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Hemodynamic responses following dynamic resistance exercise in young and older adult women

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HEMODYNAMIC RESPONSES FOLLOWING DYNAMIC RESISTANCE EXERCISE IN YOUNG AND OLDER ADULT WOMEN

A Dissertation

Submitted to the Graduate Faculty of the Louisiana State University and Agricultural and Mechanical College in partial fulfillment of the requirements for the degree of Doctor of Philosophy

in

The Department of Kinesiology

by

Rafael Reyes
B.S. University of The Andes, Venezuela, 1984
M.Sc. Barquisimeto Pedagogical University, Venezuela, 1992
May, 2004
DEDICATION

This dissertation is especially dedicated to my entire family whose support and encouragement made this endeavor possible. Special mention is to my wife, Thais, my daughter, Thairy, and my son, Rafael Andrés who were with me each minute throughout this effort. Thanks for your support and patient all these years.
ACKNOWLEDGEMENTS

I would like to especially acknowledge and say thanks to Dr. Robert Wood, my advisor, for the support and guidance he extended to me throughout these years, and for taking me through the window of intellectual independence and spiritual freedom. I was many years looking for them. God bless you and thank you.

My thanks are also extended to the members of the committee, Drs. Robert Wood, Richard Magill, Michael Welsch, Arnold Nelson, Li Li, and Jacqueline Stephens for their individual and group support and input in this project. It was a pleasure to work with you.

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TABLE OF CONTENTS

DEDICATION .................................................................................................................. ii

ACKNOWLEDGEMENTS ............................................................................................. iii

LIST OF TABLES .......................................................................................................... vi

LIST OF FIGURES ........................................................................................................ ix

ABSTRACT .................................................................................................................... xi

1.  INTRODUCTION .................................................................................................... 1

2.  MATERIAL AND METHODS ................................................................................ 7
    2.1. Participants and Study Design ........................................................................... 7
    2.2. Materials and Equipment .................................................................................. 8
    2.3. Procedure ......................................................................................................... 9
    2.4. Data Reduction and Analysis .......................................................................... 16
    2.5. Statistical Analysis ......................................................................................... 20

3.  RESULTS ............................................................................................................... 23
    3.1. Participant Characteristics ............................................................................. 23
    3.2. General Effect of the Resistance Exercise (Main Effect of Test Condition) ....... 26
    3.3. Moderating Effect of Resistance Exercise Condition (5-RM vs. 15-RM) ........... 32
    3.4. Moderating Effect of Age ............................................................................. 41
    3.5. Relationships Between Indicators of Autonomic and Vascular Function and Hemodynamic Responses During and Following RE ................. 60
    3.6. Summary of the Results .............................................................................. 67

4.  DISCUSSION ......................................................................................................... 69
    4.1. Participant Characteristics ............................................................................. 69
    4.2. The General Hemodynamic Responses During and Following RE ............... 70
    4.3. Moderating Effect of Resistance Exercise Condition (5-RM vs. 15-RM) ......... 77
    4.4. Moderating Effect of Age ............................................................................. 81
    4.5. Relationships Between Indicators of Autonomic and Vascular Function and Hemodynamic Responses During and Following RE ................. 88
    4.6. Limitations of the Study ............................................................................. 92
    4.7. Summary and Conclusions ........................................................................... 95

REFERENCES ............................................................................................................. 101

APPENDIX A. BACKGROUND AND SIGNIFICANCE ............................................. 114

APPENDIX B. EXTENDED RESULTS SECTION ..................................................... 140

iv
LIST OF TABLES

Table 2.1 Preliminary Assessments and Test Familiarization ...................................10
Table 2.2 Experimental Resistance Exercise Testing Protocol.............................14
Table 2.3 List of Hemodynamic Variables and Definitions ....................................17
Table 3.1 Participant’s Characteristics.................................................................24
Table 3.2 Pre-exercise Autonomic Indicators from 5-min segments......................24
Table 3.3 Pre-exercise Autonomic Indicators from 10-min segments......................24
Table 3.4 Resting and After Occlusion BP, HR, and Vascular Function Indices......25
Table 3.5 Pre-exercise BP, HR, and Vascular Function Indices..............................25
Table 3.6 General Effect of RE (Test Condition) on Arterial BP and HR
Before, During, & Immediately After RE in Women.........................................27
Table 3.7 General Effect of RE (Test Condition) on Arterial BP and HR
Before, During & For 1 Hour After RE in Women .........................................29
Table 3.8 General Effect of RE (Test Condition) on HRV Indices (5-minute
segment) Before and Immediately After RE in Women.....................................30
Table 3.9 General Effect of RE (Test Condition) on HRV Indices (10-minute
segment) Before and For 1 Hour After RE in Women .....................................31
Table 3.10 General Effect of RE (Test Condition) on Forearm Blood Inflow and
Vascular Resistance Before and For 1 Hour After RE in Women .....................32
Table 3.11 Exercise Condition Effect on Arterial Blood Pressure and Heart Rate
Before, During, & Immediately After RE in Women ..........................................35
Table 3.12 Exercise Condition Effect on Arterial BP and HR Before, During, &
Immediately After RE in Women.......................................................................37
Table 3.13 Exercise Condition Effect on HRV Indices (5-minute segment)
Before and Immediately After RE in Women ....................................................39
Table 3.14 Exercise Condition Effect on HRV Indices (10-minute segment)
Before and For 1 Hour After RE in Women.......................................................40
Table 3.15 Exercise Condition Effect on Forearm Blood Inflow and Vascular
Resistance Before and For 1 Hour After RE in Women ....................................41
Table 3.16  Age Group Effect on Arterial BP and HR Before, During, & Immediately After RE in Women

Table 3.17  Age Group Effect on Arterial Blood Pressure and Heart Rate Before, During, & For 1 Hour After RE in Women

Table 3.18  Age Group Effect on Forearm Blood Flow and Vascular Resistance Before and For 1 Hour After RE in Women

Table 3.19  Age Group by Test Condition Interaction Effect on HRV Indices (5-minute segment) Before and Immediately After RE in Women

Table 3.20  Age Group by Test Condition Interaction Effect on HRV Indices (10-minute segment) Before and For 1 Hour After RE in Women

Table 3.21  Age Group by Exercise Condition Interaction Effect on SBP Responses During and Immediately After RE in Women

Table 3.22  Age Group by Exercise Condition Interaction Effect on DBP Responses During and Immediately After RE in Women

Table 3.23  Age Group by Exercise Condition Interaction Effect on MAP Responses During and Immediately After RE in Women

Table 3.24  Age Group by Exercise Condition Interaction Effect on SBP Responses During, & For 1 Hour After RE in Women

Table 3.25  Age Group by Exercise Condition Interaction Effect on DBP Before, During, & For 1 Hour After RE in Women

Table 3.26  Age Group by Exercise Condition Interaction Effect on MAP Responses Before, During, & For 1 Hour After RE in Women

Table 3.27  Age Group by Exercise Condition by Tests Condition Interaction Effect on Forearm Vascular Resistance Before and For 1 Hour After RE in Women

Table 3.28  Bivariate Correlation Between Indicators of Autonomic Function & SBP Before and After RE Pooling Both Young and Older Group Data

Table 3.29  Bivariate Correlation Between Indicators of Autonomic Function & SBP Before and After RE in Older Women

Table 3.30  Bivariate Correlation Between Indicators of Autonomic Function & SBP Before and After RE in Young Women
Table 3.31  Bivariate Correlation Between Indicators of Autonomic Function & SBP Before and After RE by Exercise Condition .................................................62
Table 3.32  Relationship Between Indicators of Autonomic Function & Arterial BP Responses After RE Pooling Both Young and Older Group Data ......62
Table 3.33  Bivariate Correlation Between Vascular Function Indices & SBP Before and After RE Pooling Both Young and Older Adult Data.............63
Table 3.34  Bivariate Correlation Between Vascular Function Indices & SBP Before and After RE in Older Women ..............................................................63
Table 3.35  Bivariate Correlation Between Vascular Function Indices & SBP Before and After RE in Young Women..........................................................64
Table 3.36  Bivariate Correlation Between Vascular Function Indices & DBP Before and After RE Pooling Both Young and Older Adult Data.........64
Table 3.37  Bivariate Correlation Between Vascular Function Indices & DBP Before and After RE in Older Women ..........................................................64
Table 3.38  Bivariate Correlation Between Vascular Function Indices & DBP Before and After RE in Young Women..........................................................65
Table 3.39  Bivariate Correlation Between Indicators of Autonomic Function & Vascular Function Indices Pooling Both Young and Older Group Data........................................................................................................66
Table 3.40  Bivariate Correlation Between Indicators of Autonomic Function & Vascular Function Indices in Older Women.............................................66
Table 3.41  Bivariate Correlation Between Indicators of Autonomic Function & Vascular Function Indices in Young Women.............................................66
Table 3.42  Relationship Between Indicators of Autonomic (LFnu) & Vascular Function (FVR) After RE in Young and Older Women ......................67
Table 4.1  Pre-exercise Hemodynamic Variables Assessment .................................................94
LIST OF FIGURES

Figure 2.1 Tachogram of SBP with Smoothed Data During, and Immediately After Resistance Exercise in Women ........................................................18

Figure 3.1 General Effect of RE (Test Condition) on Arterial BP and HR Before, During & Immediately After Exercise in Women .......................26

Figure 3.2 General Effect of RE (Test Condition) on Arterial BP and HR Before, During, & For 1 Hour After Exercise in Women .......................28

Figure 3.3 General Effect of RE (Test Condition) on LFnu (5-minute segment) Before & Immediately After Exercise in Women ............................30

Figure 3.4 General Effect of RE (Test Condition) on LFnu (10-minute segment) Before & For 1 Hour After Exercise in Women .................................31

Figure 3.5 General Effect of RE (Test Condition) on Forearm Vascular Resistance Before & For 1 Hour After RE in Women .......................................32

Figure 3.6 Exercise Condition Effect on Arterial Blood Pressure and Heart Rate During & Immediately After RE in Women ........................................34

Figure 3.7 Exercise Condition Effect on Arterial Blood Pressure and Heart Rate Before, During & For 1 Hour After RE in Women ............................36

Figure 3.8 Exercise Condition Effect of LFnu (5-minute segment) Before & Immediately After Exercise in Women ..................................................38

Figure 3.9 Exercise Condition Effect of LFnu (10-minute segment) Before & For 1 Hour After Exercise in Women ..................................................39

Figure 3.10 Exercise Condition Effect of Forearm Vascular Resistance Before & for 1 Hour After RE in Women ......................................................40

Figure 3.11 Age Group Effect on Arterial Blood Pressure and Heart Rate Before, During & Immediately After RE in Women .........................................42

Figure 3.12 Age Group Effect on Arterial Blood Pressure and Heart Rate Before, During & For 1 Hour After RE in Women .........................................44

Figure 3.13 Age Group Effect on Forearm Vascular Resistance Before & For 1 Hour After RE in Women ............................................................47

Figure 3.14 Age Group by Test Condition Interaction Effect on LFnu (5-minute segment) Before & Immediately After Exercise in Women .................49

Figure 3.15 Age Group by Test Condition Interaction Effect on LFnu (10-minute segment) Before & For 1 Hour After Exercise in Women ..................50
Figure 3.16 Age Group by Exercise Condition Interaction Effect on SBP Responses During & Immediately Following RE in Women

Figure 3.17 Age Group by Exercise Condition Interaction Effect on DBP Responses During & Immediately Following RE in Women

Figure 3.18 Age Group by Exercise Condition Interaction Effect on MAP Responses During & Immediately Following RE in Women

Figure 3.19 Age Group by Exercise Condition Interaction Effect on SBP Responses During & For 1 Hour After RE in Women

Figure 3.20 Age Group by Exercise Condition Interaction Effect on DBP Responses During & For 1 Hour After RE in Women

Figure 3.21 Age Group by Exercise Condition Interaction Effect on MAP Responses Before, During & For 1 Hour After RE in Women

Figure 3.22 Age Group by Exercise Condition by Test Condition Interaction Effect on FVR Before & For 1 Hour After RE in Women

Figure 4.1 Relationship Between LFnu at Rec 0-5 and Mean Rate on SBP Recovery After RE in Young and Older Adult Women

Figure 4.2 Relationship Between Resting Forearm Venous Outflow and Magnitude of SBP Drop After RE in Young and Older Women

Figure 4.3 Relationship Between Forearm Venous Outflow After Arterial Occlusion and Magnitude of SBP Drop After RE in Older Women

Figure 4.4 Relationship Between LFnu at Rec 0-5 & Magnitude of MAP Recovery After RE in Young and Older Adult Women

Figure 4.5 Relationship Between LFnu at Rec 0-5 & Forearm Vascular Resistance After RE in Older Adult Women

Figure 4.6 Relationship Between LFnu at Rec 0-5 & Forearm Vascular Resistance After RE in Young Adult Women
ABSTRACT

PURPOSE: This investigation examined the effects of age, exercise and test condition on hemodynamic variables, autonomic and vascular function in relation to resistance exercise (RE). The associations among these variables were also examined. METHODS: Sixteen young (21.4±1.4 yrs) and 16 older (69.7±3.9 yrs) women performed 5- and 15-repetition maximal (RM) of knee extension RE. Continuous blood pressure (BP) and electrocardiography (ECG) data were recorded. The dependent variables are reported at pre-exercise, peak exercise and recovery period. Heart rate variability data were derived from 5- and 10-min segments before and after exercise. Resting and after arterial occlusion forearm vascular function indices and pre- and post-exercise resting forearm blood flow and forearm vascular resistance (FVR) were measured using plethysmography technique. ANOVA with repeated measures was used for statistical analysis. LSD was used where post hoc comparison required. Pearson correlation and linear regression were used to examine associations between autonomic and vascular function indices and hemodynamic parameters; α=0.05. RESULTS: Resistance exercise resulted in increased BP (↑SBP=36.6±2.2; ↑DBP=27.4±1.6 mmHg) and HR (↑37.8±1.6 beat/min). This was followed by a drop in BP below pre-exercise level throughout the recovery period up to 60 minutes. The 15-RM condition resulted in higher SBP at peak exercise (15-RM: 155.7±3.7 vs. 5-RM: 142.3±3.7 mmHg) and in the early phase of recovery (local min: 15-RM=127.0±2.7 vs. 5-RM=120.4±2.6 mmHg); however, the 5-RM condition resulted in greater 1’ and 3’ post-exercise SBP recovery ratios (15-RM: 1’=0.78±0.01; 3’=0.76±0.01 vs. 5-RM: 1’=0.84±0.01; 3’=0.82±0.01). Older women had higher SBP throughout the testing period, and higher 1’ and 3’ recovery ratios (Young: 1’=0.80±0.01;
3\'=0.78\pm0.01 vs. Old: 1\'=0.83\pm0.01; 3\'=0.80\pm0.01). However, the older women experienced greater drops in BP (SBP: young=-0.02\pm2.6 vs. old=-9.4\pm2.3; DBP: young=-3.5\pm1.8 vs. old=-9.8\pm1.9 mmHg) during the recovery period. FVR after RE increased above pre-exercise only in the young (p<0.03). Low-frequency variations in HR were related to recovery of mean arterial pressure (young: r=0.66, p<0.001; older: r=0.79; p<0.001) and FVR (young: r=0.93, p=0.001; old: r=0.95; p<0.001).

CONCLUSION: Age-group differences in post-exercise BP drop, characterized by a greater decline in BP in older adults, might be attributed to smaller increases in vascular resistance in older women.
1. INTRODUCTION

The sudden or prolonged reduction of systolic and/or diastolic blood pressure (BP) below pre-exercise level (e.g., pre-exercise resting-controlled conditions) after exercise\textsuperscript{1-26} is linked to increased risk of abnormal cardiovascular events following exercise\textsuperscript{29-31, 120-124}. Furthermore, a delay in the post-exercise BP recovery is implicated as a risk factor for the development and progression of cardiovascular disease\textsuperscript{30-34, 64, 66-67, 76-77}. Interestingly, however, recent works suggest that the reduction of BP following exercise may be of significant clinical value in the non-pharmacological treatment of hypertension\textsuperscript{24-26}.

The reduction of BP below pre-exercise level after dynamic aerobic exercise is confirmed in normotensive\textsuperscript{6-7} and hypertensive individuals,\textsuperscript{1-4} as well as in several animal models (e.g., spontaneously hypertensive rats, and electrical stimulation of somatic and muscle afferents -post-stimulation hypotension).\textsuperscript{20, 24, 26} The occurrence of arterial BP drops below pre-exercise level after aerobic exercise is independent of exercise intensity,\textsuperscript{1-6, 8-10, 12, 14, 18-21} exercise duration,\textsuperscript{1-6, 9-12, 14, 18-23} exercising muscle mass,\textsuperscript{35} body recovery posture,\textsuperscript{36-37} and thermal conditions.\textsuperscript{39} In contrast, the magnitude and duration of arterial BP drops below pre-exercise level and subsequent to aerobic exercise may be influenced by exercise intensity,\textsuperscript{1, 40} exercise duration,\textsuperscript{20} exercising muscle mass,\textsuperscript{35} and thermal conditions,\textsuperscript{39} as well as the previous health history and age of the participant.\textsuperscript{1-4, 6, 12, 15, 38, 41-43} The magnitude and duration of arterial BP drops below pre-exercise level can be defined as the difference of post-exercise arterial BP minus pre-exercise arterial BP and the time during recovery in which arterial BP remains below pre-exercise arterial BP, respectively.
The mechanisms underlying post-exercise BP drops below pre-exercise level following aerobic exercise are not completely understood. Neural (e.g., sympatho inhibition) \(^{24-26, 46-48}\) and local (e.g., impairment of vascular responsiveness to \(\alpha\)-adrenergic receptor stimulation, and/or vasodilator substances) \(^{24-26, 46, 49-51}\) alterations in sympathetic vascular regulation are suggested as potentially mediating a sustained decrease in regional and systemic vascular resistance.

In contrast to aerobic exercise, information concerning the arterial BP response following dynamic resistance exercise (RE) in humans is scant and inconclusive \(^{52-57}\). While a few studies indicate the occurrence of arterial BP drops below pre-exercise level after an acute bout of RE (~10 second immediately after exercise \(^{52}\) or after 10-minute following exercise \(^{55-56}\)), other studies involving similar testing procedures reveal conflicting findings \(^{53-54, 57}\). The disparate findings may be reconciled as a consequence of a lack of standardization of BP monitoring, which in these studies, primarily involved intermittent auscultatory techniques, \(^{52-54, 56-57}\) often starting as late as 1 minute into the recovery period \(^{52-54, 56-57}\).

Of additional interest is the fact that this line of inquiry has been limited to healthy younger adults (age range=18-33 yrs) \(^{52-57}\). Although age-related changes in the cardiovascular system (e.g., increases in vascular resistance) and differences in post-exercise BP drops below pre-exercise level after aerobic exercise have been revealed, to date no study has examined post-exercise hemodynamic response after RE in older adults. Inasmuch as the guidelines for exercise prescription for older adults recommend including RE as part of a well-rounded physical fitness program for this population, \(^{65, 73}\)
further investigation of the hemodynamic responses to such activity is necessary, as they may have implications for risk, and therefore influence exercise prescription.

The potential implications of post-exercise arterial BP drops below pre-exercise level with respect to the risks and benefits of exercise, particularly in older adults, have been of interest to our laboratory. In pursuing this line of inquiry we executed two pilot studies. First, we examined the feasibility and reliability of using continuous arterial tonometry as a means of measuring BP prior to, during, and following incremental dynamic RE (i.e., 20, 30, 40, 50, 60, 70, 80, and 90% of 5-repetition maximal) in younger adults. The data from this initial investigation revealed a reliable BP signal during all conditions except during RE at 90% of 5-RM (repetition maximal). Moreover, the data revealed a significant main effect of exercise condition on post-exercise arterial BP drops ($p = 0.03$), with exercise with lower repetitions (i.e., low volume) and greater intensities (i.e., high workload) being associated with post-exercise pressures dropping further below pre-exercise conditions.

In the second pilot study we examined the BP responses of older adults at 20, 40, 60, 80% of 5-RM. Once again, the findings suggested an exercise condition dependent influence on post-exercise blood pressures ($p = 0.0001$). However, the designs of both pilot-studies only provided weak inferences with regard to the influence of exercise condition on post-exercise BP drops below pre-exercise level. These weakness included the (1) the existence of a possible order effect of the consecutive incremental protocols, (2) the limited recordings of post-exercise BP readings following RE (hemodynamic responses only observed throughout 4-min recovery periods following each increment of work), and (3) an invoked number of repetition maximum (i.e., 12-, and 15-repetitions for
the pilot studies with younger and older adults, respectively) resulting in exercise failure at high intensities. Moreover, differences in protocols between the two studies make age-group comparison problematic.

Therefore, the main purpose of this investigation was to examine the acute hemodynamic responses with an emphasis on continuous systolic and diastolic BP and HR before, during, immediately after, and for 1 hour after a single bout of 5-RM and 15-RM (5- and 15-repetition maximal, respectively) bilateral knee-extension RE in healthy young and older adult women. In particular, this study examined the effects of test condition (general effect of RE), exercise condition (5-RM, and 15-RM), and age group (younger and older adults), as well the age x exercise condition interaction on the *Magnitude, and Rate of post-exercise BP drop below pre-exercise level* following RE.

A secondary objective of this investigation was to examine indicators of autonomic (i.e., heart rate variability profiles [SDNN, pnn50, LFnu, and LF/HF ratio]) and vascular function (i.e., forearm blood flow [FBF], and forearm vascular resistance {FVR}) before and after a single bout of 5-RM and 15-RM bilateral knee-extension RE in healthy young and older adult women. In particular, this study examined the effects of test condition (general effect of RE), exercise condition (5-RM and 15-RM), and age group (young and older adults), as well as the age x exercise condition interaction on indicators of autonomic and vascular function following RE.

Furthermore, considering the available evidence in literature indicating age- and fitness-related changes in autonomic nervous system (ANS) balance and vascular function, the final objective of this study was to describe associations between indicators of autonomic (i.e., SDNN, pnn50, LFnu, and LF/HF ratio) and vascular function (i.e.,
forearm blood inflow [FBF], forearm vascular resistance [FVR], forearm venous capacitance [FVC], forearm venous outflow [FVO]) and pattern of post-exercise hemodynamic parameters (i.e., magnitude and rate of arterial BP change after RE).

