; Maiorano et al., 1989) using isometric exercise as a cardiovascular test. Although the pressor response is elicited by different compensatory mechanisms, the cardiovascular response is still commonly characterized by a marked increase in blood pressure during isometric exercise.

2.9.1 Cardiovascular Response to Isometric Exercise in Cardiac Disease and Hypertension

Kino et al. (1975) examined the cardiovascular response to maximal or near-maximal isometric exercise among old normal subjects (ON) and age-matched subjects with coronary artery disease (CAD) and hypertensive heart disease (HHD). The results of the study showed no difference in heart rate response at rest and during exercise for ON versus CAD and ON versus HHD. They concluded that even near-maximal isometric exercise does not seem to be an effective screening test for cardiac disease and hypertension. However, Abboud et al. (1981) indicated that the augmented pressor response in patients with cardiac disease and hypertension can be reflected

by isometric exercise. In addition, Jetté et al. (1990) investigated left ventricular function using combined exercise (handgrip during supine cycling) in myocardial infarct patients. Their results showed a higher blood pressure response during combined exercise, which was associated with a higher left ventricular end diastolic pressure (LVEDP). They stated that the normal hemodynamic response to isometric exercise was due to sympathetic stimulation which includes a major increase in heart rate and left ventricular contractility. However, for the diseased heart, the greater pressor response is due to peripheral vasoconstriction. Julius (1990) investigated the cardiovascular response in normotension and borderline hypertension. He concluded that greater cardiovascular response in borderline hypertensive subjects is due to an increased alpha-adrenergic sensitivity combined with a decreased beta-adrenergic sensitivity.

2.9.2 Effect of Age on Hemodynamic Response to Isometric Exercise

It is important to note that various investigations on the effect of age on the blood pressure response to isometric exercise have led to different results. Kino et al. (1975) examined the cardiovascular response to maximal or near-maximal isometric exercise between old normal subjects (ON; age, 54-78 yr) and young normal subjects (YN; age, 23-31 yr). The results of the study showed significantly lower heart rate response in ON at rest and during isometric exercise. They indicated that the aging process is associated with a decreased heart rate and a depressed contractility due to a decreased sympathetic tone. Petrofsky and Lind (1975) investigated the effect of age (4 groups; 20-29, 30-39, 40-49, and over-50 yr) on the cardiovascular response to isometric exercise. The results indicated that the rise in heart rate is higher in younger men and the rise in systolic blood pressure is significantly greater in the older men at rest and during all periods of 40% MVC handgrip contraction. They indicated that higher systolic blood pressure is due to a decreased compliance of the arterial tree,

whereas there is no difference in diastolic blood pressure due to the unaffected systemic vascular resistance. Baldwa et al. (1983) examined the cardiovascular response to static exercise in subjects above 50 yrs versus below 50 yrs. The results showed a much higher increase in systolic and diastolic blood pressures as well as a lower increase in heart rate during 30 %MVC handgrip contraction in the elder subjects. These authors indicated that, in agreement with Petrofsky and Lind (1975), the greater rise in blood pressure observed in older subjects is due to increase in total peripheral resistance (increased arterial stiffness) rather than the increase in cardiac output. Goldstraw and Warren (1985) investigated how increasing age (elder; mean age, 73 yr; younger; mean age, 30 yr) affects the cardiovascular responses to graded static exercise at 10, 20 and 30% MVC levels. The results showed that both systolic and diastolic blood pressures are significantly higher at rest and the differential is maintained during static exercise in older subjects. They concluded that increasing age does not influence the pattern of the cardiovascular response to static exercise, although the resting blood pressure is higher in older subjects than that in younger subjects. In addition, Van Loan et al. (1989) investigated the cardiovascular responses of older men (older: 50-71 yr; younger: 18-31 yr) using both large and small muscle groups and performing isometric contractions at different % MVC levels (15, 30, 45, and 60% MVC). The results showed a greater blood pressure response to isometric exercise using different muscle groups and performing at various % MVC levels in older males. They indicated that the differences between the two age groups are due to changes in the vascular system associated with aging. Conversely, McDermott et al. (1974) examined the cardiovascular response to one-third of isometric handgrip contraction in two different age groups (older; mean age, 46.8 yr; younger; mean age, 25.3 yr). The results indicated the changes in blood pressure and heart rate responses to isometric exercise are similar for both age groups in spite of a hyperadrenergic state