With respect to characterizing the *Magnitude of post-exercise BP changes*, the parameters of interest involved pre-exercise BP, peak exercise BP, local minimum BP, magnitude of BP drop from peak to local minimum, and local minimum minus pre-exercise BP. Moreover, 1-minute, and 3-minute post-exercise BP ratios (i.e., expressed relative to peak BP) are reported inasmuch as these parameters appear to have predictive validity with respect to risk of cardiovascular diseases.  

With respect to *Rate of Post-exercise BP drops*, the parameters of interest included time from peak exercise BP to local minimum and mean rate of BP drop. While these are primary variables of interest, we also report other interesting phenomena, which include *Duration of Post-exercise BP Drop* below pre-exercise level and *Post-exercise BP Recovery Overshoot*. With respect to *Duration of Post-exercise BP Drop* below pre-exercise level, we report absolute BP values at 1’, 3’, 5’, 10’, 15’, 20’, 30’, 40’, 50’, and 60’ following RE. Lastly, the pilot studies reveal a consistent local maximum BP following RE. This suggests the existence of a *Post-exercise BP Recovery Overshoot* phenomenon. While this has not been previously described, its consistent appearance in our pilot studies suggests that it is indeed a physiologic event. We therefore report *Post-exercise BP recovery Overshoot* as the difference between local max and local min that immediately before it, the time from local min to local max, and the mean rate of recovery (difference/time). The observations for these hemodynamic parameters are compared against indicators of autonomic (i.e., R-
R interval, SDNN, pnn50, LFnu, and LF/HF ratio) and vascular function (i.e., FBF, FVR, FVC, and FVO).

Data from our pilot studies suggest a significant main effect of exercise condition on the magnitude of post-exercise BP drop below pre-exercise level such that lower RM (i.e., high intensity/low volume) exercise condition was associated with larger magnitude of BP drop below pre-exercise level after RE. With respect to the other parameters of interest (i.e., rate, duration, and BP recovery overshoot), there are no published studies upon which to base a hypothesis in this regard. Similarly, the questions as to the potential to observe significant main effects of age or an appearance of a significant age x exercise condition interaction on any of the parameters of post-exercise BP drop below pre-exercise are also empirical. However, due to the influence of age on a variety of cardiovascular functional characteristics, an influence of age on post-exercise BP drop is certainly not unexpected.

More specifically, it was hypothesized that post-exercise BP drops below pre-exercise level after RE will be directly associated with: (1) the exercise condition, and (2) resting autonomic and vascular function indices, and that post-exercise BP drops below pre-exercise level after RE will differ between young and old women.
2. MATERIAL AND METHODS

2.1. Participants and Study Design

College-aged women (age range = 20 to 25 yrs) enrolled in undergraduate classes at Louisiana State University in Baton Rouge, and older adult women (age range = 60 to 70 yrs) from St. James Place Continuing Retirement Community in Baton Rouge were invited to participate in this study. The study was approved by Louisiana State University Institutional Review Board. Respondents provided informed consent to participate in the study and were screened for the presence of diseases or conditions that would place them at high risk for adverse responses to exercise. Exclusion criteria for the study included any manifestation of cardiovascular (e.g., history of surviving sudden cardiac death, recent myocardial infarction, unstable angina, high blood pressure, frequent or complex ventricular ectopy), metabolic (e.g., diabetes mellitus), orthopedic or neurological (e.g., arthritis, osteoporosis, cognitive dysfunction) disease, or are on any medication (e.g., digitalis), which could affect the results of this study. Therefore, participant hemodynamics were studied in apparently healthy young and older adult women before, during, immediately after, and for 1 hour after performing a single bout of 5-RM and 15-RM bilateral knee-extension RE. The number of participants for this study was based on an effect size of exercise condition on post-exercise DBP response following RE of 0.91 (SD = 6.3; Expected difference = 5.7 mmHg), and an intraclass correlation coefficient for continuous BP recordings under various conditions of 0.75. In order to attain a power of 0.8, given the effect size and reliability of the data, in total 32 participants are required.
2.2. Materials and Equipments

The Health Status Questionnaire was used to screen for history of disease and other significant medical problems/conditions, and the Aerobic Center Longitudinal Study Physical Activity Questionnaire to estimate the participant’s initial level of habitual physical activity. A Monark 818E (Stockholm, Sweden) cycle-ergometer, a Polar HR monitor (Woodbury, NY), a sphygmomanometer, and a rating of perceived exertion (RPE Borg’s 20-point scale) were used during the sub-maximal grade exercise test to estimate participant’s cardiorespiratory exercise capacity (estimated maximal $O_2$ uptake).

A Hokanson EC-5R plethysmography system (Bellevue, WA) was used to assess forearm vascular function (blood inflow, vascular resistance, venous capacitance, and venous outflow) from the right arm at rest and following 5 minutes of forearm arterial occlusion. The same Hokanson EC-5R plethysmography system (Bellevue, WA) was used to assess forearm arterial blood inflow and vascular resistance from the right arm at pre-exercise condition and 4, 7, 10, 15, 20, 30, 40, 50, and 60 minutes after performing a single bout of 5-RM, and 15-RM of bilateral knee-extension RE. The Colin Model 7000 (Colin Instrument, San Antonio, TX) non-invasive tonometric BP device was used to capture the blood pressure waves throughout the exercise testing protocol. The Colin non-invasive continuous BP device makes use of arterial tonometry at the wrist to provide a continuous pressure wave. In comparison to intra-arterial measures of BP, various investigations have reported coefficients of correlation in the range of 0.81-0.91. The reliability of the device has been demonstrated at rest. This Colin 7000 was interfaced with a Biopac MP100 Data Acquisition System (Santa Barbara, CA) to collect continuous BP data at 200 Hz. In addition, ECG electrodes were placed on the participant’s chest, and interfaced with the Biopac MP100 data acquisition system (Santa Barbara, CA).
Barbara, CA) to collect continuous ECG at 200 Hz. ECG data were reduced to derive heart rate variability (mean R-R interval, SDNN, pnn50, LFnu, and LF/HF ratio), and assess autonomic function using the guidelines set forth by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart rate variability parameters were derived from 5- and 10-minute segments recorded during each exercise condition according to the following test periods: 5-minute segments (pre-exercise, recovery 0-5’, recovery 1-6’, recovery 2-7’, recovery 3-8’, recovery 4-9’, and recovery 5-10’), and 10-minute segments (pre-exercise, recovery 0-10’, recovery 10-20’, recovery 20-30’, recovery 30-40’, recovery 40-50’, and recovery 50-60’). Acqknowledge 4.0 software (Biopac System Incorporated, Santa Barbara, CA) was used to prepare the BP and ECG data for reduction and analysis, and Excel Macro and MATLAB 5.3 software was utilized to reduce the data providing the variables of interest (described below). A seated knee extension exercise device (Med-X Corporation, Ocala, FL) was used to provide the workloads for each exercise testing protocol, and a metronome to establish the cadence for concentric and eccentric phases of bilateral knee extension RE.

2.3. Procedure

The study procedure consisted of (a) three separate visits of preliminary assessments and familiarization with the exercise testing protocol, and (b) two separate visits of resistance exercise testing assessments. All five visits to the laboratory were made over a 4-week period, and each session lasted between 60 and 90 minutes.
2.3.1. Preliminary assessments and test familiarization

The preliminary assessments and familiarization with the exercise testing protocol were completed following the order indicated in Table 2.1.

<table>
<thead>
<tr>
<th>Visit</th>
<th>Preliminary Assessment/ Familiarization</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Consent Form, Health Status Questionnaires</td>
</tr>
<tr>
<td></td>
<td>Height, and Weight Measurements</td>
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<tr>
<td></td>
<td>Sub-maximal Cardiorespiratory Test</td>
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<td></td>
<td>Familiarization with the Knee-Extension Strength Test</td>
</tr>
<tr>
<td>2</td>
<td>Physical Activity Questionnaire</td>
</tr>
<tr>
<td></td>
<td>Hip, Waist, Thigh Circumference Measurements</td>
</tr>
<tr>
<td></td>
<td>Bilateral Knee-Extension Strength Test (5-RM)</td>
</tr>
<tr>
<td>3</td>
<td>Forearm Maximal Vasodilatory Capacity</td>
</tr>
<tr>
<td></td>
<td>Bilateral Knee-Extension Strength Test (15-RM)</td>
</tr>
</tbody>
</table>

During the **first visit**, the investigator explained the consent form in detail, and obtained the participant’s written consent to take part in the study. Thereafter, the participants completed a Health Status Questionnaire\(^8^6\) to screen for history of disease and other significant medical problems/conditions, and height and weight measurements were made using standard laboratory techniques. During this same session, the participant performed the YMCA sub-maximal cycle-ergometer exercise test for the purpose of describing the estimated cardiorespiratory (CR) exercise capacity of the population, and assuring that the participants did not have adverse CR responses to sub-maximal work\(^6^5\). Prior to the test, the participant was prepared as follows: (1) a Polar (Woodbury, NY) HR monitor was secured around the participant’s thoracic region, and (2) an adult blood pressure cuff was secured around the participant’s upper left arm. Heart rate (HR) and blood pressure were recorded in the supine, seated, and standing positions prior to the test, throughout each stage of the test, and for a minimum of 6
minutes post-test. Rating of Perceived Exertion (Borg 20 point scale) was recorded during each stage of the test. The investigator adhered strictly to the YMCA sub-maximal exercise protocol, and the test was terminated when the participant achieved a HR equal to 75-85% of her age-predicted maximum (220-age), achieved an RPE of 13, requested to stop, or demonstrated signs or symptoms that indicated termination of the test (ACSM)\textsuperscript{14}. Following termination, the participant pedaled for 4 minutes at a low-load and hemodynamic recovery was monitored for a period of no less than 6 minutes.

After the sub-maximal exercise test was completed, the participant was introduced to knee-extension resistance exercise device (Med-X Corporation, Ocala, FL), and instructed in proper knee-extension RE technique, and exercise cadence. At this time, the participant performed 3-4 trials of 5 repetitions of bilateral knee-extension exercise, starting with a weight equal to 60% of the participant’s body weight (light intensity). Thereafter, the technician gradually increased the load on the Med-X machine (between 10 to 20% of the starting weight). Participants recovered for approximately 4 minutes. Lastly, the participant walked on a motorized treadmill for 5 minutes at a pace determined to be fairly light (RPE = 11). This was followed by some light quadriceps and hamstring stretching exercises.

During this second visit, the participant completed the Aerobic Center Longitudinal Study Physical Activity Questionnaire\textsuperscript{68} to estimate the participant’s initial level of habitual physical activity. Next, the participant was asked to engage in hip, waist, and thigh circumference measurements with the purpose of estimating the hip/waist ratio, and exercising muscle mass involved, respectively. Thereafter, the participant performed the 5-repetition maximal Bilateral Knee-Extension Strength Test (the maximal resistance that
participants could be moved throughout the full range of motion for 5 repetitions). The bilateral knee-extension strength test involved gradually increases the load on the Med-X machines until the participant could perform no more than 5 repetitions in a controlled manner. Prior to the test, the participant engaged in light quadriceps and hamstring stretching activities, followed by at least 5 knee-extensions, not to exceed 60% of the participant body weight. Subsequently, the participant performed for each trial at least 5 repetitions in a controlled manner, and reported the Rating of Perceived Exertion (Borg 20 point scale) for each trial. For each successive trial, the weight was increased between 10 to 20% of previous weight used. In this fashion, the 5-RM was ascertained within 5 trials. Between trials, participants recovered for approximately 4 minutes. Following the 5-RM test, the participant performed a cool-down that involved treadmill walking for 5 minutes at a pace determined to be fairly light (RPE = 11). This was followed by light quadriceps and hamstring stretching exercises.

During the third visit, forearm arterial (blood inflow and vascular resistance) and venous (capacitance and outflow) function indices at rest and following 5 minutes of forearm arterial occlusion were obtained from the right arm using mercury strain-gauge plethysmography. Upon arrival at the laboratory, the participant completed the 24-hour physical activity report questionnaire, and thereafter the participant rested for a period of 30 minutes in a supine position. ECG electrodes were placed on the participant's torso and the ECG data were collected. A standard BP cuff was affixed to the participant's upper aspect of the left arm. The BP cuff was attached to a Colin Model 7000 non-invasive continuous BP monitor. This monitor makes use also of a piezo-resistance device attached to the participant's left wrist. This device was used to detect BP
continuously. A blood pressure cuff was also positioned around the participant’s upper right arm, a pediatric cuff was placed around the participant’s right wrist, and a mercury-in-silastic strain gauge was placed around the forearm approximately 10 cm distal to the olecranon process\(^85, 91-92, 126\). Both arms were resting at the same level and position while resting and during the test. One minute before the measurement of resting forearm blood inflow, the wrist cuff was inflated to 240 mmHg to occlude hand blood flow and thus isolate the forearm. A resting forearm blood inflow measure was acquired immediately on inflation of the upper arm using an upper arm venous occluding pressure of 7 mmHg below diastolic blood pressure. Forearm venous capacitance was measured after an additional 6 minutes of venous occlusion, and venous outflow following the release of pressure\(^87-88\). Resting forearm vascular function indices were measured twice at both the third and fifth minute of the pre-exercise time-frame measurements, and averaged. Thereafter, peak forearm blood flow measurements following 5 minutes of arterial occlusion (reactive hyperemia) were achieved by inflating the cuff on the upper arm to 240-mmHg for 5 minutes. Peak forearm arterial and venous function indices were determined as described above\(^85, 91-92, 126\).

After the forearm maximal vasodilatory-capacity test was completed, the participant performed 15-repetition maximal Bilateral Knee-Extension Strength Test (maximal resistance that could be moved through the full range of motion for 15 repetitions). This test was performed following a procedure similar to that used to determine the 5-RM. The only differences was that 15-RM strength test involved gradually increasing the load on the Med-X machines until the participant could perform up to 15 repetitions in controlled manner, and starting weight represented approximately 40% of participant’s
body weight. Participants warmed-up and cooled-down by engaging in low intensity treadmill walking and leg stretches. Then, the participant performed trials of at least 15 repetitions, at a starting weight equal to 40% of the participant’s body weight, and reported the Rating of Perceived Exertion (Borg 20 point scale) for each trial performed. For each successive trial, the weight was increased between 10 to 15% of previous weight used. Thus, the 15-RM was reached within five trials. Between trials, participants recovered for approximately 4 minutes. Lastly, the participant was asked to walk for 5 minutes on the treadmill, at the same pace used prior to the test, followed by some light quadriceps and hamstring stretching exercises.

2.3.2. Experimental Resistance Exercise Protocol

The experimental RE protocol assessed the acute hemodynamic responses (SBP, DBP, and HR) before, during, immediately after, and for 1 hour after performing a single bout of 5-RM, and 15-RM of bilateral knee-extension RE. Both resistance exercise-testing assessments were made in a random order, during two different days (visits number four, and five) at least one week apart (see Table 2.2). For both resistance exercise conditions, the participant was asked to fast at least for 8 hours, and restrict physical exercise, alcohol intake, and smoking for 24 hours preceding these assessment sessions.

<table>
<thead>
<tr>
<th>Table 2.2 Experimental Resistance Exercise Testing Protocol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visit</td>
</tr>
<tr>
<td>-------</td>
</tr>
<tr>
<td>4</td>
</tr>
<tr>
<td></td>
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<td>5</td>
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</tbody>
</table>
Upon arriving at the laboratory for the **fourth visit**, the participant rested quietly for a period of 15-min while seated on Med-X knee-extension device. The participant’s feet rested on a wood-block, before and after exercise. While resting, the participants completed the 24-physical activity questionnaire. ECG electrodes were placed on the participant's torso and the ECG data were collected. The standard BP cuff and tonometric blood pressure device were respectively affixed to the participant's upper aspect of the left arm and left wrist, and thereafter the Colin non-invasive BP monitor was calibrated. In addition, a blood pressure cuff was positioned around the participant’s upper right arm, a pediatric cuff was placed around the participant’s right wrist, and a mercury-in-silastic strain gauge was placed around the forearm approximately 10 cm distal to the olecranon process. Both arms were resting at the same level and position while resting and during the test. Following the 15-minute resting period, pre-exercise hemodynamic measurements were obtained during a 10-minute period, and thereafter the participant was instructed to perform the number of repetition maximal (5-RM, or 15-RM) of bilateral knee-extensions RE, randomly assigned. Each repetition was performed in 4 seconds (2 sec for the concentric phase, and 2 sec for the eccentric phase). The exercise phase was followed by a 60-minute recovery period in which the BP, and ECG data were recorded. Lastly, participants engaged in a 5-minute treadmill walking cool down at a pace determined to be fairly light (RPE = 11), which was followed by light quadriceps and hamstring stretching activities.

During the **fifth visit**, the assessment of the acute hemodynamic response to the following testing exercise conditions (15-RM, or 5-RM) was made in the same condition described in the visit number four.
2.4. Data Reduction and Analysis

The continuous BP waves were converted to tachograms of SBP and DBP, and then resample at 4 Hz. The data were then smoothed by passing the waveform through a low-pass digital filter with a frequency cutoff of 0.05 Hz using Excel 5.0 software. Excel 5.0 macros were then used to generate the pressure variables listed in Table 2.3 (Figure 2.1). Similarly the ECG waveforms were converted to tachograms of heart period, and resampled to 4Hz. The resultant tachograms were then submitted to scripts written for MatLab 5.3, for the purpose of deriving mean heart period (mean R-R interval), standard deviation of normal R-R interval (SDNN), low-frequency power of normalized units (LFnu), the ratio of low frequency to high frequency power (LF/HF ratio), and percentile of normal R-R intervals greater than 50 mille-seconds different from its predecessor (pnn50).

Data reduction and analysis for forearm arterial and venous function data were obtained according to previously established procedures. Resting blood inflow was recorded at a paper speed of 5 mm/seconds, and values were derived from the slope drawn at a best-fit tangent using the first 2-3 pulses. Calculations were made as a function of 60 seconds divided by the horizontal distance (mm) needed for the slope to rise vertically from pre-exercise to the top of the recording paper and multiplied by the full chart range. Peak forearm blood inflow was recorded at a paper speed of 25 mm/seconds. Analyses were performed using a slope drawn at a best fit tangent to the curves of the first 3 pulse waves post cuff release. Blood flows were then calculated from 60 seconds multiplied by the paper speed, and divided by the horizontal distance (mm) needed for the volume slope to increase by 20 mm vertically. Forearm vascular
<table>
<thead>
<tr>
<th>Hemodynamic Variables</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Magnitude of BP change</strong></td>
<td>Pre-exercise BP: 5-minute average, collected after at least 10 minutes of sitting and completed 2 minutes prior to exercise</td>
</tr>
<tr>
<td></td>
<td>Peak Exercise: Highest value observed during exercise</td>
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<tr>
<td></td>
<td>Local Minimum: The first local minimum BP value attained after exercise</td>
</tr>
<tr>
<td></td>
<td>Magnitude of Increase: The difference between Peak Exercise – Pre-exercise BP</td>
</tr>
<tr>
<td></td>
<td>Magnitude of Decline: Local minimum - Peak Exercise</td>
</tr>
<tr>
<td></td>
<td>1-minute post ratio: The ratio value dividing 1-min post-exercise BP by Peak exercise BP</td>
</tr>
<tr>
<td></td>
<td>3-minute post ratio: The ratio value dividing 3-min post-exercise BP by Peak exercise BP</td>
</tr>
<tr>
<td><strong>Rate of PEBP Decline</strong></td>
<td>Onset: Time from Peak exercise BP to local minimum BP value</td>
</tr>
<tr>
<td></td>
<td>Mean rate of Decline: The mean rate of BP drop for the period of time between the peak exercise and the first local minimum BP value</td>
</tr>
<tr>
<td><strong>Duration of PEBP Drop</strong></td>
<td>1-hr PEBP Drop: The difference between the 60-min BP recording and Pre-exercise BP.</td>
</tr>
<tr>
<td><strong>Recovery Overshoot Rate</strong></td>
<td>Local Maximum: The first local maximum BP value attained after the first local minimum</td>
</tr>
<tr>
<td></td>
<td>Magnitude of Recovery: The difference between the first local minimum BP and the first local maximum BP values</td>
</tr>
<tr>
<td></td>
<td>Time of Recovery: Time from the first local minimum to first local maximum</td>
</tr>
<tr>
<td></td>
<td>Mean rate of Recovery: The rate of BP increase for the period of time between the first local minimum and the first local maximum BP values</td>
</tr>
<tr>
<td><strong>Autonomic Indicators</strong></td>
<td>MAP: Derived from continuous BP data using Acqknowledge 4.0 software</td>
</tr>
<tr>
<td></td>
<td>R-R interval: The time period between two consecutive R-R on ECG wave</td>
</tr>
<tr>
<td></td>
<td>SDNN: The standard deviation of normal R-R intervals</td>
</tr>
<tr>
<td></td>
<td>PNN50: Percentile of normal R-R intervals greater than 50 msec diff. from its predecessor</td>
</tr>
<tr>
<td></td>
<td>LFnu: Low frequency power of normalized unit</td>
</tr>
<tr>
<td></td>
<td>LF/HF ratio: The ratio dividing low frequency power by high frequency power.</td>
</tr>
<tr>
<td><strong>Forearm Vascular function</strong></td>
<td>Forearm Blood Inflow: Mean rate of change of forearm diameter after venous occlusion</td>
</tr>
<tr>
<td></td>
<td>Forearm Vascular Resistance: Mean arterial pressure divided by the forearm blood inflow</td>
</tr>
<tr>
<td></td>
<td>Forearm Venous Capacitance: Change in forearm-volume graph after 6 minutes of venous filling</td>
</tr>
<tr>
<td></td>
<td>Forearm Venous Outflow: Vertical drop of forearm-volume after the release of venous occlusion</td>
</tr>
</tbody>
</table>

resistance was then derived by dividing mean arterial pressure by the forearm blood inflow. Forearm venous capacitance was calculated as the vertical distance (mm) representing the change in forearm-volume graph following 6 minutes of venous filling. Forearm venous outflow was derived from a tangent line representing the vertical drop in the volume-graph from the excursion line and drawn at 0.5 second and 2-seconds.
following the release of the venous occlusion pressure. All vascular indices were expressed in milliliters per 100 milliliters of tissue per minute. 

Figure 2.1 Tachogram of SBP with Smoothed Data During, and Immediately After Resistance Exercise in Women.

In summary, the following provides a list of variables that are reported subsequent to the data analysis (These variables area summarized in Table 2.3).