associated with aging. Sagiv et al. (1988) examined the effect of age on left ventricular function and hemodynamic responses to isometric exercise, using two muscle groups in young (23+/-3 yr) and old (67+/-4 yr) males. The results showed a similar blood pressure response at 30% isometric contraction for handgrip and deadlift in young and old males. They indicated that there was no significant age associated with left ventricular function and hemodynamic responses during isometric exercise in both young and old males. Ordway and Wekstein. (1979) investigated the effect of age (3 groups; 21-42, 65-70, and 73-91 yr) on the cardiovascular response to two % MVC levels (15 and 30% MVC). The data showed a decline in the diastolic blood pressure response as a factor of age. They stated that the reduced diastolic blood pressure response may only be a function of the reduced heart rate response with increasing age. However, although most of studies showed a significantly lower heart rate response to isometric exercise with older subjects in comparison to younger subjects. It appears that the effect of age on blood pressure response to isometric exercise has not been entirely defined.

2.9.3 Effect of Training on Hemodynamic Response to Isometric Exercise

Studies have been conducted to examine the hemodynamic response to isometric exercise in trained subjects with both dynamic and static exercises (Shepherd et al., 1981). Both exercise training induces cardiovascular adaptations including both structure and function (Longhurst et al., 1981; Keul et al., 1981; Mumford and Prakash, 1981). Longhurst et al. (1981) compared the effects of isometric and dynamic training on the hemodynamic response to isometric handgrip contraction. The results of this study showed that there were different hemodynamic response to static exercise between endurance runners and weight lifters. They concluded that dynamic training can alter both the absolute and relative left ventricular mass, and the cardiovascular response to isometric exercise. Maiorano et al. (1989) examined the blood pressure

response to isometric handgrip contraction at 30% MVC in dynamically trained males compared to untrained males. They indicated that blood pressure tends to be lower following regular dynamic exercise at rest and during isometric exercise. Keul et al. (1981) supported the conclusion that reduced heart rate and blood pressure during isometric exercise are associated with decreased sympathetic outflow following endurance training .

Normal aging is associated with decreased heart rate and depressed contractility as well as increased blood pressure due to a decreased sympathetic tone and a decreased elasticity of the vasculature. In patients with left ventricular dysfunction, the increase in blood pressure response is due primarily to an exaggerated sympathetically mediated vasoconstriction increasing the systemic vascular resistance. In patients with hypertension, the increase in blood pressure response is due to an increase in the sensitivity of alpha-adrenergic receptors mediated vasoconstriction. Trained subjects decrease the blood pressure response to isometric exercise due to decline in sympathetic outflow.

2.10 Summary

Although both the reflex and central control mechanisms have not yet been fully delineated, the relative importance of these two neural control mechanisms in determining the cardiovascular response to isometric exercise is related to the intensity of the exercise, the mass of muscle involved in the exercise, the type of muscle fibres recruited in the exercise, the number of motor units recruited, and the effectiveness of blood flow to meet the increased metabolic needs of the contracting muscle. However, there is a progressive decline in muscle strength and mass, progressive changes in muscle fibre type, progressive decrease in motor units, progressive reduction in the ability of the baroreflex and sympathetic tone, and a progressive decrease in blood vessel compliance, with normal human aging.

Therefore, further investigation is required to provide insight into age differences on the pressor response to isometric handgrip contractions with and without blood occlusion.

III

METHODOLOGY

3.1 Introduction

This study was designed to compare the pressor response between older and younger males to a maximal voluntary isometric handgrip contraction (MVC) and at 20, 40, 60% MVC with and without upper arm blood occlusion.

3.2 Subject Selection

Ten presumably healthy males between the ages of 20 to 29 years and ten between the ages of 50 to 59 years volunteered for the study. The subjects were selected on the basis of the following criteria:

1) did not suffer from a physical ailment which could affect their performance
2) were non-smokers
3) had a normal resting blood pressure and a normal blood pressure response to a dynamic exercise
4) provided a negative answer to all questions on the modified PAR-Q
5) did not take any medication that could alter their cardiovascular response
6) agreed to participate in all phases of the experiment

3.3 Apparatus

The following equipment was employed:

For Anthropometric Measurements:

1) K-E anthropometric tape
2) Lange callipers (Cambridge Scientific Industries, Cambridge, MD)
3) Scale

For Blood Pressure Response to the CAFT:

1) Stethoscope

2) Sphygmomanometer (Almedic Ltd, Montreal, Canada)

3) Ergometer steps

4) CAFT cassette tape

5) Cassette recorder

6) Timer or stopwatch

For Determination of MVC:

1) Air Pressure Gauge (H.O. Trerice Co., Detroit, Michigan) connected by surgical tubing to a rubber squeeze bulb

For Hemodynamic response to Isometric Tests:

1) Finapres 2300 Blood Pressure Monitor (Ohmeda; Louisville, CO) connected to an IBM computer (IBM 5151, IBM Co, Armonk, New York) and printer (IBM 5152, IBM Co, Armonk, New York)

For Blood Occlusion:

1) Sphygmomanometer (Almedic Ltd, Montreal, Canada)

3.4 Study Protocol

The experiments were conducted on two separate days and in a quiet room. At the preliminary testing session (day 1), the subjects were explained the nature of the testing and their involvement in the study. Subjects were then requested to complete a modified PAR-Q (Appendix A) (Jetté, Quenneville and Sidney, 1992) and to read and sign an informed consent form. The subject's height, weight, and skinfolds (biceps, triceps, subscapular, iliac crest, and medial calf) were measured (Jetté, 1983). From these measurements body mass index (BMI) and percentage of body fat were calculated. The subjects were then seated for at least 5 minutes. The resting BP was then determined in duplicate (by auscultation). If the resting BP met the selection criterion (SBP<140, DBP<90), the subjects then performed the modified Canadian Aerobic Fitness Test (CAFT) in order to screen out exercise hypertensive subjects (ie.

>45 mmHg from rest to stage A of the CAFT) (Jetté et al., 1991). The results of the last CAFT stage completed by the subjects were used to calculate predicted maximum oxygen consumption (Jetté et al., 1976). If the subjects showed a normal BP response to the CAFT, they then performed, after a 10 minute rest, maximal voluntary handgrip contraction (using the Trerice apparatus). The mean of the best two trials was utilized to establish handgrip MVC. Each maximal contraction lasted two to five seconds. A one minute rest was given between each contraction (Lind et al. 1964). Following these preliminary tests, the subjects were then instructed to familiarize themselves with the wearing the Finapres.

On day 2 of the experiment, the subjects were requested not to participate in physical exercise and not to either consume caffeine or alcohol prior to the testing. The testing session was conducted between 08:00 and 12:00 hours, at least forty-eight hours following the preliminary testing session. When the subjects arrived, they were seated for ten minutes. Resting and exercise systolic blood pressure, diastolic blood pressure, mean arterial pressure, and heart rate were measured using the Finapres BP Monitor (Hildebrandt et al., 1990). Handgrip contractions were performed at different % MVC levels using the Trerice pressure gauge and a rubber squeeze bulb. During the handgrip contractions, the subjects were instructed to avoid the Valsalva manoeuvre as much as possible. All handgrip contractions were performed in the sitting position, arm extended at the side. The experimental protocol was conducted as follows:

1): The subjects performed dominant handgrip contractions at 20%, 40%, and 60% MVC for 30 seconds each and at MVC for 12 seconds. The order of different % MVC levels and MVC was randomly presented for each subject. Each test was performed with a 5-minute rest period between each contraction.

2): After a 10-minute rest, the identical intensities were performed with blood occlusion by placing a sphygmomanometer cuff around the upper arm and inflated to

200 mmHg. A 10-minute rest period was given between each contraction.

3.5 Data Analysis

Descriptive analyses and analyses of variance comparing means for the two age groups were computed using the SAS program (SAS Institute Inc., 1985).

The independent variables in this study included two age groups (20-29 and 50-59 yrs), with and without blood occlusion, and at 20%, 40%, 60% MVC, and 100% MVC. The dependent variables included systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR) during isometric handgrip contractions. A 2x2x4 factorial design ANOVA with repeated measures was conducted on the mean values for the exercise cardiovascular responses was used to determine if there were any significant differences for BP and HR with respect to the two age groups at the different % MVC levels with and without blood occlusion of the isometric exercises. When a significant F was attained, post-hoc comparisons among means were presented, using Scheffé's methods. Results at the 0.05 level of probability were accepted as statistically significant.

Linear regression analysis was calculated for cardiovascular responses. The slope and intercept of cardiovascular responses to the various isometric contractions were calculated and were entered in a 2x2x4 (age group x blood flow condition x intensity) factorial design analysis of variance with repeated measures to determine if there were any significant differences among the means. An alpha level of 0.05 was accepted as a standard for statistical significance.