**Pre-exercise values:** The pre-exercise values for SBP, DBP, MAP, and HR are the mean of the values obtained during the first 5-minute taken from the 10-minute pre-exercise resting-period, at least 15 minutes after being seated in the knee-extension device, and 2 minutes before starting exercise conditions. Pre-exercise values for 5-minute HRV indices (i.e., R-R interval, SDNN, pnn50, LFnu, and LF/HF ratio) were derived within the same time frame (the first 5-minute taken from the 10-minute pre-exercise resting-period) used to acquire BP parameters, and during the entire 10-minute
pre-exercise resting-period. The pre-exercise values for forearm blood inflow were obtained at the third and fifth minute of the pre-exercise resting-period, and averaged.

**Exercise values:** The exercise values include the peak exercise values for SBP, DBP, MAP, and HR attained during each exercise conditions, and the difference between the peak-exercise and the pre-exercise SBP, DBP, MAP, and HR values (magnitude of increase) for each exercise condition.

**Post-exercise values:** The post-exercise values include: (1) the local min BP, (2) the local max BP, (3) the time difference between the peak exercise BP and the local min BP, (4) the time difference between the local min BP and the local max BP, and (5) BP values recorded at 1’, 3’, 5’, 10’, 15’, 20’, 30’, 40’, 50’, and 60’ after exercise.

The other parameters of interest were derived from these measurements, which included (1) the difference between the peak exercise BP, and the post-exercise local min BP, (2) the recovery ratio by dividing the 1’ post-exercise BP by the peak exercise BP, (3) the recovery ratio by dividing the 3’ post-exercise BP by the peak exercise BP, (4) the mean rate of BP drop for the period of time between the peak exercise BP, and the post-exercise local min BP, (5) the difference between the local min BP and the local max BP, and (6) the rate of BP increase for the period of time between the local min BP and the local max BP.

The post-exercise values for autonomic function indices (i.e., heart rate variability profiles [R-R interval, SDNN, pnn50, LFnu, and LF/HF ratio]) were obtained for both 5-, and 10-minute segments. Post-exercise 5-minute segment HRV data were derived starting the first 5-minute segment just right after the cessation of the activity, and the subsequent 5-minute segments were taken 1 minute after the starting point of the prior
segment (R0-5, R1-6, R2-7, R3-8, R4-9, and R5-10). Post-exercise 10-minute HRV data were derived starting the first 10-minute segment just right after the cessation of the activity and the subsequent 10-minute segments were taken 10 minutes after the starting point of the prior segment (R0-10, R10-20, R20-30, R30-40, R40-50, and R50-60).

The post-exercise values for forearm blood inflow and forearm vascular resistance were obtained at 4’, 7’, 10’, 15’, 20’, 30’, 40’, 50’, and 60’ after both exercise conditions.

Additionally, the relationships between indicators of autonomic (i.e., SDNN, pnn50, LFnu, LF/HF ratio) and vascular function (i.e., forearm blood flow, forearm vascular resistance, forearm venous capacitance, and forearm venous outflow at rest and following 5 minutes of arterial occlusion), and hemodynamic responses (i.e., magnitude and rate of post-exercise BP change) following both exercise conditions were examined.

2.5. Statistical Analysis

1.) Age-group differences in descriptive parameters (e.g., age, height, weight) and hemodynamic parameters (e.g., resting arterial BP, HR, and vascular function indices) were analyzed using simple t-tests; 2.) age group by exercise condition differences on hemodynamic parameters (e.g., indicators of autonomic function, magnitude of increase, magnitude of drop) were analyzed using 2 (Age: young vs. old) by 2 (Exercise condition: 5-RM vs. 15-RM) ANOVAs; 3.) main effects of test condition, exercise condition and age group, and interaction effects on hemodynamic parameters (e.g., SBP, DBP, MAP, and HR) before, during, and immediately after RE were analyzed using 2 (Age: young vs. old) by 2 (Exercise Condition: 5-RM vs. 15-RM) by 4 (Time: pre-exercise, peak, local min and local max) mixed model ANOVAs with repeated measures on BP and HR data for all test periods; 4.) main effects of test condition, exercise condition and age group,
and interaction effects on hemodynamic parameters (e.g., SBP, DBP, MAP, and HR) before, during, and for 1 hour after RE were analyzed using 2 (Age: young vs. old) by 2 (Exercise condition: 5-RM vs. 15-RM) by 12 (Time: pre-exercise, peak, 1’, 3’, 5’, 10’, 15’, 20’, 30’, 40’, 50’, and 60’) mixed model ANOVA with repeated measures on BP and HR data for all test periods; 5.) main effects of test condition, exercise condition and age group, and interaction effects on autonomic function indices (i.e., heart rate variability profiles) before and immediately after RE were analyzed using 2 (Age: young vs. old) by 2 (Exercise condition: 5-RM vs. 15-RM) by 7 (Time: pre-exercise, R0-5’, R1-6’, R2-7’, R3-8’, R4-9’, and R5-10’) mixed model ANOVAs with repeated measures on HRV indicators (R-R interval, SDNN, pnn50, LFnu, and LF/HF ratio) for all test periods; 6.) main effects of test condition, exercise condition and age group, and interaction effects on autonomic function indices (i.e., heart rate variability profiles) before and for 1 hour after RE were analyzed using 2 (Age: young vs. old) by 2 (Exercise condition: 5-RM vs. 15-RM) by 10 (Time: pre-exercise, 4’, 7’, 10’, 15’, 20’, 30’, 40’, 50’, and 60’) mixed model ANOVAs with repeated measures on HRV indicators (R-R interval, SDNN, pnn50, LFnu, and LF/HF ratio) for all test periods; 7.) main effects of test condition, exercise condition and age group, and interaction effects on vascular function indices (e.g., forearm blood flow, and forearm vascular resistance) before and for 1-hour after RE were analyzed using 2 (Age: young vs. old) by 2 (Exercise condition: 5-RM vs. 15-RM) by 10 (Time: pre-exercise, 4’, 7’, 10’, 15’, 20’, 30’, 40’, 50’, and 60’) mixed model ANOVAs with repeated measures on vascular function indices (i.e., forearm blood flow, and forearm vascular resistance) for all test periods; and 8.) associations between post-exercise hemodynamic parameters and indicators of autonomic function, and post-
exercise hemodynamic parameters and vascular function indices were examined using Pearson Correlation and Simple Linear Regression.

All tests were conducted using an alpha of 0.05, and LSD post hoc pairwise comparisons were used to examine for differences between post-exercise and pre-exercise hemodynamic measurements as required.
3. RESULTS

The following is a summary of the results of the experiment. An extended results section appears in Appendix B of this dissertation.

3.1. Participant Characteristics

Of the 40 respondents, 32 passed screening and elected to continue with the study. Of the 8 respondents who did not participate in the study, 2 young women withdrew before providing informed consent, and 6 older women did not meet the inclusion criteria. The latter either had signs or symptoms consistent with high risk for adverse responses to exercise according to the ACSM guidelines, such as orthopedic or neurological disease (n = 2), high blood pressure (n = 2), or were on medication that could affect the results of this study (n = 2). Therefore, the following data are from 32 apparently healthy White participants, 16 young women (mean age = 21 ± 2.3 yrs) and 16 older women (mean age = 69 ± 2.3 yrs). Descriptive statistics for the participant’s characteristics, pre-exercise autonomic indicators from 5-, 10-minute segments, resting vascular function indices, and pre-exercise vascular function indices are reported from Table 3.1 to 3.5, respectively.

The results of t-test comparisons revealed age-group differences in (1) participant’s characteristics: weight (p = 0.04), waist-hip ratio (p = 0.02), estimated VO2 max (p = 0.0001), 5-RM (p = 0.0001), and 15-RM (p = 0.002); (2) pre-exercise autonomic indicators: R-R interval (p = 0.004/0.0001), SDNN (p = 0.0001/0.0001); and pnn50 (p = 0.0001/ 0.0001); (3) resting arterial BP, HR, and vascular function indices: SBPrest (p = 0.0001), MAPrest (p = 0.0001) FVRrest (p = 0.05), SBPocc (p = 0.05), DBPocc (p = 0.05), MAPocc (p = 0.05), and FVRocc (p = 0.006); and (4) pre-exercise arterial BP, HR, and vascular function indices: SBP (p = 0.0001), MAP (p = 0.01), and HR (p = 0.001), (Table 3.1-3.5, respectively).
### Table 3.1  Participant’s characteristics

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>Age (year)</td>
<td>21.7 ± 1.6</td>
<td>69.8 ± 3.9  *</td>
</tr>
<tr>
<td>Height (inch)</td>
<td>63.4 ± 2.2</td>
<td>63.9 ± 2.9</td>
</tr>
<tr>
<td>Weight (lbs)</td>
<td>127.6 ± 19.8</td>
<td>143.7 ± 22.9  *</td>
</tr>
<tr>
<td>Waist/Hip ratio</td>
<td>0.74 ± 0.03</td>
<td>0.79 ± 0.06  *</td>
</tr>
<tr>
<td>Est. VO₂ (ml/kg/min)</td>
<td>39.8 ± 9.0</td>
<td>25.9 ± 3.9  *</td>
</tr>
<tr>
<td>5-RM (ft-lbs)</td>
<td>200.5 ± 40.8</td>
<td>128.9 ± 39.6  *</td>
</tr>
<tr>
<td>15-RM (ft-lbs)</td>
<td>119.4 ± 29.4</td>
<td>92.4 ± 13.3  *</td>
</tr>
<tr>
<td>Right Leg circumference (cm)</td>
<td>57.0 ± 5.1</td>
<td>56.4 ± 5.0</td>
</tr>
<tr>
<td>Left Leg circumference (cm)</td>
<td>56.6 ± 4.7</td>
<td>55.8 ± 5.0</td>
</tr>
</tbody>
</table>

Values are mean ± SD. 5-RM = 5-/ 15-RM = 5-/15 repetition maximum. * = Diff. from young group (p < 0.05). + = trend for age-group differences (p = 0.11).

### Table 3.2  Pre-exercise Autonomic Indicators from 5-min segments

<table>
<thead>
<tr>
<th>Age group</th>
<th>Heart Rate variability Profiles</th>
<th>R-R Interval</th>
<th>SDNN</th>
<th>Pnn50</th>
<th>LFnu</th>
<th>LF/HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young</td>
<td>5-RM</td>
<td>855.8 ± 130.5</td>
<td>55.9 ± 17.3</td>
<td>19.3 ± 15.7</td>
<td>0.61 ± 0.15</td>
<td>2.11 ± 1.64</td>
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<tr>
<td></td>
<td>15-RM</td>
<td>873.3 ± 139.9</td>
<td>64.1 ± 19.0</td>
<td>24.8 ± 19.1</td>
<td>0.60 ± 0.15</td>
<td>1.92 ± 1.35</td>
</tr>
<tr>
<td></td>
<td>Average</td>
<td>864.6 ± 133.3</td>
<td>59.9 ± 18.3</td>
<td>22.1 ± 17.5</td>
<td>0.60 ± 0.15</td>
<td>2.01 ± 1.48</td>
</tr>
<tr>
<td>Older</td>
<td>5-RM</td>
<td>966.6 ± 136.2 *</td>
<td>33.7 ± 11.6 *</td>
<td>5.0 ± 9.3 *</td>
<td>0.58 ± 0.17</td>
<td>1.99 ± 1.76</td>
</tr>
<tr>
<td></td>
<td>15-RM</td>
<td>960.8 ± 122.5 *</td>
<td>31.6 ± 13.0 *</td>
<td>4.8 ± 9.2 *</td>
<td>0.58 ± 0.18</td>
<td>1.87 ± 1.38</td>
</tr>
<tr>
<td></td>
<td>Average</td>
<td>963.8 ± 127.6 *</td>
<td>32.6 ± 12.2 *</td>
<td>4.9 ± 9.1 *</td>
<td>0.58 ± 0.17</td>
<td>1.93 ± 1.56</td>
</tr>
</tbody>
</table>

Values are mean ± SD. * = Different from young group (p < 0.05).

### Table 3.3  Pre-exercise Autonomic Indicators from 10-min segments

<table>
<thead>
<tr>
<th>Age group</th>
<th>Heart Rate variability Profile</th>
<th>R-R Interval</th>
<th>SDNN</th>
<th>Pnn50</th>
<th>LFnu</th>
<th>LF/HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young</td>
<td>05-RM</td>
<td>846.9 ± 132.5</td>
<td>66.8 ± 20.1</td>
<td>18.9 ± 15.2</td>
<td>0.65 ± 0.13</td>
<td>2.40 ± 1.70</td>
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<tr>
<td></td>
<td>15-RM</td>
<td>849.7 ± 126.7</td>
<td>71.8 ± 22.8</td>
<td>21.0 ± 15.6</td>
<td>0.63 ± 0.14</td>
<td>2.16 ± 1.42</td>
</tr>
<tr>
<td></td>
<td>Average</td>
<td>848.3 ± 127.5</td>
<td>69.2 ± 21.3</td>
<td>22.1 ± 17.5</td>
<td>0.64 ± 0.13</td>
<td>2.29 ± 1.55</td>
</tr>
<tr>
<td>Older</td>
<td>05-RM</td>
<td>960.8 ± 130.7 *</td>
<td>46.4 ± 26.4 *</td>
<td>8.2 ± 15.0 *</td>
<td>0.61 ± 0.16</td>
<td>2.19 ± 1.82</td>
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<tr>
<td></td>
<td>15-RM</td>
<td>954.7 ± 120.7 *</td>
<td>43.2 ± 14.7 *</td>
<td>5.2 ± 9.0 *</td>
<td>0.60 ± 0.18</td>
<td>2.20 ± 1.69</td>
</tr>
<tr>
<td></td>
<td>Average</td>
<td>957.8 ± 123.9 *</td>
<td>45.0 ± 21.7 *</td>
<td>6.8 ± 12.6 *</td>
<td>0.61 ± 0.17</td>
<td>2.19 ± 1.73</td>
</tr>
</tbody>
</table>

Values are mean ± SD. * = Different from young group (p < 0.05).
Table 3.4  Resting and After Occlusion Vascular Function Indices

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Young</th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HR</strong> (beat/min)</td>
<td>62.1 ± 13.4</td>
<td>59.9 ± 5.2</td>
</tr>
<tr>
<td><strong>SBP</strong> (mm Hg)</td>
<td>101.1 ± 5.7</td>
<td>124.3 ± 13.4 *</td>
</tr>
<tr>
<td><strong>DBP</strong> (mm Hg)</td>
<td>60.4 ± 6.7</td>
<td>64.4 ± 7.1 +</td>
</tr>
<tr>
<td><strong>MAP</strong> (mm Hg)</td>
<td>74.0 ± 6.0</td>
<td>84.3 ± 7.7 *</td>
</tr>
<tr>
<td><strong>FBF</strong> (mL/100mL/min)</td>
<td>1.90 ± 0.78</td>
<td>1.66 ± 0.70</td>
</tr>
<tr>
<td><strong>FVR</strong> (U)</td>
<td>44.5 ± 16.2</td>
<td>60.2 ± 25.4 *</td>
</tr>
<tr>
<td><strong>FVC</strong> (mL/100mL/min)</td>
<td>3.95 ± 0.73</td>
<td>3.89 ± 1.07</td>
</tr>
<tr>
<td><strong>FVO</strong> (mL/100mL/min)</td>
<td>48.2 ± 9.82</td>
<td>41.1 ± 8.70 +</td>
</tr>
</tbody>
</table>

After Occlusion

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Young</th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HR</strong> (beat/min)</td>
<td>59.0 ± 8.6</td>
<td>62.7 ± 16.5</td>
</tr>
<tr>
<td><strong>SBP</strong> (mm Hg)</td>
<td>104.6 ± 7.6</td>
<td>131.8 ± 27.7 *</td>
</tr>
<tr>
<td><strong>DBP</strong> (mm Hg)</td>
<td>60.9 ± 4.8</td>
<td>68.0 ± 8.1 *</td>
</tr>
<tr>
<td><strong>MAP</strong> (mm Hg)</td>
<td>75.5 ± 5.2</td>
<td>89.3 ± 13.8 *</td>
</tr>
<tr>
<td><strong>FBF</strong> (mL/100mL/min)</td>
<td>21.9 ± 5.54</td>
<td>18.3 ± 5.43 +</td>
</tr>
<tr>
<td><strong>FVR</strong> (U)</td>
<td>3.61 ± 0.86</td>
<td>5.37 ± 2.00 *</td>
</tr>
<tr>
<td><strong>FVC</strong> (mL/100mL/min)</td>
<td>2.87 ± 0.92</td>
<td>2.80 ± 1.18</td>
</tr>
<tr>
<td><strong>FVO</strong> (mL/100mL/min)</td>
<td>43.0 ± 12.5</td>
<td>43.1 ± 13.3</td>
</tr>
</tbody>
</table>

Values are mean ± SD. HR = Heart rate. SBP = Systolic blood pressure. DBP = Diastolic blood pressure. MAP = Mean arterial blood pressure. FBF = Forearm blood inflow. FVR = Forearm vascular resistance. FVC = Forearm venous capacitance. FVO = Forearm venous outflow.

* = Different from young group (p < 0.05). + = trend towards an age group difference (p >0.05).

Table 3.5  Pre-exercise Vascular Function Indices

<table>
<thead>
<tr>
<th>5-RM</th>
<th>15-RM</th>
<th>Average</th>
<th>5-RM</th>
<th>15-RM</th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HR</strong></td>
<td>72.5 ±12.0</td>
<td>71.2 ±12.0</td>
<td>71.9 ±11.8</td>
<td>64.0 ± 8.8</td>
<td>62.3 ± 7.7</td>
</tr>
<tr>
<td><strong>SBP</strong></td>
<td>104.4 ± 9.0</td>
<td>105.0 ± 6.4</td>
<td>104.7 ± 7.7</td>
<td>121.3 ± 11.7</td>
<td>123.7 ± 15.1</td>
</tr>
<tr>
<td><strong>DBP</strong></td>
<td>59.3 ±10.2</td>
<td>58.9 ± 8.3</td>
<td>59.1 ± 9.2</td>
<td>57.9 ± 8.6</td>
<td>60.0 ± 9.5</td>
</tr>
<tr>
<td><strong>MAP</strong></td>
<td>74.3 ± 9.6</td>
<td>74.3 ± 7.2</td>
<td>74.3 ± 8.4</td>
<td>79.0 ± 8.3</td>
<td>81.2 ±10.0</td>
</tr>
<tr>
<td><strong>FBF</strong></td>
<td>1.93 ± 0.4</td>
<td>1.93 ± 0.6</td>
<td>1.90 ± 0.4</td>
<td>2.12 ± 0.6</td>
<td>1.92 ± 0.7</td>
</tr>
<tr>
<td><strong>FVR</strong></td>
<td>38.3 ± 6.5</td>
<td>39.0 ± 9.9</td>
<td>39.4 ± 8.1</td>
<td>38.8 ± 9.5</td>
<td>43.4 ±11.1</td>
</tr>
</tbody>
</table>

Values are mean ± SD. HR = Heart rate (beat/min). SBP = Systolic blood pressure (mm Hg). DBP = Diastolic blood pressure (mm Hg). MAP = Mean arterial blood pressure (mm Hg). FBF = Forearm blood inflow (mL/100mL/min). FVR = Forearm Vascular Resistance (Units).

* = Different from young group (p < 0.05).
3.2. General Effect of the Resistance Exercise (Main Effect of Test Condition).

As expected, in both short-term (i.e., before, during, and immediately after of RE) and long-term (i.e., before, during, and for 1-hour after RE) recovery period assessments of hemodynamic responses during and following RE (Figures 3.1-3.5, and Tables 3.6-3.10),

![Diagram showing changes in blood pressure (BP) and heart rate (HR) before, during, and after resistance exercise (RE). The diagram includes symbols indicating statistical significance (* for different from pre-exercise and † for different from all recovery periods).](image)

**Figure 3.1** General Effect of RE (Test Condition) on Arterial BP and HR Before, During & Immediately After Exercise in Women.
Values are mean ± SD. Blood pressure (BP) and heart rate (HR) before exercise (Pre-), during (Peak), and immediately after (Local min and local max) RE. SBP = Systolic Blood Pressure. DBP = Diastolic Blood Pressure. MAP = Mean Arterial Blood Pressure. HR = Heart Rate. * = Different from pre-exercise ($p < 0.05$). † = Diff from all recovery periods ($p < 0.05$).
Table 3.6  General Effect of RE (Test Condition) on Arterial BP and HR Before, During, & Immediately After RE in Women

<table>
<thead>
<tr>
<th></th>
<th>SBP</th>
<th>DBP</th>
<th>MAP</th>
<th>HR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-</td>
<td>113.5 ± 14.1</td>
<td>59.1 ± 9.0</td>
<td>77.6 ± 8.9</td>
<td>67.6 ± 11.0</td>
</tr>
<tr>
<td>Peak</td>
<td>149.1 ± 22.1 * †</td>
<td>86.5 ± 15.5 * †</td>
<td>107.6 ± 15.1 * †</td>
<td>105.4 ± 20.3 * †</td>
</tr>
<tr>
<td>Local min</td>
<td>123.8 ± 18.5 *</td>
<td>56.0 ± 12.5 *</td>
<td>78.2 ± 12.8</td>
<td>77.9 ± 18.4 *</td>
</tr>
<tr>
<td>Local max</td>
<td>127.1 ± 18.3 *</td>
<td>59.2 ± 12.1</td>
<td>80.6 ± 13.0 *</td>
<td>81.7 ± 18.4 *</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Blood pressure (BP) and heart rate (HR) before (Pre-), during (Peak), and immediately after (Local min and local max) resistance exercise. SBP = Systolic blood pressure. DBP = Diastolic blood pressure. HR = Heart rate. MAP = Mean arterial blood pressure.

* = Different from young group (p ≤ 0.05). † = Diff from all recovery periods (p ≤ 0.05).

there were significant main effects of the test condition on SBP (p = 0.0001), DBP (p = 0.0001), MAP (p = 0.0001), and HR (p = 0.0001) as well as in the assessments before and after RE on HRV profiles (p = 0.0001) and FVR (p = 0.003) (Figure 3.1-3.5, Table 3.6-3.10, respectively).

In general, RE was associated with an increase in arterial BP and HR (Extended Results Section, Table B.7, B.12, & B.17). Immediately following the cessation of exercise, these parameters returned towards pre-exercise levels; however, in the case of SBP, pressure dropped to a local min pressure that was above pre-exercise level (Table B.7) and did not return completely to pre-exercise level until 5’ into recovery. In the case of DBP, the pressures dropped below pre-exercise almost immediately (~20 seconds to reach local min) (Table B.12), returned to pre-exercise level shortly thereafter, and then (~1 minute into recovery) dropped below pre-exercise again, and continued to drop gradually over the ensuing hour. MAP followed a pattern nearly identical to the DBP. In the hour following the resistance exercise, SBP reached its nadir by about 10 minutes into the recovery period (mean ± SE: R10’ = -3.97 ± 1.64 mm Hg; p = 0.018), with no significant changes thereafter (mean ± SE: R60’ = -4.75 ± 1.74 mm hg; p = 0.008). In contrast, DBP dropped slightly over time with the result being that DBP observed at 1’
post exercise (mean ± SE: R1’ = -2.79 ± 1.12 mm Hg; \( p = 0.015 \)) was lower than pre-exercise level with a further gradual reduction throughout the 60’ recovery period (mean ± SE: R60’ = -6.24 ± 1.34 mm Hg; \( p = 0.0001 \)). Similarly, MAP was lower than pre-exercise level at 5’ following the activity (mean ± SE: R5’ = -2.53 ± 1.18 mm Hg; \( p = 0.037 \)) and continued to decline throughout the recovery period (mean ± SE: R60’ = -5.64 ± 1.33 mm Hg; \( p = 0.015 \)) (Figure 3.2, and Table 3.7).

![Graph showing changes in BP and HR before, during, and after exercise](image)

**Figure 3.2** General Effect of RE (Test Condition) on Arterial BP and HR Before, During, & For 1 Hour After Exercise in Women

Values are mean ± SD. Blood Pressure and heart rate before (Pre-), during (Peak), and for 1 hour after RE. SBP = Systolic blood pressure. DBP = Diastolic blood pressure. MAP = Mean arterial pressure. HR = Heart rate. * = Diff from pre-exercise (\( p \leq 0.05 \)). † = Diff from all recovery periods (\( p \leq 0.05 \)). # = tended to be different from pre-exercise (\( p \leq 0.15 \)).
Table 3.7  General Effect of RE (Test Condition) on Arterial BP and HR
Before, During, & For 1 Hour After RE in Women

<table>
<thead>
<tr>
<th>SBP</th>
<th>DBP</th>
<th>MAP</th>
<th>HR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-</td>
<td>113.5 ± 14.1</td>
<td>59.1 ± 9.0</td>
<td>77.6 ± 8.9</td>
</tr>
<tr>
<td>Peak</td>
<td>149.1 ± 22.1</td>
<td>86.5 ± 15.5</td>
<td>107.6 ± 15.1</td>
</tr>
<tr>
<td>R1’</td>
<td>120.3 ± 16.7</td>
<td>56.4 ± 11.7</td>
<td>78.2 ± 11.5</td>
</tr>
<tr>
<td>R3’</td>
<td>116.7 ± 15.8</td>
<td>55.6 ± 11.4</td>
<td>76.4 ± 11.3</td>
</tr>
<tr>
<td>R5’</td>
<td>113.9 ± 14.8</td>
<td>55.1 ± 11.4</td>
<td>75.2 ± 10.9</td>
</tr>
<tr>
<td>R10’</td>
<td>109.6 ± 13.3</td>
<td>53.6 ± 10.7</td>
<td>72.7 ± 9.9</td>
</tr>
<tr>
<td>R15’</td>
<td>109.4 ± 13.8</td>
<td>54.5 ± 10.3</td>
<td>73.4 ± 9.7</td>
</tr>
<tr>
<td>R20’</td>
<td>111.0 ± 14.9</td>
<td>54.4 ± 10.3</td>
<td>74.0 ± 10.0</td>
</tr>
<tr>
<td>R30’</td>
<td>110.3 ± 14.5</td>
<td>53.0 ± 10.4</td>
<td>72.7 ± 10.1</td>
</tr>
<tr>
<td>R40’</td>
<td>110.7 ± 14.9</td>
<td>54.0 ± 11.1</td>
<td>73.6 ± 10.9</td>
</tr>
<tr>
<td>R50’</td>
<td>110.3 ± 14.3</td>
<td>52.8 ± 10.5</td>
<td>72.3 ± 10.1</td>
</tr>
<tr>
<td>R60’</td>
<td>108.9 ± 15.4</td>
<td>52.9 ± 10.9</td>
<td>72.1 ± 10.2</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Systolic (SBP) and diastolic (DBP) blood pressure, mean arterial pressure (MAP), and heart rate (HR) before (Pre-), during (Peak), and immediately after (Local min and local max) resistance exercise (RE). * = Diff from pre-exercise ($p < 0.05$). † = Diff from all recovery periods ($p < 0.05$). # = tended to be diff from pre-exercise ($p < 0.15$).

Heart rate increased during the exercise bout and returned towards pre-exercise level (Extended Results Section, Table B.17), but before reaching pre-exercise level, increased slightly over a period of ~30 seconds, and from there recovered to pre-exercise level by about 3 minutes into the recovery period, with no significant change from that time point forward (Figure 3.1-3.2, & Table 3.6-3.7). The HRV data were also subject to a significant effect of test period. Of particular relevance is the appearance of a lower than pre-exercise level LFnu (Figure 3.3, & Table 3.8) and LF/HF ratio in the 5-minute segments (R1-6, R2-7, and R3-8) beginning 1 minute after exercise, and a tendency for LFnu and LF/HF ratio to increase slightly throughout the recovery period with the result being that LFnu at R40-50 and R50-60 (Figure 3.4, & Table 3.9), and LF/HF ratio at R30-40, R40-50, and R50-60 higher than pre-exercise level. The time domain data are more difficult to interpret in so far as the increase in SDNN and pnn50 are representative of non-stationary time signals that occur with exercise. Otherwise, pre-exercise did not
appear to be different from recovery (Table 3.8-3.9). The FVR data were also subject to a significant effect of test period \((p = 0.003)\). In general, post-exercise FVRs were higher than pre-exercise (Figure 3.5, & Table 3.10).

![Figure 3.3 General Effect of RE (Test Condition) on LFnu (5-minute segments) Before & Immediately After Exercise in Women](image)

Values are mean ± SD. LFnu (5-minute segments) before exercise (Pre-) and immediately after RE. * = Different from Pre-exercise \((p < 0.05)\).

![Table 3.8 General Effect of RE (Test Condition) on HRV Indices (5-minute segment) Before and Immediately After RE in Women](image)

<table>
<thead>
<tr>
<th>Age group</th>
<th>R-R Interval</th>
<th>SDNN</th>
<th>Pnn50</th>
<th>LFnu</th>
<th>LF/HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-</td>
<td>913.4 ± 138.8</td>
<td>49.1 ± 25.0</td>
<td>13.8 ± 16.4</td>
<td>0.59 ± 0.16</td>
<td>1.97 ± 1.51</td>
</tr>
<tr>
<td>Rec0-5</td>
<td>874.5 ± 133.0*†</td>
<td>84.2 ± 34.1*†</td>
<td>17.5 ± 18.2*</td>
<td>0.61 ± 0.13</td>
<td>1.96 ± 1.36</td>
</tr>
<tr>
<td>Rec1-6</td>
<td>899.9 ± 134.2*†</td>
<td>67.6 ± 29.6*†</td>
<td>18.5 ± 19.2*</td>
<td>0.56 ± 0.16*</td>
<td>1.63 ± 1.32*</td>
</tr>
<tr>
<td>Rec2-7</td>
<td>917.2 ± 136.2</td>
<td>56.1 ± 25.4*†</td>
<td>18.2 ± 19.3*</td>
<td>0.55 ± 0.15*</td>
<td>1.63 ± 1.21*</td>
</tr>
<tr>
<td>Rec3-8</td>
<td>922.4 ± 135.3</td>
<td>50.3 ± 26.1</td>
<td>16.4 ± 17.8*</td>
<td>0.55 ± 0.17*</td>
<td>1.67 ± 1.29*</td>
</tr>
<tr>
<td>Rec4-9</td>
<td>919.7 ± 135.2</td>
<td>48.5 ± 26.2</td>
<td>14.9 ± 17.2</td>
<td>0.58 ± 0.15</td>
<td>1.78 ± 1.31#</td>
</tr>
<tr>
<td>Rec5-10</td>
<td>916.7 ± 135.0</td>
<td>47.9 ± 25.4</td>
<td>13.7 ± 16.5</td>
<td>0.59 ± 0.14</td>
<td>1.87 ± 1.34</td>
</tr>
</tbody>
</table>

Values are mean ± SD. HRV = Heart rate variability. * = Different from pre-exercise \((p < 0.05)\). † = Diff from all recovery periods \((p < 0.05)\). # =tended to be diff from pre-exercise \((p < 0.15)\).
Figure 3.4 General Effect of RE (Test Condition) on LFnu (10-minute segments) Before & For 1 Hour After Exercise in Women
Values are mean ± SD. LFnu (10-minute segments) before exercise (Pre-), and for 1 hour after RE. * = Different from pre-exercise (p < 0.05).

Table 3.9 General Effect of RE (Test Condition) on HRV Indices (10-minute segment) Before and For 1 Hour After RE in Women

<table>
<thead>
<tr>
<th>Age group</th>
<th>Heart Rate variability Profile</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>R-R Interval</td>
<td>SDNN</td>
<td>Pnn50</td>
<td>LFnu</td>
<td>LF/HF</td>
</tr>
<tr>
<td>Pre-</td>
<td>903.0 ± 136.4</td>
<td>57.5 ± 24.5</td>
<td>13.6 ± 15.3</td>
<td>0.63 ± 0.15</td>
<td>2.24 ± 1.63</td>
</tr>
<tr>
<td>Rec0-10</td>
<td>894.6 ± 134.6*†</td>
<td>74.8 ± 28.1*†</td>
<td>16.0 ± 16.3*</td>
<td>0.61 ± 0.12</td>
<td>1.92 ± 1.18</td>
</tr>
<tr>
<td>Rec10-20</td>
<td>911.2 ± 140.5</td>
<td>48.7 ± 21.4*#</td>
<td>12.4 ± 15.2</td>
<td>0.64 ± 0.14</td>
<td>2.31 ± 1.62</td>
</tr>
<tr>
<td>Rec20-30</td>
<td>906.2 ± 134.9</td>
<td>52.0 ± 22.4*</td>
<td>12.7 ± 13.8</td>
<td>0.64 ± 0.15</td>
<td>2.33 ± 1.50</td>
</tr>
<tr>
<td>Rec30-40</td>
<td>909.2 ± 132.9</td>
<td>54.2 ± 24.9</td>
<td>13.2 ± 14.1</td>
<td>0.65 ± 0.16</td>
<td>2.65 ± 1.95*</td>
</tr>
<tr>
<td>Rec40-50</td>
<td>910.7 ± 132.2</td>
<td>52.3 ± 22.4</td>
<td>13.2 ± 13.1</td>
<td>0.67 ± 0.13*</td>
<td>2.58 ± 1.71*</td>
</tr>
<tr>
<td>Rec50-60</td>
<td>912.4 ± 127.7</td>
<td>56.9 ± 24.3</td>
<td>13.9 ± 12.9</td>
<td>0.67 ± 0.13*</td>
<td>2.63 ± 1.67*</td>
</tr>
</tbody>
</table>

Values are mean ± SD. HRV = Heart rate variability. * = Diff from pre-exercise (p ≤ 0.05). † = Diff from all recovery periods (p ≤ 0.05). # = diff from Rec20-30, Rec30-40, & Rec50-60 (p ≤ 0.15).
The general effect of resistance exercise (5-RM vs. 15-RM) on forearm vascular resistance and blood flow is illustrated in Table 3.10 and Figure 3.5. The table shows the mean forearm blood flow (FBF) and vascular resistance (FVR) before and after the exercise condition. The figure demonstrates the change in vascular resistance before and after the exercise protocol. The data indicate that the 15-RM condition resulted in higher SBPs during the exercise and recovery phases compared to the 5-RM condition.

3.3. Moderating Effect of Resistance Exercise Condition (5-RM vs. 15-RM)

3.3.1. Main effect of Exercise Condition: In comparison to the 5-RM condition, the 15-RM condition resulted in higher SBPs in the assessment from pre-exercise through the exercise period (i.e., peak exercise), and in the early phase of recovery (i.e., local min and peak exercise).
local max) \((p = 0.047)\) (Figure 3.7, Table 3.11, & Extended Results Section, Table B.7), and a tendency to result in higher DBPs (Extended Results Section, Table B.12) and MAPs for the same assessment \((p = 0.11\) and \(0.057, \) respectively\) (Figures 3.6, & Table 3.11). Further, the 15-RM condition was associated with a larger magnitude of DBP drop from peak to local min \((\text{mean } \pm \text{ SD}; 15-\text{RM} = 32.4 \pm 15.3 \text{ vs. } 5-\text{RM} = 28.5 \pm 12.2 \text{ mm Hg}; \ p = 0.02)\), a tendency for a larger magnitude of DBP recovery from local min to local max \((\text{mean } \pm \text{ SD}; 15-\text{RM} = 3.34 \pm 4.5 \text{ vs. } 5-\text{RM} = 3.01 \pm 2.0; \ p = 0.08)\), and a longer time period for DBP to drop from peak exercise to local min \((\text{mean } \pm \text{ SD}; 15-\text{RM} = 20.5 \pm 1.23 \text{ vs. } 5-\text{RM} = 17.6 \pm 1.25 \text{ sec}; \ p = 0.006)\) (Extended Results Section, Table B.13).

The 5-RM exercise condition was associated with greater post-exercise SBP/peak \((p = 0.001)\), DBP/peak \((p = 0.009)\) (Extended Results Section, Table B.10, & B.15, respectively), and MAP/peak ratios \((p = 0.001)\). With regard to HR, the results of 2 x 2 ANOVA revealed that the 15-RM condition tended to result in a larger magnitude of HR increase \((p = 0.13)\) from pre- to peak-exercise (Extended Results Section, Table B.17).

3.3.2 Exercise condition x Test condition interaction: There was a significant exercise-condition by test condition interaction on MAP \((p = 0.03)\) in the long-term recovery period assessment such that peak exercise MAPs were higher than pre-exercise level and all recovery periods with both exercise conditions, but MAPs were lower than pre-exercise from R10’ \((p =0.016)\) to R60’ \((p =0.004)\) with the 5-RM condition, and only at R10’ \((p =0.07)\), R30’ \((p =0.02)\), and R60’ \((p =0.02)\) with the 15-RM condition. Further, follow up pairwise comparisons revealed that the 15-RM exercise condition resulted in higher MAPs at peak exercise \((p = 0.002)\), R15’ \((p = 0.03)\), R20’ \((p = 0.05)\), and R50’ \((p = 0.008)\) as compared to MAPs with the 5-RM condition (Figure 3.7, & Table 3.12).
Figure 3.6  Exercise Condition Effect on Arterial Blood Pressure and Heart Rate During & Immediately After RE in Women

Values are mean ± SD. Systolic (SBP) and diastolic (DBP) blood pressure, mean arterial pressure (MAP), and heart rate before (Pre-), during (Peak), and immediately after (Local min and local max) RE. * = Diff. from 5-RM condition ($p < 0.05$). # = tended to be diff. from 5-RM condition ($p < 0.15$). † / ‡ = 5- / 15-RM value diff from pre-exercise ($p < 0.05$).
Table 3.11 Exercise Condition Effect on Arterial Blood Pressure and Heart Rate Before, During, & Immediately After RE in Women

<table>
<thead>
<tr>
<th></th>
<th>SBP</th>
<th>DBP</th>
<th>MAP</th>
<th>HR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-05</td>
<td>112.3±13.3</td>
<td>58.6±9.4</td>
<td>77.1±8.5</td>
<td>68.5±11.3</td>
</tr>
<tr>
<td>Pre-15</td>
<td>114.7±14.9</td>
<td>59.5±8.8</td>
<td>78.2±9.4</td>
<td>66.6±10.8</td>
</tr>
<tr>
<td>Peak-05</td>
<td>142.1±16.2†+</td>
<td>82.6±14.9†+</td>
<td>102.7±12.9†+</td>
<td>103.8±19.0†+</td>
</tr>
<tr>
<td>Peak-15</td>
<td>156.0±25.0‡*+</td>
<td>90.2±15.3‡#+</td>
<td>112.3±15.7‡†+</td>
<td>106.9±21.6‡#+</td>
</tr>
<tr>
<td>L. min-05</td>
<td>120.1±11.8†</td>
<td>54.1±10.9†</td>
<td>76.1±9.2</td>
<td>76.0±16.0†</td>
</tr>
<tr>
<td>L. min-15</td>
<td>127.5±22.8‡*</td>
<td>57.8±13.8‡#</td>
<td>80.2±15.4‡#</td>
<td>79.7±20.6‡</td>
</tr>
<tr>
<td>L. max-05</td>
<td>123.5±11.6†</td>
<td>57.2±10.3</td>
<td>77.7±10.1</td>
<td>79.6±15.6†</td>
</tr>
<tr>
<td>L. max-15</td>
<td>130.5±22.8‡*</td>
<td>61.1±13.5‡#</td>
<td>83.3±14.9‡#</td>
<td>83.7±20.8‡</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Systolic (SBP) and diastolic (DBP) blood pressure, mean arterial pressure (MAP), and heart rate (HR) before (Pre-), during (Peak), and immediately after (Local min and local max) resistance exercise. * = Diff from 5-RM condition ($p < 0.05$). # = tended to be diff from 5-RM condition ($p < 0.15$). † / ‡ = 5- / 15-RM values diff from pre-exercise ($p < 0.05$). + = diff all recovery periods ($p < 0.05$).

The data for SBP and DBP were similar, but the interaction terms did not quite reach statistical significance ($p = 0.06$, and 0.06, respectively). Follow-up pairwise comparisons revealed differences between pre-exercise and post-exercise values by exercise condition as indicated in Figure 3.7 and Table 3.12.

With regard to HRV, there was an exercise condition by test condition interaction effect on LFnu (10-minute segments) following RE ($p = 0.04$) indicating that the 15-RM exercise condition was associated with a lower than pre-exercise LFnu in the 10-minute segment following the activity (R0-10); as well as higher than pre-exercise LFnu in the 10-minute segments at R20-30 ($p = 0.03$), R40-50 ($p = 0.01$), and R50-60 ($p = 0.03$).
Figure 3.7 Exercise Condition Effect on Arterial Blood Pressure and Heart Rate Before, During, & For 1 Hour After RE in Women
Values are mean ± SD. Systolic (SBP) and diastolic (DBP) blood pressure, mean arterial pressure (MAP), and heart rate (HR) before (Pre-), during (Peak), and for 1 hour after RE. * = Diff from 5-RM condition ($p < 0.05$). # = tended to be diff from 5-RM condition ($p < 0.15$). † / ‡ = Post-exercise 5- / 15-RM values diff from pre-exercise ($p < 0.05$).
Table 3.12  Exercise Condition Effect on Arterial BP and HR Before, During, & Immediately After RE in Women

<table>
<thead>
<tr>
<th></th>
<th>SBP</th>
<th>DBP</th>
<th>MAP</th>
<th>HR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-05</td>
<td>112.3 ±13.3</td>
<td>58.6 ± 9.4</td>
<td>77.1 ± 8.5</td>
<td>68.5 ±11.3</td>
</tr>
<tr>
<td>Pre-15</td>
<td>114.7 ±14.9</td>
<td>59.5 ± 8.8</td>
<td>78.2 ± 9.4</td>
<td>66.6 ±10.8</td>
</tr>
<tr>
<td>Peak-05</td>
<td>142.1 ±16.2†</td>
<td>82.6 ±14.9†</td>
<td>102.7 ±12.9†</td>
<td>103.8 ±19.0†</td>
</tr>
<tr>
<td>Peak-15</td>
<td>156.0 ±25.0‡*</td>
<td>90.2 ±15.3‡*</td>
<td>112.3 ±15.7‡*</td>
<td>106.9 ±21.6‡*</td>
</tr>
<tr>
<td>R1’05</td>
<td>118.9 ±10.1†</td>
<td>56.4 ±11.1</td>
<td>77.6 ± 8.9</td>
<td>73.6 ±12.5</td>
</tr>
<tr>
<td>R1’-15</td>
<td>121.5 ±21.3‡</td>
<td>56.4 ±12.5</td>
<td>78.7 ±13.7</td>
<td>76.0 ±13.9</td>
</tr>
<tr>
<td>R3’-05</td>
<td>115.4 ±10.1</td>
<td>55.5 ±10.8†</td>
<td>75.8 ± 9.3</td>
<td>67.5 ±12.7</td>
</tr>
<tr>
<td>R3’-15</td>
<td>117.9 ±19.9</td>
<td>55.7 ±12.2‡</td>
<td>77.0 ±13.1</td>
<td>68.1 ±11.6</td>
</tr>
<tr>
<td>R5’-05</td>
<td>114.3 ±10.8</td>
<td>55.3 ±11.1†</td>
<td>75.2 ± 9.3</td>
<td>66.1 ±10.5</td>
</tr>
<tr>
<td>R5’-15</td>
<td>113.6 ±18.0</td>
<td>55.0 ±11.9</td>
<td>75.1 ±12.4</td>
<td>66.9 ±12.8</td>
</tr>
<tr>
<td>R10’-05</td>
<td>110.0 ±12.3</td>
<td>53.9 ±11.7†</td>
<td>72.7 ±10.6†</td>
<td>66.0 ± 9.4</td>
</tr>
<tr>
<td>R10’-15</td>
<td>109.2 ±14.3</td>
<td>53.3 ± 9.9</td>
<td>72.7 ± 9.3</td>
<td>66.0 ±10.3</td>
</tr>
<tr>
<td>R15’-05</td>
<td>107.4 ±13.6</td>
<td>52.5 ±10.4†</td>
<td>70.9 ± 9.8†</td>
<td>68.6 ±12.1</td>
</tr>
<tr>
<td>R15’-15</td>
<td>111.4 ±14.0‡</td>
<td>56.5 ± 9.8‡</td>
<td>75.9 ± 9.2</td>
<td>66.5 ±10.7</td>
</tr>
<tr>
<td>R20’-05</td>
<td>108.7 ±13.5</td>
<td>52.3 ±10.7†</td>
<td>71.6 ± 9.3†</td>
<td>68.7 ±10.7</td>
</tr>
<tr>
<td>R20’-15</td>
<td>113.1 ±16.0‡</td>
<td>56.4 ± 9.6‡</td>
<td>76.3 ±10.2‡</td>
<td>66.5 ±10.1</td>
</tr>
<tr>
<td>R30’-05</td>
<td>110.0 ±12.6</td>
<td>52.2 ±10.1†</td>
<td>71.7 ± 8.7†</td>
<td>68.6 ±10.8</td>
</tr>
<tr>
<td>R30’-15</td>
<td>110.5 ±16.3</td>
<td>53.8 ±10.8‡</td>
<td>73.7 ±11.2‡</td>
<td>65.7 ±10.6</td>
</tr>
<tr>
<td>R40’-05</td>
<td>108.7 ±13.1</td>
<td>53.2 ± 9.6†</td>
<td>72.3 ± 8.7†</td>
<td>68.6 ± 9.4</td>
</tr>
<tr>
<td>R40’-15</td>
<td>112.7 ±16.5</td>
<td>54.8 ±12.5‡</td>
<td>74.8 ±12.6</td>
<td>66.9 ±10.8</td>
</tr>
<tr>
<td>R50’-05</td>
<td>107.8 ±13.4</td>
<td>50.6 ±10.2†</td>
<td>69.5 ± 8.7†</td>
<td>66.4 ±9.2</td>
</tr>
<tr>
<td>R50’-15</td>
<td>112.7 ±14.9‡</td>
<td>54.9 ±10.5‡</td>
<td>75.1 ±10.8</td>
<td>66.5 ±10.5</td>
</tr>
<tr>
<td>R60’-05</td>
<td>108.4 ±14.0</td>
<td>51.9 ±10.8†</td>
<td>70.7 ± 9.5†</td>
<td>69.3 ±11.8</td>
</tr>
<tr>
<td>R60’-15</td>
<td>109.4 ±17.0</td>
<td>53.9 ±11.1‡</td>
<td>73.4 ±10.9‡</td>
<td>68.1 ±13.4</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Blood pressure (BP) and heart rate (HR) before (Pre-), during (Peak), and immediately after (Local min and local max) resistance exercise (RE). SBP = Systolic blood pressure. DBP = Diastolic blood pressure. HR = Heart rate. MAP = Mean arterial blood pressure. * = Diff. from 5-RM condition (\(p < 0.05\)). # = tended to be diff from 5-RM condition. † / ‡ = Post-exercise 5-RM / 15-RM value diff from pre-exercise.
Conversely, the 5-RM condition was associated only with a higher than pre-exercise LFnu at R50-60 ($p = 0.04$) (Figure 3.8-3.9, & Tables 3.13-3.14). There was also trends towards exercise condition by test condition interaction effect on LFnu ($p = 0.19$) and LF/HF ratio ($p = 0.15$) in the assessment before and 1-hour after RE using 5-minute segments. Follow-up pairwise comparisons revealed differences between pre-exercise and post-exercise values by exercise condition as indicated in the Table 3.13. On the other hand, there was neither main effect of exercise condition nor exercise condition by test condition interaction effect FVR data (Figure 3.10, & Table 3.15).