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Modified Physical Activity Readiness Questionnaire (PAR-Q)

Family Name:_____ Date:_____

Given Name:_____ Age:_____

1. Has a physician ever said you have heart trouble? Yes No

2. Do you frequently have pains in your heart and chest? Yes No
3. Do you often feel faint or have spells of severe dizziness? Yes No

4. Has a physician ever said that your blood pressure was too high? Yes No

5. Do you suffer from any respiratory tract problem such as chronic bronchitis, asthma or emphysema? Yes No

6. Have you ever had or are you now suffering from any nervous disorder? Yes No

7. Do you suffer from any bone or joint problem which either has been or may be irritated by an exercise session? Yes No

8. Do you know of a valid medical reason why you should not be involved in either a regular exercise program or an exercise testing session? Yes No

9. At present, are you taking medication for blood pressure? If yes, please specify: Yes No

   Reason:_____
   Name:_____
   Dosage:_____

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

    Reason:_____
    Name:_____
    Dosage:_____

Signature:_____

Date:_____

From: Jetté, M., Quenneville, J., Sidney, K., Fitness Testing and Counselling in Health Promotion. (1992).

University of Ottawa, Faculty of Health Science, School of Human Kinetics

Consent form for isometric exercise testing

I,_____ ,authorize Yun Lin (231-2810) of the School of Human Kinetics, University of Ottawa, to administer and conduct the following fitness test and handgrip contractions. This study will be conducted under the supervision of Dr. M. Jetté, School of Human Kinetics (564-9108).

The purpose of this study is to compare the pattern of blood pressure response between older and younger males measured continuously with and without blood occlusion during isometric handgrip contractions at different percentages of maximal voluntary handgrip contraction (% MVC).

I understand that I will perform the following tests on two separate days:

On day 1, I will complete the Physical Activity Readiness Questionnaire (PAR-Q). My resting blood pressure, resting heart rate, height, weight, and skinfolds will be measured. I will then step up and down on double 20.3 cm. steps for three stages of the Canadian Aerobic Fitness Test of an intensity determined by my age group. My blood pressure and heart rate will be measured before beginning the step test, during the 1 minute interval between each stage of the test and at the 3 minute recovery following the stepping exercise. If my heart rate and blood pressure does not exceed a predetermined limit, I will perform 3 consecutive maximal handgrip contractions (MVC) by squeezing as strong as possible on a rubber bulb with my dominant hand.

On day 2, I will perform a maximal dominant handgrip contraction for 12 seconds,

followed by 30 second contractions at 20%, 40%, and 60% my maximal voluntary contraction. My blood pressure and heart rate will be measured continuously during these contractions using a Finapres BP monitor. There will be a 5-minute rest period between each contraction. Following a 10 minute rest, I will then repeat these contractions with my blood flow occluded with a sphygmomanometer cuff wrapped around the upper arm for a period of 30 second from the onset of contraction. I will receive a 10-minute rest period between each contraction.

Every effort will be made to conduct the test in such a way as to minimize discomfort and risk. I may experience some light muscular fatigue following the contraction.

I further understand that it is my responsibility to inform the testing personnel of any injury, illness, infection, or other condition, which would prevent me from fully participating in this session. In agreeing to such an examination, I waive any legal recourse against the member of the staff of the Dept of Kin, University of Ottawa from any and all claims resulting from personal injuries sustained resulting from these tests. This waiver shall be binding upon my heirs, my executors, my administrators and my personal representatives.

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

DATE:_____ SUBJECT:_____

WITNESS:_____

Day One Test Sheet

Name:_____ Sex:_____ Age:_____ Date:_____

Height:_____cm. Weight:_____kg. RHR:_____beat/min.

RSBP:_____mmHg. RDBP:_____mmHg.

Skinfold Measurements

Biceps:_____ Triceps:_____

Subscapular:_____

Iliac Crest:_____ Medial Calf:_____

CAFT

HR:_____bpm HR:_____bpm HR:_____bpm

SBP:_____mmHg SBP:_____mmHg SBP:_____mmHg

DBP:_____mmHg DBP:_____mmHg DBP:_____mmHg

MVC

1st_____ 2nd_____ 3rd_____

Day Two Test Sheet

Name:_____ Sex:_____ Age:_____ Date:_____

Height:_____cm. Weight:_____kg. RHR:_____beat/min.

RSBP:_____mmHg. RDBP:_____mmHg.

Strength Testing:

Dominant Hand.

MVC(P):_____

20 %MVC(P):_____

40 %MVC(P):_____

60 %MVC(P):_____

Study Testing:

Dominant Hand.	C Time(Sec).	Sample intervals(Sec).	R Time(Min).
-Rest:_____	60	10	1
-MVC:_____	12	2	5
-20%MVC:_____	30	5	5
-40%MVC:_____	..	..	..
-60%MVC:_____	..	..	..

10 Min Rest

Blood Occlusion.	.. (Sec).	.. .. (Sec).	.. (Min).
-MVC:_____	12	2	10
-20%MVC:_____	30	5	10
-40%MVC:_____	..	..	..
-60%MVC:_____	..	..	..