**Figure 3.8 Exercise Condition Effect on LFnu (5-minute segment) Before & Immediately After Exercise in Women**
Values are mean ± SD. LFnu (5-minute segments) by exercise condition before (Pre-) and immediately after RE. * = diff from 5-RM condition. † / ‡ = Post-exercise 5- / 15-RM value diff from pre-exercise ($p \leq 0.05$). # = diff from all recovery periods ($p \leq 0.05$).
Table 3.13  Exercise Condition Effect on HRV Indices (5-minute segment) Before and Immediately After RE in Women

<table>
<thead>
<tr>
<th>Condition</th>
<th>R-R Interval</th>
<th>SDNN</th>
<th>Pnn50</th>
<th>LFnu</th>
<th>LF/HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre.05</td>
<td>911.2 ± 142.8</td>
<td>47.6 ± 22.8</td>
<td>12.4 ± 14.8</td>
<td>0.60 ± 0.16</td>
<td>2.05 ± 1.67</td>
</tr>
<tr>
<td>Pre.15</td>
<td>915.6 ± 137.0</td>
<td>50.6 ± 27.2</td>
<td>15.2 ± 18.0</td>
<td>0.59 ± 0.17</td>
<td>1.89 ± 1.34</td>
</tr>
<tr>
<td>Rec0-5.05</td>
<td>890.9 ± 135.9 †#</td>
<td>83.7 ± 29.2 †#</td>
<td>16.6 ± 18.0 †</td>
<td>0.64 ± 0.13 †#</td>
<td>2.34 ± 1.62 †#</td>
</tr>
<tr>
<td>Rec0-5.15</td>
<td>857.6 ± 130.0 †‡</td>
<td>84.7 ± 39.0 † §</td>
<td>18.5 ± 18.7 †</td>
<td>0.58 ± 0.12*</td>
<td>1.57 ± 0.89*</td>
</tr>
<tr>
<td>Rec1-6.05</td>
<td>914.8 ± 137.2 †</td>
<td>63.7 ± 26.5 †#</td>
<td>18.2 ± 18.4 †</td>
<td>0.56 ± 0.16</td>
<td>1.80 ± 1.60</td>
</tr>
<tr>
<td>Rec1-6.15</td>
<td>884.6 ± 131.5 †‡</td>
<td>71.7 ± 32.5 †‡#</td>
<td>18.8 ± 20.4 †‡</td>
<td>0.55 ± 0.16‡</td>
<td>1.46 ± 0.93‡</td>
</tr>
<tr>
<td>Rec2-7.05</td>
<td>927.5 ± 140.5 †</td>
<td>53.8 ± 22.7 †‡</td>
<td>17.3 ± 17.7 †</td>
<td>0.57 ± 0.16</td>
<td>1.77 ± 1.40</td>
</tr>
<tr>
<td>Rec2-7.15</td>
<td>906.5 ± 133.9 †</td>
<td>58.4 ± 28.2 †‡</td>
<td>19.1 ± 21.0 †‡</td>
<td>0.54 ± 0.15‡</td>
<td>1.48 ± 0.48‡</td>
</tr>
<tr>
<td>Rec3-8.05</td>
<td>928.4 ± 139.6 †</td>
<td>49.3 ± 23.3 †‡</td>
<td>15.3 ± 16.4 †</td>
<td>0.57 ± 0.17</td>
<td>1.81 ± 1.43</td>
</tr>
<tr>
<td>Rec3-8.15</td>
<td>916.2 ± 132.7 †</td>
<td>51.3 ± 29.0 †‡</td>
<td>17.5 ± 19.4 †</td>
<td>0.53 ± 0.16‡</td>
<td>1.53 ± 1.14‡</td>
</tr>
<tr>
<td>Rec4-9.05</td>
<td>923.6 ± 140.4 †</td>
<td>48.2 ± 24.5 †‡</td>
<td>13.8 ± 15.4 †‡#</td>
<td>0.60 ± 0.15</td>
<td>1.91 ± 1.27</td>
</tr>
<tr>
<td>Rec4-9.15</td>
<td>915.8 ± 131.8 †</td>
<td>48.8 ± 28.3 †‡</td>
<td>15.9 ± 19.1 † #</td>
<td>0.55 ± 0.15‡</td>
<td>1.64 ± 1.36</td>
</tr>
<tr>
<td>Rec5-10.05</td>
<td>920.3 ± 139.3 †</td>
<td>47.8 ± 24.2 †‡</td>
<td>12.6 ± 14.6 †#</td>
<td>0.61 ± 0.13</td>
<td>1.94 ± 1.24</td>
</tr>
<tr>
<td>Rec5-10.15</td>
<td>913.0 ± 132.6 48.0 ± 27.0</td>
<td>14.9 ± 18.3 †#</td>
<td>0.58 ± 0.15</td>
<td>1.80 ± 1.45</td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ± SD. HRV = Heart rate variability. * = Diff from 5-RM condition ($p \leq 0.05$). † / ‡ = Post-exercise 5-RM / 15-RM values diff from pre-exercise level ($p \leq 0.05$). # = diff from all recovery periods ($p = \leq 0.05$).

Figure 3.9  Exercise Condition Effect on LFnu (10-minute segment) Before & For 1 Hour After Exercise in Women

Values are mean ± SD. LFnu (10-minute segments) before (Pre-) and for 1 hour after RE. † / ‡ = Post-exercise 5- / 15-RM values diff from pre-exercise level ($p \leq 0.05$). # = diff from all recovery periods ($p = \leq 0.05$)
### Table 3.14  Exercise Condition Effect on HRV Indices (10-minute segment) Before and For 1-hour After RE in Women

<table>
<thead>
<tr>
<th>Time Period</th>
<th>R-R Interval</th>
<th>SDNN</th>
<th>Pnn50</th>
<th>LFnu</th>
<th>LF/HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre.05</td>
<td>903.8 ±141.8</td>
<td>56.6 ±25.3</td>
<td>13.5 ±15.8</td>
<td>0.63 ±0.14</td>
<td>2.30 ±1.74</td>
</tr>
<tr>
<td>Pre.15</td>
<td>902.8 ±132.8</td>
<td>58.5 ±24.1</td>
<td>13.7 ±15.0</td>
<td>0.62 ±0.16</td>
<td>2.18 ±1.52</td>
</tr>
<tr>
<td>Rec0-10.05</td>
<td>905.5 ±138.0</td>
<td>72.0 ±25.6 †#</td>
<td>16.2 ±16.8†</td>
<td>0.64 ±0.12*</td>
<td>2.19 ±1.33*</td>
</tr>
<tr>
<td>Rec0-10.15</td>
<td>883.1 ±132.4‡#</td>
<td>78.0 ±31.0*‡#</td>
<td>15.8 ±16.0</td>
<td>0.58 ±0.13‡#</td>
<td>1.61 ±0.91‡#</td>
</tr>
<tr>
<td>Rec10-20.05</td>
<td>916.1 ±145.6</td>
<td>49.6 ±22.7†</td>
<td>13.3 ±16.7</td>
<td>0.64 ±0.14</td>
<td>2.37 ±1.66</td>
</tr>
<tr>
<td>Rec10-20.15</td>
<td>906.0 ±137.1</td>
<td>47.7 ±20.1‡</td>
<td>11.3 ±13.5</td>
<td>0.63 ±0.14</td>
<td>2.25 ±1.61</td>
</tr>
<tr>
<td>Rec20-30.05</td>
<td>909.2 ±130.3</td>
<td>52.3 ±24.3</td>
<td>13.4 ±14.5</td>
<td>0.63 ±0.16</td>
<td>2.27 ±1.48</td>
</tr>
<tr>
<td>Rec20-30.15</td>
<td>903.0 ±141.7</td>
<td>51.6 ±20.4</td>
<td>11.9 ±13.1</td>
<td>0.65 ±0.14‡</td>
<td>2.41 ±1.54</td>
</tr>
<tr>
<td>Rec30-40.05</td>
<td>913.8 ±134.7</td>
<td>56.2 ±24.7</td>
<td>15.1 ±16.5</td>
<td>0.66 ±0.15</td>
<td>2.66 ±1.97</td>
</tr>
<tr>
<td>Rec30-40.15</td>
<td>904.3 ±133.2</td>
<td>52.0 ±25.4</td>
<td>11.1 ±10.5</td>
<td>0.64 ±0.17</td>
<td>2.63 ±1.97</td>
</tr>
<tr>
<td>Rec40-50.05</td>
<td>915.5 ±131.7</td>
<td>53.7 ±24.3</td>
<td>13.5 ±14.0</td>
<td>0.66 ±0.15</td>
<td>2.71 ±2.01</td>
</tr>
<tr>
<td>Rec40-50.15</td>
<td>905.6 ±134.7</td>
<td>52.1 ±20.5</td>
<td>13.0 ±12.2</td>
<td>0.67 ±0.12‡</td>
<td>2.43 ±1.30</td>
</tr>
<tr>
<td>Rec50-60.05</td>
<td>922.4 ±130.6†</td>
<td>58.2 ±25.5</td>
<td>14.6 ±13.6</td>
<td>0.68 ±0.13†</td>
<td>2.72 ±1.89†</td>
</tr>
<tr>
<td>Rec50-60.15</td>
<td>901.7 ±125.8+</td>
<td>55.4 ±23.2</td>
<td>13.2 ±12.4</td>
<td>0.67 ±0.14‡</td>
<td>2.53 ±1.41</td>
</tr>
</tbody>
</table>

Values are mean ± SD. HRV = Heart rate variability. * = Diff from 5-RM condition (p < 0.05). † / ‡ = Post-exercise 5-RM / 15-RM values diff from pre-exercise level (p < 0.05). # = diff from all recovery periods (p = < 0.05).

**Figure 3.10 Exercise Condition Effect on Forearm Vascular Resistance Before & For 1 Hour After RE in Women**

Values are mean ± SD. Forearm vascular resistance (FVR) before exercise (Pre-) and for 1 hour after RE. † / ‡ = Post-exercise 5-RM / 15-RM values diff from pre-exercise level (p < 0.05).
Table 3.15  Exercise Condition Effect on Forearm Blood Flow and Vascular Resistance Before and For 1 hour After RE in Women

<table>
<thead>
<tr>
<th></th>
<th>FBF (mL/100mL/min)</th>
<th>FVR (Units)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5RM</td>
<td>15RM</td>
</tr>
<tr>
<td>Pre-</td>
<td>1.99 ± 0.50</td>
<td>1.91 ± 0.61</td>
</tr>
<tr>
<td>R4'</td>
<td>1.75 ± 0.56</td>
<td>1.75 ± 0.75</td>
</tr>
<tr>
<td>R7'</td>
<td>1.83 ± 0.78</td>
<td>1.86 ± 0.85</td>
</tr>
<tr>
<td>R10'</td>
<td>1.76 ± 0.58</td>
<td>1.56 ± 0.45</td>
</tr>
<tr>
<td>R15'</td>
<td>1.66 ± 0.70</td>
<td>1.65 ± 0.65</td>
</tr>
<tr>
<td>R20'</td>
<td>1.58 ± 0.58</td>
<td>1.72 ± 0.67</td>
</tr>
<tr>
<td>R30'</td>
<td>1.58 ± 0.56</td>
<td>2.07 ± 0.84</td>
</tr>
<tr>
<td>R40'</td>
<td>1.58 ± 0.79</td>
<td>1.75 ± 0.97</td>
</tr>
<tr>
<td>R50'</td>
<td>1.57 ± 0.55</td>
<td>1.62 ± 0.56</td>
</tr>
<tr>
<td>R60'</td>
<td>1.55 ± 0.56</td>
<td>1.55 ± 0.57</td>
</tr>
</tbody>
</table>

Values are mean ± SD. FBF = Forearm blood inflow (FVR) and forearm vascular resistance (FVR) before exercise (Pre-) and for 1-hour after RE. † / ‡ = Post-exercise 5-RM / 15-RM values diff from pre-exercise level ($p < 0.05$).

3.4. Moderating Effect of Age

3.4.1. Main effect of Age. Systolic blood pressure was higher among the older women at all testing points in both short-term ($p = 0.0001$) and long-term recovery period assessments ($p = 0.0001$) (Figure 3.11-3.12, & Table 3.16-3.17). Interestingly, during the early phase of recovery, the magnitude of SBP recovery from local min to local max tended to be smaller among the older women (mean ± SD; young-group = 3.73 ± 4.3 vs. older-group = 2.70 ± 3.5 mm Hg; $p = 0.056$) (Extended Results Section, Table B.7) and, during the long-term recovery period, SBP dropped below pre-exercise level from R10' ($p = 0.006$) to R60' ($p = 0.0001$) among the older women, but not among the young group ($p = 0.02$) (Figure 3.12, and Table 3.17). There was also a tendency ($p = 0.13$) for greater 1’ and 3’ post-exercise SBP/peak ratios among the older women (Extended Results Section, Table B.10).
Figure 3.11 Age Group Effect on Arterial Blood Pressure and Heart Rate
Before, During & Immediately After RE in Women
Values are mean ± SD. Blood pressure and heart rate before exercise (Pre-), during (Peak), and immediately after (Local min and local max) RE. SBP = Systolic blood pressure. DBP = Diastolic Blood Pressure. HR = Heart Rate. * = Different from young group \( (p \leq 0.05) \). † / ‡ = Young / Old values diff from pre-exercise \( (p \leq 0.05) \).
Table 3.16  Age Group Effect on Arterial BP and HR Before, During, & Immediately After RE in Women

<table>
<thead>
<tr>
<th></th>
<th>SBP</th>
<th>DBP</th>
<th>MAP</th>
<th>HR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-YA</td>
<td>104.7 ± 7.7</td>
<td>59.1 ± 9.2</td>
<td>75.1 ± 8.1</td>
<td>71.9 ± 11.8</td>
</tr>
<tr>
<td>Pre-OA</td>
<td>122.6 ± 13.4*</td>
<td>59.0 ± 9.0</td>
<td>80.2 ± 9.2</td>
<td>63.1 ± 8.1*</td>
</tr>
<tr>
<td>Peak-YA</td>
<td>142.7 ± 25.0 †</td>
<td>91.4 ± 14.6 †</td>
<td>108.8 ± 15.3 †</td>
<td>121.0 ± 13.8 †</td>
</tr>
<tr>
<td>Peak-OA</td>
<td>155.7 ± 20.6 ‡*</td>
<td>81.3 ± 14.8 ‡*</td>
<td>106.4 ± 15.1 ‡*</td>
<td>89.2 ± 11.1 ‡*</td>
</tr>
<tr>
<td>Local min-YA</td>
<td>114.4 ± 15.0 †</td>
<td>54.6 ± 11.0 †</td>
<td>74.5 ± 11.2</td>
<td>87.1 ± 20.1†</td>
</tr>
<tr>
<td>Local min-OA</td>
<td>133.7 ± 16.6 ‡*</td>
<td>57.5 ± 13.9</td>
<td>82.0 ± 13.5 *</td>
<td>68.4 ± 9.8 ‡*</td>
</tr>
<tr>
<td>Local max-YA</td>
<td>118.1 ± 14.9 †</td>
<td>58.6 ± 10.4</td>
<td>77.0 ± 12.1</td>
<td>92.5 ± 18.6 †</td>
</tr>
<tr>
<td>Local max-OA</td>
<td>136.3 ± 17.0 ‡*</td>
<td>59.8 ± 13.8</td>
<td>84.2 ± 13.1 ‡*</td>
<td>70.4 ± 9.3 ‡*</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Systolic (SBP) and diastolic (DBP) blood pressure, mean arterial pressure (MAP), and heart rate (HR) before (Pre-), during (Peak), and immediately after (Local min and local max) resistance exercise. YA = Young adult. OA = Older Adult. * = Diff from young group ($p < 0.05$). † / ‡ = Young / Old value diff from pre-exercise ($p < 0.15$).

In contrast, there were no age group differences in DBP at pre-exercise condition, peak exercise or in the early phase of recovery (Figure 3.11, & Table 3.16). However, DBPs were lower among the older women throughout the 1-hour recovery period ($p = 0.05$) (Figure 3.12 and Table 3.17). The magnitude of DBP drop from peak to local min was smaller among the older women (mean ± SD; young-group = 36.9 ± 12.9 vs. older-group = 23.8 ± 11.8 mm Hg; $p = 0.007$), which paired with a longer time for older women to reach local min (mean ± SD; young-group = 18.1 ± 4.6 vs. older-group = 20.0 ± 8.6 sec; $p = 0.009$) resulted in a tendency for a slower mean rate of DBP decline from peak to local min for this age group (mean ± SD; young-group = 2.1 ± 0.8 vs. older-group = 1.2 ± 0.6 mmHg/sec; $p = 0.07$) (Extended Results Section, Table B.12-B.13). Further, the magnitude of DBP recovery from local min to local max was also smaller (mean ± SD; young-group = 4.0 ± 4.4 vs. older-group = 2.3 ± 1.8 mmHg; $p = 0.02$) among the older women (Extended Results Section, Table B.12), but the magnitude of
Figure 3.12 Age Group Effect on Arterial Blood Pressure and Heart Rate Before, During & For 1 Hour After RE in Women

Values are mean ± SD. Blood Pressure and heart rate before (Pre-), during (Peak), and for 1 hour after RE. SBP = Systolic Blood Pressure. DBP = Diastolic Blood Pressure. MAP = Mean arterial pressure. HR = Heart Rate. * = Different from young group (p ≤ 0.05). † / ‡ = Young / Older adult value diff from pre-exercise (p ≤ 0.05).
### Table 3.17 Age Group Effect on Arterial Blood Pressure and Heart Rate Before, During, & For 1 Hour After RE in Women

<table>
<thead>
<tr>
<th>Age Group</th>
<th>SBP</th>
<th>DBP</th>
<th>MAP</th>
<th>HR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-YA</td>
<td>104.7±7.7</td>
<td>59.1±9.2</td>
<td>75.1±8.0</td>
<td>71.9±11.8</td>
</tr>
<tr>
<td>Pre-OA</td>
<td>122.6±13.4 *</td>
<td>59.0±9.0</td>
<td>80.2±9.2 *</td>
<td>63.1±8.1 *</td>
</tr>
<tr>
<td>Peak-YA</td>
<td>142.7±20.6 †</td>
<td>91.4±14.6 †</td>
<td>108.8±15.3 †</td>
<td>121.0±13.8 †</td>
</tr>
<tr>
<td>Peak-OA</td>
<td>155.7±22.0 ‡*</td>
<td>81.3±14.8 ‡</td>
<td>106.4±15.1 ‡</td>
<td>89.2±11.1 ‡*</td>
</tr>
<tr>
<td>R1’-YA</td>
<td>113.2±13.4 †</td>
<td>57.3±11.1</td>
<td>76.9±10.6</td>
<td>82.1±12.9 †</td>
</tr>
<tr>
<td>R1’-OA</td>
<td>127.5±16.8 ‡*</td>
<td>55.4±12.5 ‡</td>
<td>79.5±12.4</td>
<td>67.3±8.5 ‡*</td>
</tr>
<tr>
<td>R3’-YA</td>
<td>109.9±13.2 †</td>
<td>56.6±11.1 †</td>
<td>75.0±10.6</td>
<td>72.0±13.1</td>
</tr>
<tr>
<td>R3’-OA</td>
<td>123.6±15.3 *</td>
<td>54.6±11.9 ‡</td>
<td>77.8±12.0</td>
<td>63.5±9.2 *</td>
</tr>
<tr>
<td>R5’-YA</td>
<td>108.3±11.2</td>
<td>56.0±10.3 †</td>
<td>74.3±9.1</td>
<td>70.7±12.8</td>
</tr>
<tr>
<td>R5’-OA</td>
<td>119.8±15.8 *</td>
<td>54.3±12.6 ‡</td>
<td>76.1±12.6 ‡</td>
<td>62.2±8.6 *</td>
</tr>
<tr>
<td>R10’-YA</td>
<td>104.8±10.9</td>
<td>55.7±11.0 †</td>
<td>72.9±9.8</td>
<td>70.1±9.5</td>
</tr>
<tr>
<td>R10’-OA</td>
<td>114.6±13.8 ‡*</td>
<td>51.4±10.2 ‡</td>
<td>72.5±10.2 ‡</td>
<td>61.7±8.1 ‡*</td>
</tr>
<tr>
<td>R15’-YA</td>
<td>103.8±12.1</td>
<td>57.2±10.4</td>
<td>73.9±10.2</td>
<td>71.8±11.7</td>
</tr>
<tr>
<td>R15’-OA</td>
<td>115.2±13.2 ‡*</td>
<td>51.7±9.4 ‡*</td>
<td>72.8±9.4 ‡*</td>
<td>63.1±9.3 *</td>
</tr>
<tr>
<td>R20’-YA</td>
<td>105.1±10.9</td>
<td>56.9±10.0</td>
<td>74.5±9.2</td>
<td>72.2±10.3</td>
</tr>
<tr>
<td>R20’-OA</td>
<td>117.0±16.1 ‡*</td>
<td>51.8±10.1 ‡</td>
<td>73.5±10.8 ‡</td>
<td>62.8±8.0 *</td>
</tr>
<tr>
<td>R30’-YA</td>
<td>103.9±11.7</td>
<td>55.3±10.6 †</td>
<td>72.7±10.5</td>
<td>71.6±11.0</td>
</tr>
<tr>
<td>R30’-OA</td>
<td>116.8±14.3 ‡*</td>
<td>50.7±9.7 ‡*</td>
<td>72.7±9.7 ‡*</td>
<td>62.5±8.3 *</td>
</tr>
<tr>
<td>R40’-YA</td>
<td>105.3±12.2</td>
<td>57.0±10.2</td>
<td>74.4±10.2</td>
<td>73.1±8.8</td>
</tr>
<tr>
<td>R40’-OA</td>
<td>116.3±15.6 ‡*</td>
<td>50.9±11.4 ‡*</td>
<td>72.9±11.7 ‡*</td>
<td>62.2±8.2 ‡*</td>
</tr>
<tr>
<td>R50’-YA</td>
<td>105.4±10.8</td>
<td>55.6±9.7 †</td>
<td>73.2±9.6</td>
<td>71.0±9.3</td>
</tr>
<tr>
<td>R50’-OA</td>
<td>115.4±15.8 ‡*</td>
<td>49.9±10.6 ‡*</td>
<td>71.4±10.7 ‡*</td>
<td>61.8±8.0 ‡*</td>
</tr>
<tr>
<td>R60’-YA</td>
<td>104.7±13.4</td>
<td>56.4±11.7</td>
<td>73.7±10.8</td>
<td>75.0±13.2</td>
</tr>
<tr>
<td>R60’-OA</td>
<td>113.2±16.4 ‡*</td>
<td>49.3±8.9 ‡*</td>
<td>70.5±9.5 ‡*</td>
<td>62.1±7.6 ‡*</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Systolic (SBP) and diastolic (DBP) blood pressure, mean arterial pressure (MAP), and heart rate (HR) before (Pre-), during (Peak), and for 1 hour RE. * = Diff. from young group ($p < 0.05$). † / ‡ = Young / Old value diff from pre-exercise ($p < 0.05$).

Post-exercise DBP drop throughout the recovery period was larger ($p = 0.05$) (Figure 3.12, and Table 3.17), as were the 1’ and 3’ post-exercise DBP/peak ratios ($p = 0.01$) (Extended Results Section, Table B.15). MAP behaved nearly identical to DBP.

There was also a main effect of age group on HR, indicating lower HRs among the older women at all testing points in both short-term ($p = 0.0001$) (Figure 3.11, and Table
3.16) and long-term recovery period assessments ($p = 0.0001$) (Figure 3.12 and Table 3.17). There was also a smaller magnitude of HR increase from pre-exercise to peak exercise ($p = 0.036$) and a tendency for smaller and slower magnitudes of HR decline from peak to local min among the older women ($p = 0.058$ and $0.11$, respectively) (Extended Results Section, Table B.17- B.18).

There was a trend towards a smaller magnitude of HR recovery from local min to local max among the older women ($p = 0.15$), and thus a slower mean rate of recovery ($p = 0.03$) (Extended Results Section. Table B.18). The post-exercise drop in HR also was smaller throughout the entire recovery among the older women ($p = 0.01$). Further, there were significant effects of age group on HRV data after RE. The older women had a smaller SDNN ($p = 0.0001$) and pnn50 ($p = 0.0001$) in comparison to the young group (Extended Results Section, Table B.2-B.3, Figure B.11-B.12, & B.16-B17, respectively). Moreover, there was a significant main effect of age group on FVR ($p = 0.007$) indicating that young women had higher post-exercise FVR in comparison to the older women (Figure 3.13). Follow-up pairwise comparisons revealed that post-exercise FVRs were higher than pre-exercise within the young women, but not in older women (Figure 3.13, & Table 3.18).

### 3.4.2. Age by Test Condition Interaction

There was a significant age group by test condition interaction effect on DBP ($p = 0.001$) such that older women exhibited a smaller increase from pre- to peak exercise ($p = 0.004$), and a smaller drop from peak to local min ($p = 0.0001$) (Extended Results Section, Table B.12). Consequently, older women exhibited lower DBP at peak exercise ($p = 0.009$), but similar local min, and local max (Figure 3.11, and Table 3.16). Further, there was a nearly significant interaction term
Figure 3.13 Age Group Effect on Forearm Vascular Resistance Before & For 1 Hour After RE in Women

Values are mean ± SD. Forearm vascular resistance (FVR) before (Pre-) and for 1 hour after RE. OA = older adult. YA = young adult. * = Diff from young group ($p < 0.05$). †= YA post-exercise value diff from pre-exercise ($p < 0.05$).

Table 3.18 Age Group Effect on Forearm Blood Flow and Vascular Resistance Before and For 1 hour After RE in Women

<table>
<thead>
<tr>
<th>Time</th>
<th>FBF (mL/100mL/min) Young</th>
<th>FBF (mL/100mL/min) Old</th>
<th>FVR (Units) Young</th>
<th>FVR (Units) Old</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-</td>
<td>1.88 ± 0.41</td>
<td>2.02 ± 0.67</td>
<td>39.4 ± 8.1</td>
<td>40.8 ± 10.2</td>
</tr>
<tr>
<td>R4'</td>
<td>1.54 ± 0.56</td>
<td>1.95 ± 0.69</td>
<td>54.1 ± 22.5 †</td>
<td>42.5 ± 14.9 *</td>
</tr>
<tr>
<td>R7'</td>
<td>1.76 ± 0.99</td>
<td>1.92 ± 0.59</td>
<td>50.7 ± 21.2 †</td>
<td>40.3 ± 12.0 *</td>
</tr>
<tr>
<td>R10'</td>
<td>1.49 ± 0.50</td>
<td>1.83 ± 0.50</td>
<td>52.5 ± 17.6 †</td>
<td>42.3 ± 13.5 *</td>
</tr>
<tr>
<td>R15'</td>
<td>1.49 ± 0.65</td>
<td>1.82 ± 0.66</td>
<td>56.1 ± 25.3 †</td>
<td>44.7 ± 18.3 *</td>
</tr>
<tr>
<td>R20’</td>
<td>1.57 ± 0.70</td>
<td>1.73 ± 0.55</td>
<td>54.1 ± 25.2 †</td>
<td>44.7 ± 13.3 *</td>
</tr>
<tr>
<td>R30’</td>
<td>1.60 ± 0.72</td>
<td>2.05 ± 0.71</td>
<td>55.1 ± 20.3 †</td>
<td>37.3 ± 11.9 *</td>
</tr>
<tr>
<td>R40’</td>
<td>1.70 ± 0.65</td>
<td>1.57 ± 0.52</td>
<td>56.1 ± 24.1 †</td>
<td>46.8 ± 12.8 *</td>
</tr>
<tr>
<td>R50’</td>
<td>1.46 ± 0.60</td>
<td>1.73 ± 0.47</td>
<td>57.2 ± 23.4 †</td>
<td>43.5 ± 13.2 *</td>
</tr>
<tr>
<td>R60’</td>
<td>1.50 ± 0.69</td>
<td>1.60 ± 0.42</td>
<td>59.3 ± 28.2 †</td>
<td>44.3 ± 15.4 *</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Forearm blood inflow (FBF) and forearm vascular resistance (FVR) before (Pre-) and for 1-hour after RE. * = Diff from young group ($p < 0.05$). †= Young adult post-exercise value diff from pre-exercise ($p < 0.05$).
for DBP throughout the 1-hour recovery period ($p = 0.13$). Follow-up pairwise comparison revealed that DBP among the older women dropped earlier (Old = R1’ vs. young = R3’) and further below pre-exercise level, with DBPs at time from R15’ to R60’ being lower among the older women as compared to their younger counterparts. MAP behaved nearly identical to DBP (Figure 3.12 and Table 3.17).

ANOVA revealed significant age group by test condition interaction effects on HR ($p = 0.0001$), in addition to the main effect of age revealing lower HRs in older women throughout the testing period ($p = 0.0001$) in both short-, and long-term assessments. Follow-up pairwise comparisons revealed that the older group had lower than pre-exercise post-exercise HRs at R10’, R40’, R50’, and R60’ (Figure 3.12 and Table 3.17).

With regard to HRV data, there were significant age group by test condition interaction effects on pnn50 ($p = 0.001$), LFnu ($p = 0.02$), and LF/HF ratio ($p = 0.01$). Among the young group, LFnu and LF/HF ratio were lower than pre-exercise in the 5-minute segments beginning 1 minute after exercise (R1-6, R2-7, and R3-8), and thereafter tended to increase slightly throughout the recovery period such that LFnu was higher than pre-exercise level in the 10-minute segments beginning 20 minutes after exercise (R20-30, R30-40, R40-50, and R50-60) (Figure 3.14-3.15, and Table 3.19-3.20). On the other hand, among the older adults, LFnu was higher than pre-exercise level in the 5 minute segment immediately after the activity (R0-5), shortly after returned to pre-exercise level (R1-6), and then was higher than pre-exercise in the 10-minute segments beginning 40 minutes after exercise (R40-50, and R50-60) (Figure 3.14-3.15, and Table 3.19-3.20).
Figure 3.14  Age Group by Test Condition Interaction Effect on LFnu (5-minute segment) Before & Immediately After Exercise in Women
Values are mean ± SD. LFnu (5-minute segments) before (Pre-) and immediately after RE. † / ‡ = Young / Old post-exercise value diff from Pre-exercise (p < 0.05).

Table 3.19  Age Group by Test Condition Interaction Effect on HRV Indices (5-minute segment) Before and Immediately After RE in Women

<table>
<thead>
<tr>
<th>Age group</th>
<th>Heart Rate variability Profile</th>
<th>R-R Interval</th>
<th>SDNN</th>
<th>Pnn50</th>
<th>LFnu</th>
<th>LF/HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre.YA</td>
<td></td>
<td>864.6 ±133.3</td>
<td>62.2 ±22.2</td>
<td>22.1 ±17.5</td>
<td>0.60 ±0.15</td>
<td>2.01 ±1.48</td>
</tr>
<tr>
<td>Pre.OA</td>
<td></td>
<td>963.8 ±127.6*</td>
<td>35.6 ±20.2*</td>
<td>4.9 ±9.1*</td>
<td>0.58 ±0.17</td>
<td>1.93 ±1.56</td>
</tr>
<tr>
<td>Rec0-5.YA</td>
<td></td>
<td>817.7 ±123.4†</td>
<td>101.4 ±32.8†</td>
<td>26.8 ±19.7†</td>
<td>0.59 ±0.13</td>
<td>1.77 ±1.22</td>
</tr>
<tr>
<td>Rec0-5.OA</td>
<td></td>
<td>933.1 ±117.8‡*</td>
<td>66.4 ±25.5‡*</td>
<td>7.6 ±9.3‡*</td>
<td>0.63 ±0.14‡</td>
<td>2.16 ±1.48</td>
</tr>
<tr>
<td>Rec1-6.YA</td>
<td></td>
<td>847.5 ±127.0</td>
<td>83.1 ±29.2</td>
<td>29.1 ±19.9</td>
<td>0.54 ±0.15†</td>
<td>1.43 ±1.10†</td>
</tr>
<tr>
<td>Rec1-6.OA</td>
<td></td>
<td>954.1 ±120.8*</td>
<td>51.7 ±20.3*</td>
<td>7.3 ±10.2*</td>
<td>0.58 ±0.16</td>
<td>1.84 ±1.50</td>
</tr>
<tr>
<td>Rec2-7.YA</td>
<td></td>
<td>866.2 ±129.0</td>
<td>69.2 ±24.5</td>
<td>28.7 ±19.6</td>
<td>0.54 ±0.14†</td>
<td>1.47 ±0.94†</td>
</tr>
<tr>
<td>Rec2-7.OA</td>
<td></td>
<td>969.8 ±125.5*</td>
<td>42.4 ±18.5*</td>
<td>7.0 ±11.1*</td>
<td>0.57 ±0.17</td>
<td>1.79 ±1.43</td>
</tr>
<tr>
<td>Rec3-8.YA</td>
<td></td>
<td>869.8 ±125.9</td>
<td>65.4 ±24.1</td>
<td>26.0 ±18.0</td>
<td>0.56 ±0.13†</td>
<td>1.64 ±1.13†</td>
</tr>
<tr>
<td>Rec3-8.OA</td>
<td></td>
<td>976.6 ±124.3‡*</td>
<td>34.7 ±17.6*</td>
<td>6.3 ±10.7*</td>
<td>0.54 ±0.20</td>
<td>1.71 ±1.46</td>
</tr>
<tr>
<td>Rec4-9.YA</td>
<td></td>
<td>864.3 ±121.5</td>
<td>63.3 ±24.4</td>
<td>23.7 ±18.2</td>
<td>0.59 ±0.13</td>
<td>1.79 ±1.23</td>
</tr>
<tr>
<td>Rec4-9.OA</td>
<td></td>
<td>977.0 ±125.9‡*</td>
<td>33.2 ±18.1*</td>
<td>5.5 ±9.6*</td>
<td>0.56 ±0.17</td>
<td>1.76 ±1.41</td>
</tr>
<tr>
<td>Rec5-10.YA</td>
<td></td>
<td>859.2 ±119.0</td>
<td>61.6 ±25.1</td>
<td>21.7 ±17.6</td>
<td>0.61 ±0.13</td>
<td>1.91 ±1.26</td>
</tr>
<tr>
<td>Rec5-10.OA</td>
<td></td>
<td>976.1 ±125.8‡*</td>
<td>33.7 ±16.6*</td>
<td>5.2 ±9.6*</td>
<td>0.58 ±0.15</td>
<td>1.87 ±1.34</td>
</tr>
</tbody>
</table>

Values are mean ± SD. HRV = Heart rate variability. * = Different from young group (p < 0.05). † / ‡ = Young / Old group post-exercise values diff from pre-exercise level (p < 0.05)
Figure 3.15  Age Group by Test Condition Interaction Effect on LFnu (10-minute segments) Before & For 1 Hour After Exercise in Women
Values are mean ± SD. LFnu (10-minute segments) before (Pre-) and for 1-hour after RE. YA = young adult. OA = Older adult. † / ‡ = Young / Old post-exercise value diff from Pre-exercise (p < 0.05).

Table 3.20 Age Group by Test Condition Interaction Effect on HRV Indices (10-minute segment) Before and For 1-Hour After RE in Women

<table>
<thead>
<tr>
<th>Age group</th>
<th>R-R Interval</th>
<th>SDNN</th>
<th>Pnn50</th>
<th>LFnu</th>
<th>LF/HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre.YA</td>
<td>848.3 ±127.5</td>
<td>69.2 ±21.3</td>
<td>19.9 ±15.2</td>
<td>0.64 ±0.13</td>
<td>2.29 ±1.55</td>
</tr>
<tr>
<td>Pre.OA</td>
<td>957.8 ±123.9</td>
<td>45.0 ±21.7</td>
<td>6.8 ±12.6</td>
<td>0.61 ±0.17</td>
<td>2.19 ±1.73</td>
</tr>
<tr>
<td>Rec0-10.YA</td>
<td>834.1 ±120.3†</td>
<td>90.0 ±24.8†</td>
<td>23.6 ±16.6†</td>
<td>0.60 ±0.12†</td>
<td>1.76 ±1.02†</td>
</tr>
<tr>
<td>Rec0-10.OA</td>
<td>955.1 ±121.7</td>
<td>58.5 ±21.8‡</td>
<td>7.9 ±11.5</td>
<td>0.63 ±0.13</td>
<td>2.08 ±1.33</td>
</tr>
<tr>
<td>Rec10-20.YA</td>
<td>849.7 ±122.4</td>
<td>58.9 ±18.3‡</td>
<td>17.5 ±16.2</td>
<td>0.66 ±0.14</td>
<td>2.51 ±1.73</td>
</tr>
<tr>
<td>Rec10-20.OA</td>
<td>972.6 ±131.7†</td>
<td>37.9 ±19.2‡</td>
<td>7.0 ±12.1</td>
<td>0.61 ±0.15</td>
<td>2.10 ±1.50</td>
</tr>
<tr>
<td>Rec20-30.YA</td>
<td>842.0 ±112.1</td>
<td>64.2 ±17.8</td>
<td>18.1 ±14.1</td>
<td>0.67 ±0.12†</td>
<td>2.56 ±1.42†</td>
</tr>
<tr>
<td>Rec20-30.OA</td>
<td>970.3 ±126.1‡</td>
<td>38.9 ±19.4</td>
<td>6.9 ±11.0</td>
<td>0.60 ±0.16</td>
<td>2.09 ±1.57</td>
</tr>
<tr>
<td>Rec30-40.YA</td>
<td>844.2 ±105.0</td>
<td>68.2 ±20.2</td>
<td>18.4 ±12.4</td>
<td>0.68 ±0.14†</td>
<td>2.78 ±1.80†</td>
</tr>
<tr>
<td>Rec30-40.OA</td>
<td>974.2 ±127.3‡</td>
<td>39.2 ±20.6</td>
<td>7.7 ±13.8</td>
<td>0.65 ±0.18</td>
<td>2.51 ±2.13</td>
</tr>
<tr>
<td>Rec40-50.YA</td>
<td>841.4 ±95.0</td>
<td>62.9 ±18.3</td>
<td>18.7 ±11.6</td>
<td>0.68 ±0.11†</td>
<td>2.56 ±1.38†</td>
</tr>
<tr>
<td>Rec40-50.OA</td>
<td>980.0 ±128.7‡</td>
<td>42.3 ±21.8</td>
<td>7.4 ±12.1</td>
<td>0.65 ±0.15</td>
<td>2.60 ±2.03</td>
</tr>
<tr>
<td>Rec50-60.YA</td>
<td>843.4 ±93.3</td>
<td>70.5 ±21.0</td>
<td>19.3 ±11.8</td>
<td>0.67 ±0.13†</td>
<td>2.63 ±1.62†</td>
</tr>
<tr>
<td>Rec50-60.OA</td>
<td>981.3 ±120.9‡</td>
<td>42.3 ±18.6</td>
<td>8.1 ±11.7‡</td>
<td>0.67 ±0.13‡</td>
<td>2.63 ±1.75‡</td>
</tr>
</tbody>
</table>

Values are mean ± SD. HRV = Heart rate variability. YA = Young adult. OA = Older Adults. * = Different from young group (p ≤ 0.05). † / ‡ = Young / Old group post-exercise values diff from pre-exercise level (p ≤ 0.05).
3.4.3. Age by Exercise Condition Interaction. The 3-way ANOVA did not reveal significant age by exercise condition interaction effects. However, there were a number of trends in the data that may be of interest. There was a tendency for the 15-RM condition to result in higher SBP, DBP, and MAP ($p = 0.08, 0.07, \text{ and } 0.06, \text{ respectively}$) at peak exercise, local min, and local max in the older group, but not in the young group. In comparison to the 5-RM condition, follow-up pairwise comparisons revealed that the 15-RM resulted in higher SBP, DBP, and MAP at peak exercise, local min, and local max only among the older women (Figure 3.16-3.18 and Table 3.21–3.23).

![Figure 3.16 Age Group by Exercise Condition Interaction Effect on SBP Responses During & ImmediatelyFollowing RE in Women](image)

Values are mean ± SD. Systolic blood pressure (SBP) before (Pre-), during (Peak), and immediately (local min and local max) after RE. * = Diff from 5-RM condition value in the older group ($p \leq 0.006$). † / ‡ = 5- / 15-RM Old group values diff from pre-exercise level ($p < 0.05$). $\$/ # = 5- / 15-RM young group values diff from pre-exercise level ($p \leq 0.05$).
### Table 3.21 Age Group by Exercise Condition Interaction Effect of SBP Responses During and Immediately After RE in Women

<table>
<thead>
<tr>
<th></th>
<th>Young 5RM</th>
<th>Young 15RM</th>
<th>Old 5RM</th>
<th>Old 15RM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-</td>
<td>104.4 ± 8.0</td>
<td>105.0 ± 6.4</td>
<td>121.3 ± 11.7</td>
<td>123.7 ± 15.1</td>
</tr>
<tr>
<td>Peak</td>
<td>138.7 ± 15.0 $</td>
<td>147.0 ± 25.1 $</td>
<td>145.9 ± 17.2†</td>
<td>164.3 ± 22.6‡*</td>
</tr>
<tr>
<td>Local min</td>
<td>115.6 ± 12.9 $</td>
<td>113.0 ± 17.3</td>
<td>125.2 ± 7.9</td>
<td>141.0 ± 18.8‡*</td>
</tr>
<tr>
<td>Local max</td>
<td>119.4 ± 11.9 $</td>
<td>116.6 ± 17.9 $</td>
<td>128.2 ± 9.6</td>
<td>143.5 ± 19.1‡*</td>
</tr>
</tbody>
</table>

Values are mm Hg and represent mean ± SD. Systolic blood pressure (SBP) before (Pre-), during (Peak), and immediately (Local min and local max) after RE. * = Diff from 5-RM condition in the older group ($p < 0.006$). † / ‡ = 5- / 15-RM Old group values diff from pre-exercise level ($p < 0.05$). $ / # = 5- / 15-RM young group values diff from pre-exercise level ($p < 0.05$).

#### Figure 3.17 Age Group by Exercise Condition Interaction Effect on DBP Responses During & Immediately Following RE in Women

Values are mean ± SD. Diastolic blood pressure (DBP) before (Pre-), during (Peak), and immediately (local min and local max) after RE. * = Diff from 5-RM condition value in older group ($p ≤ 0.04$). † / ‡ = 5- / 15-RM older group values diff from pre-exercise level ($p ≤ 0.05$). $ / # = 5- / 15-RM young group values diff from pre-exercise level ($p ≤ 0.05$).
Table 3.22  Age Group by Exercise Condition Interaction Effect of DBP Responses During and Immediately After RE in Women

<table>
<thead>
<tr>
<th></th>
<th>Young 5RM</th>
<th>Young 15RM</th>
<th>Old 5RM</th>
<th>Old 15RM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre</td>
<td>59.3 ± 10.2</td>
<td>58.9 ± 8.3</td>
<td>57.9 ± 8.6</td>
<td>59.9 ± 9.5</td>
</tr>
<tr>
<td>Peak</td>
<td>90.4 ± 14.3 $</td>
<td>92.6 ± 15.4 #</td>
<td>73.8 ± 9.9 †</td>
<td>87.9 ± 15.4‡*</td>
</tr>
<tr>
<td>Local min</td>
<td>55.8 ± 11.6</td>
<td>53.3 ± 10.4 #</td>
<td>52.3 ±10.0 †</td>
<td>62.0 ± 15.5 *</td>
</tr>
<tr>
<td>Local max</td>
<td>59.6 ± 10.5</td>
<td>57.5 ± 10.4</td>
<td>54.4 ± 9.5</td>
<td>64.6 ± 15.4 *</td>
</tr>
</tbody>
</table>

Values are in mm Hg and represent mean ± SD. Diastolic blood pressure (DBP) before (Pre-), during (Peak), and immediately (local min and local max) after resistance exercise (RE).

* = Diff from the 5-RM condition value in the older group (Pairwise comparisons, p < 0.04).
† / ‡ = 5- / 15-RM older group values diff from pre-exercise level (p < 0.05).
$ / # = 5- / 15-RM young group values diff from pre-exercise level (p < 0.05).

Figure 3.18 Age Group by Exercise Condition Interaction Effect on MAP Responses During & Immediately Following RE in Women

Values are mean ± SD. Mean arterial pressure (MAP) before (Pre-), during (Peak), and immediately (local min and local max) after RE. * = Diff from 5-RM condition value in older group (p ≤ 0.04). † / ‡ = 5- / 15-RM older group values diff from pre-exercise level (p ≤ 0.05). $ / # = 5- / 15-RM young group values diff from pre-exercise level (p ≤ 0.05).
Table 3.23 Age Group by Exercise Condition Interaction Effect of MAP Responses During and Immediately After RE in Women

<table>
<thead>
<tr>
<th></th>
<th>Young 5RM</th>
<th>Young 15RM</th>
<th>Old 5RM</th>
<th>Old 15RM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-</td>
<td>75.4 ± 8.6</td>
<td>74.9 ± 7.7</td>
<td>79.0 ± 8.3</td>
<td>81.2 ±10.3</td>
</tr>
<tr>
<td>Peak</td>
<td>106.1 ±14.0$</td>
<td>111.6 ±16.6#</td>
<td>98.8 ±10.8 †</td>
<td>113.0 ±15.4‡*</td>
</tr>
<tr>
<td>Local min</td>
<td>76.5 ±11.0</td>
<td>72.4 ±11.4</td>
<td>75.6 ± 7.1</td>
<td>87.5 ±15.4‡*</td>
</tr>
<tr>
<td>Local max</td>
<td>77.2 ±12.2</td>
<td>76.8 ±12.3</td>
<td>78.3 ± 7.4</td>
<td>89.4 ±15.0‡*</td>
</tr>
</tbody>
</table>

Values are in mm Hg and represent mean ± SD. Mean arterial pressure (MAP) before (Pre-), during (Peak), and immediately (local min and local max) after resistance exercise (RE).

* = Diff from the 5-RM condition value in the older group ($p < 0.04$). † / ‡ = 5- / 15-RM older group values diff from pre-exercise level ($p < 0.05$). $ / # = 5- / 15-RM young group values diff from pre-exercise level ($p < 0.05$).

There was also a trend ($p = 0.16$) for higher SBPs throughout all testing periods (i.e., before, during and for 1-hour after RE assessment) with the 15-RM condition among the older women, but not among the young women (Figure 3.19 & Table 3.24). Similarly, follow up pairwise comparisons revealed higher SBP at peak exercise ($p = 0.0001$), R1’ ($p = 0.03$), and R3’ ($p = 0.03$), and a tendency to be higher at R15’ ($p = 0.17$), R20’ ($p = 0.11$), R40’ ($p = 0.08$), and R50’ ($p = 0.06$) with the 15-RM condition among the older women, but not among the young group (Figure 3.19, & Table 3.24).

Moreover, there was a trend ($p = 0.19$) for higher DBPs throughout all testing periods from pre-exercise, through exercise period, and during the long-term recovery period with the 15-RM exercise condition among the older women, but not among the young women (Figure 3.20, and Table 3.25). Follow-up pairwise comparisons revealed higher DBPs at peak exercise ($p = 0.02$), R15’ ($p = 0.04$), R20’ ($p = 0.03$), and R50’ ($p = 0.006$) with the 15-RM exercise condition among the older women, but not among the young group (Figure 3.20, & Table 3.25).
3.4.4. Age by Exercise Condition by Test Condition Interaction. Inferences about main effects on SBP, DBP, and MAP may be tempered by nearly significant three-way (test condition x age group x exercise condition) interactions (\(p = 0.10, 0.10, \) and 0.08, respectively). More specifically, the pressure values at peak exercise, local min, and local max appear to be higher following the 15-RM exercise condition among the older women, but not in the young adults (Figure 3.16-3.18). Furthermore, the magnitude of post-exercise DBP drops tended to be greater following the 5-RM exercise condition in comparison to the 15-RM condition among the older women, but not among the young group (Figure 3.19-3.21, and Table 3.2-3.26).

**Figure 3.19 Age Group by Exercise Condition Interaction Effect on SBP Responses During & For 1 Hour After RE in Women**

Values are mean ± SD. Systolic blood pressure (SBP) before (Pre-), during (Peak), and for 1 hour after RE. * = Diff from the 5-RM condition value in the older group (\(p \leq 0.05\)). + = tended to be diff from the 5-RM condition in the older group (\(p \leq 0.15\)). † / ‡ = 5- / 15-RM older group values diff from pre-exercise level (\(p \leq 0.05\)). $ / # = 5- / 15-RM young group values diff from pre-exercise level (\(p \leq 0.05\)).
Table 3.24  Age Group by Exercise Condition Interaction Effect on SBP Responses During, & For 1 Hour After RE in Women

<table>
<thead>
<tr>
<th></th>
<th>Young 5-RM</th>
<th>Young 15-RM</th>
<th>Old 5-RM</th>
<th>Old 15-RM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-</td>
<td>104.4 ± 8.9</td>
<td>105.0 ± 6.4</td>
<td>121.3 ± 11.7</td>
<td>123.7 ± 15.1</td>
</tr>
<tr>
<td>Peak</td>
<td>138.7 ± 15.0 $</td>
<td>146.9 ± 25.1 #</td>
<td>145.9 ± 17.2 †</td>
<td>164.3 ± 22.6 ‡ *</td>
</tr>
<tr>
<td>R1'</td>
<td>116.2 ± 10.9 $</td>
<td>110.0 ± 15.3</td>
<td>122.1 ± 8.4</td>
<td>132.3 ± 20.9 ‡ *</td>
</tr>
<tr>
<td>R3'</td>
<td>112.5 ± 10.6 $</td>
<td>107.2 ± 15.4</td>
<td>118.7 ± 8.5</td>
<td>127.9 ± 18.7 *</td>
</tr>
<tr>
<td>R5'</td>
<td>110.9 ± 9.3 $</td>
<td>105.5 ± 12.8</td>
<td>118.1 ± 11.5</td>
<td>121.2 ± 19.1</td>
</tr>
<tr>
<td>R10'</td>
<td>106.9 ± 12.5</td>
<td>102.5 ± 8.7</td>
<td>113.6 ± 11.3 †</td>
<td>115.5 ± 15.9 ‡</td>
</tr>
<tr>
<td>R15'</td>
<td>102.7 ± 12.5</td>
<td>105.0 ± 11.9</td>
<td>112.7 ± 13.1 †</td>
<td>117.3 ± 13.3 ‡ +</td>
</tr>
<tr>
<td>R20'</td>
<td>104.6 ± 9.6</td>
<td>105.6 ± 12.4</td>
<td>113.5 ± 15.9 †</td>
<td>120.2 ± 16.1 +</td>
</tr>
<tr>
<td>R30'</td>
<td>104.6 ± 9.3</td>
<td>103.1 ± 14.1</td>
<td>116.0 ± 13.5 †</td>
<td>117.5 ± 15.5 ‡</td>
</tr>
<tr>
<td>R40'</td>
<td>105.3 ± 9.6</td>
<td>105.4 ± 14.7</td>
<td>112.6 ± 15.6 †</td>
<td>119.5 ± 15.4 +</td>
</tr>
<tr>
<td>R50'</td>
<td>104.6 ± 9.7</td>
<td>106.2 ± 12.1</td>
<td>111.5 ± 16.3 †</td>
<td>118.8 ± 14.9 +</td>
</tr>
<tr>
<td>R60'</td>
<td>106.4 ± 11.2</td>
<td>102.9 ± 15.6</td>
<td>110.6 ± 16.7 †</td>
<td>115.5 ± 16.3 ‡</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Systolic blood pressure before (Pre-), during (Peak), and for 1 hour after RE. * = Diff. from the 5-RM condition in the older group ($p < 0.05$). + = tended to be diff from the 5-RM condition in the older group ($p < 0.15$). † / ‡ = 5- / 15-RM older group values diff from pre-exercise ($p < 0.05$). $ / # = 5- / 15-RM young group values diff from pre-exercise ($p < 0.05$).

Figure 3.20 Age Group by Exercise Condition Interaction Effect on DBP Responses During & For 1 Hour After RE in Women

Values are mean ± SD. Diastolic blood pressure (DBP) before (Pre-), during (Peak), and for 1 hour after RE. * = Diff. from 5-RM condition in the older group ($p < 0.05$). + = tended to be diff from 5-RM condition in the older group ($p < 0.15$). † / ‡ = 5- / 15-RM older group values diff from pre-exercise ($p < 0.05$). $ / # = 5- / 15-RM young group values diff from pre-exercise ($p < 0.05$).
Table 3.25  Age Group by Exercise Condition Interaction Effect on DBP Before, During, & For 1 Hour After RE in Women

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th></th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5-RM</td>
<td>15-RM</td>
<td>5-RM</td>
</tr>
<tr>
<td>Pre-</td>
<td>59.3 + 10.2</td>
<td>58.9 + 8.3</td>
<td>57.9 + 8.6</td>
</tr>
<tr>
<td>Peak</td>
<td>90.4 + 14.3 $</td>
<td>92.6 + 15.4 #</td>
<td>73.8 + 9.9 †</td>
</tr>
<tr>
<td>R1’</td>
<td>59.3 + 11.7</td>
<td>55.1 + 10.4 #</td>
<td>53.0 + 9.7</td>
</tr>
<tr>
<td>R3’</td>
<td>58.8 + 11.2</td>
<td>54.3 + 10.9 #</td>
<td>51.7 + 9.5 †</td>
</tr>
<tr>
<td>R5’</td>
<td>58.1 + 11.4</td>
<td>53.7 + 8.8 #</td>
<td>52.2 + 10.2†</td>
</tr>
<tr>
<td>R10’</td>
<td>56.7 + 13.0</td>
<td>54.6 + 8.7 #</td>
<td>50.6 + 9.5 †</td>
</tr>
<tr>
<td>R15’</td>
<td>55.8 + 9.8</td>
<td>58.7 + 11.2</td>
<td>48.7 + 10.2†</td>
</tr>
<tr>
<td>R20’</td>
<td>56.2 + 10.1</td>
<td>57.6 + 10.2</td>
<td>47.9 + 10.0†</td>
</tr>
<tr>
<td>R30’</td>
<td>55.2 + 9.6</td>
<td>55.5 + 12.0</td>
<td>48.8 + 9.8 †</td>
</tr>
<tr>
<td>R40’</td>
<td>56.9 + 7.4</td>
<td>57.1 + 12.8</td>
<td>48.9 + 10.3 †</td>
</tr>
<tr>
<td>R50’</td>
<td>55.0 + 8.0</td>
<td>56.2 + 11.5</td>
<td>45.6 + 10.4 †</td>
</tr>
<tr>
<td>R60’</td>
<td>56.9 + 10.6</td>
<td>55.9 + 13.1</td>
<td>46.2 + 7.9 †</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Diastolic blood pressure before (Pre-), during (Peak), and for 1 hour after RE. * = Diff. from 5-RM condition in the older group (p < 0.05). + = tended to be diff from 5-RM condition in the older group (p < 0.15). † / ‡ = 5- / 15-RM older group values diff from pre-exercise (p < 0.05). $ / # = 5- / 15-RM young group values diff from pre-exercise (p < 0.05).

Figure 3.21  Age Group by Exercise Condition Interaction Effect on MAP Responses Before, During & For 1 Hour After RE in Women

Values are mean ± SD. Mean arterial pressure (MAP) before (Pre-), during (Peak), and for 1 hour after RE. * = Diff. from 5-RM condition in the older group (p < 0.05). + = tended to be diff from 5-RM condition in the older group (p < 0.15). † / ‡ = 5- / 15-RM older group values diff from pre-exercise (p < 0.05). $ / # = 5- / 15-RM young group values diff from pre-exercise (p < 0.05).
Table 3.26  Age Group by Exercise Condition Interaction Effect on MAP Responses Before, During, & For 1 Hour After RE in Women

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5-RM</td>
<td>15-RM</td>
</tr>
<tr>
<td>Pre-</td>
<td>75.4 ± 8.6</td>
<td>74.9 ± 7.7</td>
</tr>
<tr>
<td>Peak</td>
<td>106.4 ±14.0 $</td>
<td>111.6 ±16.6 #</td>
</tr>
<tr>
<td>R1’</td>
<td>78.8 ± 9.9</td>
<td>74.8 ±11.3</td>
</tr>
<tr>
<td>R3’</td>
<td>77.0 ±10.1</td>
<td>72.9 ±11.0</td>
</tr>
<tr>
<td>R5’</td>
<td>76.2 ± 9.6</td>
<td>72.3 ± 8.4</td>
</tr>
<tr>
<td>R10’</td>
<td>73.6 ±12.1</td>
<td>72.2 ± 6.7</td>
</tr>
<tr>
<td>R15’</td>
<td>71.7 ± 9.8</td>
<td>76.4 ±10.3</td>
</tr>
<tr>
<td>R20’</td>
<td>73.3 ± 7.9</td>
<td>75.7 ±10.6</td>
</tr>
<tr>
<td>R30’</td>
<td>72.3 ± 8.3</td>
<td>73.3 ±12.8</td>
</tr>
<tr>
<td>R40’</td>
<td>74.0 ± 5.8</td>
<td>74.7 ±13.6</td>
</tr>
<tr>
<td>R50’</td>
<td>71.8 ± 7.4</td>
<td>74.8 ±11.6</td>
</tr>
<tr>
<td>R60’</td>
<td>73.7 ± 9.0</td>
<td>73.7 ±12.7</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Mean arterial pressure before (Pre-), during (Peak), and for 1 hour after RE. * = Diff. from 5-RM condition in the older group (p < 0.05). + = tended to be diff from 5-RM condition in the older group (p < 0.15). † / ‡ = 5- / 15-RM older group values diff from pre-exercise (p < 0.05). $ / # = 5- / 15-RM young group values diff from pre-exercise (p < 0.05).

There was a significant three-way (test condition x age group x exercise condition) interaction on post-exercise FVR (p = 0.04), suggesting that FVRs were influenced by exercise condition in the young adults, but not in the older adults (Figure 3.22, & Table 3.27). Follow-up pairwise comparisons within each age group revealed that post-exercise FVRs after the 5-RM condition were higher than pre-exercise level at all recovery time points (except R7’) among the young group, but only at R4’, R20’, and R40’ among the older group. Further, post-exercise FVRs after the 15-RM condition were higher than pre-exercise level at R4’, R7’, R30’, R40’, and R60’ among the young group, but lower than pre-exercise level at R7’ and R30’ among the older women (Figure 3.22, & Table 3.27).
Figure 3.22 Age Group by Exercise Condition by Test Condition Interaction
Effect on FVR Before & For 1 Hour After RE in Women
Values are mean ± SD. Forearm vascular resistance (FVR) before (Pre-) and for 1 hour after RE. † / ‡ = 5- / 15-RM older group values diff from pre-exercise ($p < 0.05$). $/$ # = 5- / 15-RM young group values diff from pre-exercise ($p < 0.05$).
Table 3.27  Age Group by Exercise Condition by Test Condition Interaction
Effect on Forearm Vascular Resistance Before and For 1 Hour After
RE in Women

<table>
<thead>
<tr>
<th></th>
<th>FBF (mL/100mL/min)</th>
<th>FVR (Units)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Young</td>
<td>Old</td>
</tr>
<tr>
<td>Pre-05</td>
<td>1.90 ± 0.35</td>
<td>2.01 ± 0.61</td>
</tr>
<tr>
<td>Pre-15</td>
<td>1.85 ± 0.46</td>
<td>1.97 ± 0.73</td>
</tr>
<tr>
<td>R4’-05</td>
<td>1.70 ± 0.63</td>
<td>1.80 ± 0.50</td>
</tr>
<tr>
<td>R4’-15</td>
<td>1.39 ± 0.45</td>
<td>2.11 ± 0.83</td>
</tr>
<tr>
<td>R7’-05</td>
<td>1.85 ± 0.95</td>
<td>1.81 ± 0.61</td>
</tr>
<tr>
<td>R7’-15</td>
<td>1.67 ± 1.05</td>
<td>2.05 ± 0.55</td>
</tr>
<tr>
<td>R10’-05</td>
<td>1.52 ± 0.52</td>
<td>1.99 ± 0.56</td>
</tr>
<tr>
<td>R10’-15</td>
<td>1.46 ± 0.49</td>
<td>1.67 ± 0.39</td>
</tr>
<tr>
<td>R15’-05</td>
<td>1.48 ± 0.53</td>
<td>1.85 ± 0.82</td>
</tr>
<tr>
<td>R15’-15</td>
<td>1.51 ± 0.77</td>
<td>1.79 ± 0.49</td>
</tr>
<tr>
<td>R20’-05</td>
<td>1.55 ± 0.65</td>
<td>1.61 ± 0.53</td>
</tr>
<tr>
<td>R20’-15</td>
<td>1.59 ± 0.77</td>
<td>1.86 ± 0.56</td>
</tr>
<tr>
<td>R30’-05</td>
<td>1.38 ± 0.43</td>
<td>1.78 ± 0.60</td>
</tr>
<tr>
<td>R30’-15</td>
<td>1.82 ± 0.89</td>
<td>2.32 ± 0.72</td>
</tr>
<tr>
<td>R40’-05</td>
<td>1.60 ± 0.65</td>
<td>1.57 ± 0.52</td>
</tr>
<tr>
<td>R40’-15</td>
<td>1.80 ± 0.65</td>
<td>1.68 ± 0.45</td>
</tr>
<tr>
<td>R50’-05</td>
<td>1.43 ± 0.56</td>
<td>1.71 ± 0.50</td>
</tr>
<tr>
<td>R50’-15</td>
<td>1.50 ± 0.65</td>
<td>1.74 ± 0.45</td>
</tr>
<tr>
<td>R60’-05</td>
<td>1.59 ± 0.67</td>
<td>1.51 ± 0.48</td>
</tr>
<tr>
<td>R60’-15</td>
<td>1.41 ± 0.72</td>
<td>1.70 ± 0.34</td>
</tr>
</tbody>
</table>

Values are mean ± SD. FBF = Forearm blood inflow (FVR) and forearm vascular resistance (FVR) before (Pre-) and for 1-hour after RE. † / ‡ = 5- / 15-RM older group values diff from pre-exercise ($p < 0.05$). $ / # = 5- / 15-RM young group values diff from pre-exercise ($p < 0.05$).

3.5. Relationships Between Indicators of Autonomic and Vascular Function and Hemodynamic Responses During and Following RE

3.5.1. Autonomic Function & Hemodynamic Response During and Following RE.

Bivariate correlations between indicators of autonomic function (SDNN, pnn50, LFnu, and LF/HF ratio) and patterns of arterial BP (SBP) responses before and following RE (mean pre-exercise, magnitude and mean rate of decline, and magnitude and rate of recovery) by age group and exercise condition are presented below from Table 3.28 to Table 3.31.
Table 3.28  Bivariate Correlation Between Indicators of Autonomic Function & SBP Before and After RE Pooling both Young and Older Group Data

<table>
<thead>
<tr>
<th></th>
<th>SDNN</th>
<th>pnn50</th>
<th>LFnu,rest</th>
<th>LF/HF,rest</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP</td>
<td>Rest</td>
<td>Rest</td>
<td>Rest</td>
<td>Rec0-5</td>
</tr>
<tr>
<td>Mean pre-exercise</td>
<td>r = -0.47 *</td>
<td>r = -0.37 *</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magnitude of recovery</td>
<td></td>
<td>r = 0.40 *</td>
<td>r = 0.36 *</td>
<td>r = 0.29 * r = 0.28 *</td>
</tr>
<tr>
<td>Mean rate of recovery</td>
<td></td>
<td>r = 0.46 *</td>
<td>r = 0.44 *</td>
<td>r = 0.38 * r = 0.36 *</td>
</tr>
</tbody>
</table>

* = $p \leq 0.05$.

Table 3.29  Bivariate Correlation Between Indicators of Autonomic Function & SBP Before and After RE in Older Women

<table>
<thead>
<tr>
<th></th>
<th>SDNN</th>
<th>pnn50</th>
<th>LFnu</th>
<th>LF/HF ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP</td>
<td>Rest</td>
<td>Rest</td>
<td>Rest</td>
<td>Rec0-5</td>
</tr>
<tr>
<td>Mean pre-exercise</td>
<td>r = -0.29 +</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magnitude of Recovery</td>
<td></td>
<td>r = 0.54 *</td>
<td>r = 0.44 *</td>
<td>r = 0.45 * r = 0.32 +</td>
</tr>
<tr>
<td>Mean rate of recovery</td>
<td></td>
<td>r = 0.59 *</td>
<td>r = 0.53</td>
<td>r = 0.51 * r = 0.38 *</td>
</tr>
</tbody>
</table>

* = $p \leq 0.05$. + = $p \leq 0.15$.

Table 3.30  Bivariate Correlation Between Indicators of Autonomic Function & SBP Before and After RE in Young Women

<table>
<thead>
<tr>
<th></th>
<th>SDNN</th>
<th>pnn50</th>
<th>LFnu</th>
<th>LF/HF ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP</td>
<td>Rest</td>
<td>Rest</td>
<td>Rest</td>
<td>Rec0-5</td>
</tr>
<tr>
<td>Mean pre-exercise</td>
<td>r = 0.37 *</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magnitude of Recovery</td>
<td></td>
<td>r = 0.40 *</td>
<td></td>
<td>r = 0.32 +</td>
</tr>
<tr>
<td>Mean rate of recovery</td>
<td></td>
<td>r = 0.31 +</td>
<td>r = 0.50 *</td>
<td>r = 0.42 *</td>
</tr>
</tbody>
</table>

* = $p \leq 0.05$. + = $p \leq 0.15$. 
Table 3.31  Bivariate Correlation Between Indicators of Autonomic Function & SBP Before and After RE by Exercise Condition

<table>
<thead>
<tr>
<th>Autonomic Indicators</th>
<th>Mean Rate of SBP Recovery</th>
<th>Magnitude of SBP Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>LFnu at rest (5-RM)</td>
<td>r = 0.49 *</td>
<td>r = 0.34 +</td>
</tr>
<tr>
<td>LFnu at rest (15-RM)</td>
<td>r = 0.42 *</td>
<td>r = 0.42 *</td>
</tr>
<tr>
<td>LFnu at Rec 0-5 (5-RM)</td>
<td>r = 0.46 *</td>
<td>r = 0.27 +</td>
</tr>
<tr>
<td>LFnu at Rec 0-5 (15-RM)</td>
<td>r = 0.42 *</td>
<td>r = 0.42 *</td>
</tr>
<tr>
<td>LF/HF ratio at rest (5-RM)</td>
<td>r = 0.39 *</td>
<td>r = 0.26 +</td>
</tr>
<tr>
<td>LF/HF ratio at rest (15-RM)</td>
<td>r = 0.37 *</td>
<td>r = 0.32 +</td>
</tr>
<tr>
<td>LF/HF ratio at Rec 0-5 (5-RM)</td>
<td>r = 0.38 *</td>
<td></td>
</tr>
<tr>
<td>LF/HF ratio at Rec 0-5 (15-RM)</td>
<td>r = 0.41 *</td>
<td>r = 0.39 *</td>
</tr>
</tbody>
</table>

* = p ≤ 0.05. + = p ≤ 0.15.

In addition, linear regression analysis examining the relationship between autonomic indicators (LFnu Rec 0-5) and magnitude of BP recovery (SBP, DBP, and MAP) from local min to local max are presented in the Table 3.32.

Table 3.32  Relationship Between Indicators of Autonomic Function & Arterial BP Responses After RE Pooling Both Young and Older Group Data

<table>
<thead>
<tr>
<th>LFnu Rec0-5</th>
<th>Young</th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>β</td>
</tr>
<tr>
<td>Magnitude of SBP recovery</td>
<td>r = 0.67 *</td>
<td>0.93 ± 0.19 †</td>
</tr>
<tr>
<td>Magnitude of DBP recovery</td>
<td>r = 0.69 *</td>
<td>1.00 ± 0.19 †</td>
</tr>
<tr>
<td>Magnitude of MAP recovery</td>
<td>r = 0.66 *</td>
<td>0.95 ± 0.20 †</td>
</tr>
</tbody>
</table>

* = p ≤ 0.05. † = p ≤ 0.0001. LFnu = Low frequency power at recovery 0-5. SBP = Systolic blood pressure. DBP = Diastolic blood pressure MAP = Mean arterial blood pressure

3.5.2. Vascular Function Indices & Hemodynamic Response During and Following RE.

Bivariate correlations between vascular function indices (FBF, FVR, FVC, and FVO) and patterns of arterial BP (SBP and DBP) responses before and following RE (pre-exercise, magnitude and rate of decline, magnitude and rate of recovery, and magnitude of post-exercise BP drop) by age group are presented below from Table 3.33 to Table 3.38.
### Table 3.33 Bivariate Correlation Between Vascular Function Indices & SBP Before and After RE Pooling Both Young and Older Adult Data

<table>
<thead>
<tr>
<th>SBP</th>
<th>FBF</th>
<th>FVC</th>
<th>FVO</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Occlusion</td>
<td>Rest</td>
</tr>
<tr>
<td>Mean pre-exercise</td>
<td>r = -0.35 *</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1' SBP/peak ratio</td>
<td>r = -0.32 *</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3' SBP/peak ratio</td>
<td>r = -0.26 *</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10' PE SBP drop</td>
<td></td>
<td></td>
<td>r = 0.36 *</td>
</tr>
<tr>
<td>15' PE SBP drop</td>
<td></td>
<td></td>
<td>r = 0.33 *</td>
</tr>
<tr>
<td>20' PE SBP drop</td>
<td></td>
<td></td>
<td>r = 0.38 *</td>
</tr>
<tr>
<td>30' PE SBP drop</td>
<td></td>
<td></td>
<td>r = 0.33 +</td>
</tr>
<tr>
<td>40' PE SBP drop</td>
<td></td>
<td></td>
<td>r = 0.39 *</td>
</tr>
<tr>
<td>50' PE SBP drop</td>
<td></td>
<td></td>
<td>r = 0.30 *</td>
</tr>
<tr>
<td>60' PE SBP drop</td>
<td></td>
<td></td>
<td>r = 0.29 *</td>
</tr>
</tbody>
</table>

* = p ≤ 0.05. + = p ≤ 0.15. SBP = Systolic blood pressure. FBF = Forearm Blood inflow. FVC = Forearm venous capacitance. FVO = Forearm venous outflow. R10’ = SBP value at 10’ after RE. PE SBP drop = Magnitude of post-exercise (PE) SBP drop below pre-exercise level.

### Table 3.34 Bivariate Correlation Between Vascular Function Indices & SBP Before and After RE in Older Women

<table>
<thead>
<tr>
<th>SBP</th>
<th>FBF</th>
<th>FVC</th>
<th>FVO</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Rest</td>
<td>Occlusion</td>
</tr>
<tr>
<td>1' SBP/peak ratio</td>
<td>r = -0.38 *</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3' SBP/peak ratio</td>
<td>r = -0.25 +</td>
<td>r = -0.36 +</td>
<td>r = -0.31 +</td>
</tr>
<tr>
<td>10' PE SBP drop</td>
<td></td>
<td></td>
<td>r = 0.34 +</td>
</tr>
<tr>
<td>15' PE SBP drop</td>
<td></td>
<td></td>
<td>r = 0.40 *</td>
</tr>
<tr>
<td>20' PE SBP drop</td>
<td></td>
<td></td>
<td>r = 0.33 *</td>
</tr>
<tr>
<td>30' PE SBP drop</td>
<td></td>
<td></td>
<td>r = 0.36 *</td>
</tr>
<tr>
<td>40' PE SBP drop</td>
<td></td>
<td></td>
<td>r = 0.34 +</td>
</tr>
<tr>
<td>50' PE SBP drop</td>
<td></td>
<td></td>
<td>r = 0.42 *</td>
</tr>
<tr>
<td>60' PE SBP drop</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = p ≤ 0.05. + = p ≤ 0.15. PE SBP drop = Post-exercise systolic blood pressure drop. FBF = Forearm Blood inflow. FVC = Forearm venous capacitance. FVO = Forearm venous outflow.
### Table 3.35  Bivariate Correlation Between Vascular Function Indices & SBP
Before and After RE in Young Women

<table>
<thead>
<tr>
<th></th>
<th>FBF</th>
<th>FVC</th>
<th>FVO</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Occlusion</td>
<td>Rest</td>
</tr>
<tr>
<td>Magnitude of increase</td>
<td>r = 0.34 +</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magnitude of decline</td>
<td>r = 0.40 *</td>
<td>r = 0.39 +</td>
<td></td>
</tr>
<tr>
<td>1⁄SBP/peak ratio</td>
<td>r = -0.36 +</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3⁄SBP/peak ratio</td>
<td>r = -0.35 +</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = p ≤ 0.05. + = p ≤ 0.15. SBP = Systolic blood pressure. FVC = Forearm venous capacitance. FVO = Forearm venous outflow.

### Table 3.36  Bivariate Correlation Between Vascular Function Indices & DBP
Before and After RE Pooling Both Young and Older Adult Data

<table>
<thead>
<tr>
<th></th>
<th>FBF</th>
<th>FVC</th>
<th>FVO</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Occlusion</td>
<td>Rest</td>
</tr>
<tr>
<td>Magnitude of increase</td>
<td>r = 0.37 *</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magnitude of decline</td>
<td>r = 0.40 *</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean rate of decline</td>
<td>r = 0.27 *</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10’ PE DBP drop</td>
<td>r = 0.35 *</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15’ PE DBP drop</td>
<td>r = 0.32 *</td>
<td></td>
<td></td>
</tr>
<tr>
<td>40’ PE DBP drop</td>
<td>r = 0.27 *</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = (p ≤ 0.05). + = (p ≤ 0.15). PE DBP drop = Post-exercise diastolic blood pressure. FBF = Forearm Blood inflow. FVC = Forearm venous capacitance. FVO = Forearm venous outflow.

### Table 3.37  Bivariate Correlation Between Vascular Function Indices & DBP
Before and After RE in Older Women

<table>
<thead>
<tr>
<th></th>
<th>FBF</th>
<th>FVC</th>
<th>FVO</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Occlusion</td>
<td>Rest</td>
</tr>
<tr>
<td>Local min</td>
<td>r = -0.26 +</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Local max</td>
<td>r = -0.30 +</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magnitude of decline</td>
<td>r = 0.32 +</td>
<td>r = 0.26 +</td>
<td>r = 0.36 +</td>
</tr>
<tr>
<td>Mean rate of decline</td>
<td>r = 0.43 *</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Rate of Recovery</td>
<td>r = 0.51 *</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = (p ≤ 0.05). + = (p ≤ 0.15). DBP = Diastolic blood pressure. FBF = Forearm Blood inflow. FVC = Forearm venous capacitance. FVO = Forearm venous outflow.
Table 3.38  Bivariate Correlation Between Vascular Function Indices & DBP
Before and After RE in Young Women

<table>
<thead>
<tr>
<th></th>
<th>FBF</th>
<th>FVC</th>
<th>FVO</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean pre-exercise</td>
<td>r = 0.39 *</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Local min</td>
<td>r = 0.42 *</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Local max</td>
<td>r = 0.47 *</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Magnitude of increase</td>
<td>r = 0.47 *</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Magnitude decline</td>
<td>r = 0.34 +</td>
<td></td>
</tr>
</tbody>
</table>

* = p ≤ 0.05. + = p ≤ 0.15. DBP = Diastolic blood pressure. FBF = Forearm blood inflow. FVC = Forearm venous capacitance. FVO = Forearm venous outflow.

In addition, within session reliability and inter-session reliability for resting forearm blood inflow (FBF<sub>rest</sub>) and forearm vascular resistance (FVR<sub>rest</sub>) were derived for each age group using intraclass correlation (ICC). The results of within session reliability revealed ranges of ICCs between 0.63 and 0.95 for FBF<sub>rest</sub> and between 0.63 and 0.92 for FVR<sub>rest</sub> for both age groups. However, the results of inter-session reliability (before 5-RM and 15-RM exercise conditions) revealed ICCs of 0.17 and 0.63 for FBF<sub>rest</sub> and FVR<sub>rest</sub> among the older group, and ICCs of 0.40 and 0.62 for FBF<sub>rest</sub> and FVR<sub>rest</sub> among the young group, respectively (Extended Results Section, Tables B.27-B29).

3.5.3. Autonomic Function & Vascular Function Indices. Bivariate correlations examining relationships between indicators of autonomic function and vascular function (at rest and following arterial occlusion) by age group are presented below in Table 3.39 and 3.42.
Table 3.39 Bivariate Correlation Between Indicators of Autonomic Function & Vascular Function Indices Pooling Both Young and Older Group Data

<table>
<thead>
<tr>
<th>Vascular Function</th>
<th>SDNN</th>
<th>Pnn50</th>
<th>LN LFnu.rest</th>
<th>LN LF/HF.rest</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Rest</td>
<td>Rest</td>
<td>Rec0-5</td>
</tr>
<tr>
<td>FVO (rest)</td>
<td></td>
<td></td>
<td>r = -0.27 *</td>
<td></td>
</tr>
<tr>
<td>FBF (occlusion)</td>
<td></td>
<td></td>
<td>r = 0.38 *</td>
<td>r = 0.35 *</td>
</tr>
<tr>
<td>FVR (occlusion)</td>
<td></td>
<td></td>
<td>r = -0.35 *</td>
<td>r = -0.26 +</td>
</tr>
<tr>
<td>FVO (occlusion)</td>
<td></td>
<td></td>
<td>r = 0.23 +</td>
<td></td>
</tr>
</tbody>
</table>

* = p ≤ 0.05. + = p ≤ 0.15. FBF = Forearm Blood inflow. FVR = Forearm vascular resistance. FVC = Forearm venous capacitance. FVO = Forearm venous outflow.

Table 3.40 Bivariate Correlation Between Indicators of Autonomic Function & Vascular Function Indices in Older Women

<table>
<thead>
<tr>
<th>Vascular Function</th>
<th>SDNN</th>
<th>Pnn50</th>
<th>LN LFnu.rest</th>
<th>LN LF/HF.rest</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Rest</td>
<td>Rest</td>
<td>Rec0-5</td>
</tr>
<tr>
<td>FBF (rest)</td>
<td></td>
<td></td>
<td>r = -0.48 *</td>
<td>r = -0.40 *</td>
</tr>
<tr>
<td>FVO (rest)</td>
<td></td>
<td></td>
<td>r = -0.37 +</td>
<td></td>
</tr>
<tr>
<td>FVO (occlusion)</td>
<td></td>
<td></td>
<td>r = -0.35 +</td>
<td></td>
</tr>
</tbody>
</table>

* = p ≤ 0.05. + = p ≤ 0.15. FBF = Forearm Blood inflow. FVR = Forearm vascular resistance. FVC = Forearm venous capacitance. FVO = Forearm venous outflow.

Table 3.41 Bivariate Correlation Between Indicators of Autonomic Function & Vascular Function Indices in Young Women

<table>
<thead>
<tr>
<th>Vascular Function</th>
<th>SDNN</th>
<th>Pnn50</th>
<th>LN LFnu.rest</th>
<th>LN LF/HF.rest</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Rest</td>
<td>Rest</td>
<td>Rec0-5</td>
</tr>
<tr>
<td>FVR (Rest)</td>
<td></td>
<td></td>
<td>r = 0.27 +</td>
<td></td>
</tr>
<tr>
<td>FBF (occlusion)</td>
<td></td>
<td></td>
<td>r = 0.32 +</td>
<td>r = 0.34 +</td>
</tr>
<tr>
<td>FVR (occlusion)</td>
<td></td>
<td></td>
<td>r = 0.48 *</td>
<td></td>
</tr>
<tr>
<td>FVC (occlusion)</td>
<td></td>
<td></td>
<td>r = 0.52 *</td>
<td></td>
</tr>
<tr>
<td>FVO (occlusion)</td>
<td></td>
<td></td>
<td></td>
<td>r = 0.34 +</td>
</tr>
</tbody>
</table>

* = p ≤ 0.05. + = p ≤ 0.15. FBF = Forearm Blood inflow. FVR = Forearm vascular resistance. FVC = Forearm venous capacitance. FVO = Forearm venous outflow.
Similarly, linear regression analysis examining the relationship between autonomic indicators (i.e., LFnu Rec 0-5) and vascular function indices (i.e., FVR at Rec4’) from local min to local max is presented in the Table 3.41.

Table 3.42  Relationship Between Indicators of Autonomic (LFnu) & Vascular Function (FVR) After RE in Young and Older Women

<table>
<thead>
<tr>
<th>LFnu Rec0-5</th>
<th>Young</th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>β</td>
</tr>
<tr>
<td>FVR at Rec4’</td>
<td>r = 0.93 *</td>
<td>13.14 ± 0.99 †</td>
</tr>
</tbody>
</table>

*= p < 0.05. †= p < 0.0001. LFnu = Low frequency power during the recovery period 0-5. FVR = Forearm vascular resistance 4 minutes after resistance exercise.

3.6 Summary of the Results

Resistance exercise resulted in an increase in SBP, DBP, MAP and HR in both groups and under both exercise conditions. This was followed by a drop of the hemodynamic parameters towards or below pre-exercise levels. In general, the condition of exercise (5RM vs. 15 RM) influenced the hemodynamic responses. 15RM exercise resulted in greater systolic BPs at peak exercise and in the early phases of recovery. Interestingly, however, 5RM exercise resulted in higher values for the systolic BP recovery ratios.

With respect to age, the older women had higher SBP throughout the test, but had smaller magnitudes of immediate pressure recovery (both SBP and DBP) and therefore, tendency to higher 1’ and 3’ recovery ratios. Over the course of the entire recovery period, the older adults experienced a greater and earlier magnitude of post-exercise arterial BP drops. With respect to HR, the older adults had lower HR throughout the test, a blunted magnitude and rate of change of HR in comparison to their younger counterparts.
There was some indication that age tended to modify the effects of the exercise condition; however, the interaction terms did not quite reach statistical significance. In general the greater increase in BPs observed in the 15RM condition tended to be present only in the older adults. In addition the 5-RM condition tended to result in greater post-exercise DBP drops below pre-exercise level in the older women.

In addition, autonomic modulation of the heart appeared to be related to magnitude and rate of blood pressure recovery across all subjects. Of particular interest is the observation that sympathovagal balance (LFnu/HFnu) was directly related to rate and magnitude of blood pressure recovery (local min to local max) and FVR during recovery. Interestingly, the beta values for the regression of LFnu against FVR were about 50% higher in the young women as compared to the old. Also of note are the direct associations of FVR with the 1’ and 3’ recovery ratios, the inverse association of FBF with the 1’ and 3’ recovery ratios, and the direct relationship between venous outflows and post-exercise SBP drops below pre-exercise level.
4. DISCUSSION

The main purpose of this investigation was to examine the acute hemodynamic responses before, during, immediately after, and for 1 hour after a single bout of 5-RM and 15-RM bilateral knee-extension RE in healthy young and older adult women. In particular, this study examined the effects of test condition, exercise condition and age group, as well the age by exercise condition interaction on the magnitude, and rate of post-exercise BP drop below pre-exercise level following RE.

A secondary objective was to examine indicators of autonomic (SDNN, pnn50, LFnu, and LF/HF ratio) and vascular function (FBF, and FVR) before and after a single bout of 5-RM and 15-RM bilateral knee-extension RE in healthy young and older adult women. In particular, this study examined the effects of test condition, exercise condition, and age group, as well as the age x exercise condition interaction on indicators of autonomic and vascular function after RE. Furthermore, the final objective of this study was to describe associations between indicators of autonomic (SDNN, pnn50, LFnu, and LF/HF ratio) and vascular function (FBF, FVR, FVC, and FVO) and pattern of hemodynamic parameters after RE (magnitude and rate of BP drops below pre-exercise level).

4.1. Participant Characteristics

A total of 16 college-aged women enrolled in undergraduate classes at Louisiana State University in Baton Rouge, and 16 older adult women from St James Place Continuing Retirement Community in Baton Rouge participated in this study. Descriptive statistics for the participants indicated age-related differences in physical (weight, fat distribution) and physiological (SBP, MAP, FVR) characteristics, and fitness performance (muscle strength, estimated VO$_{2\text{max}}$).
4.2. The General Hemodynamic Responses During & Following RE

As expected, RE resulted in rapid increases in SBP, DBP, MAP and HR, similar in magnitude to data from previous investigations. $^{55, 61-63, 93-96}$ While, the observed mean peak exercise pressures ($149.1 \pm 22.1/86 \pm 15.5$ mmHg) and heart rate ($105.4 \pm 20.3$ beat/min) are close to those reported by Bermon $^{61}$ and MacDonald, $^{55}$ they are somewhat lower than those reported by McCartney et al $^{62}$ and MacDougall. $^{63}$ The seemingly disparate findings across investigations may be the result of differences in number of repetitions, absolute and relative load, and the ratio of muscle mass involved in the exercise. $^{94-97}$

The mechanisms suggested as potentially mediating the hemodynamic response to RE include a potent pressor response, mechanical compression of blood vessels in the contractile muscle, and elevated intrathoracic pressure coincident with the Valsalva maneuver. $^{63, 97}$ In addition, subsequent studies have acknowledged the moderating influence of other factors such as number of repetitions, absolute and relative load, and active muscle mass, and joint angle. $^{94, 97}$ The potent pressor response during dynamic resistance exercise likely involves signals arising in the brain (central command) and in the active skeletal muscle (pressor reflex). $^{99-100}$ Central command might first raise heart rate and cardiac output by vagal withdrawal. $^{99-100}$ Additionally, the activation of muscle mechano/metaboreceptors (muscle pressor reflex), traveling mostly via Group III and IV afferent fibers to the cardiovascular areas of the medulla, might initiate sympathetic activity to bring about further increases in heart rate and arterial pressure. $^{97, 99-102}$ Moreover, as the subject performs more repetitions and begins to fatigue, both the pressor reflex and central drive activities will increase, resulting in the recruitment of additional motor units, and greater cardiovascular responses $^{94, 97}$. The third mechanism potentially
mediating the behavior of the hemodynamic variables during RE is an increased intrathoracic pressure generated by the Valsalva maneuver. The increase in intrathoracic pressure is immediately transmitted to the arterial tree, resulting in increases in both systolic and diastolic pressures.

Immediately after the last repetition, the hemodynamic variables rapidly dropped close to (SBP, MAP, and HR) or below pre-exercise (DBP). The magnitude of decline in SBP (25.3 ± 15.8 mmHg), DBP (30.5 ± 13.9 mmHg), MAP (29.1 ± 12.1 mmHg), and HR (27.5 ± 16.6 beats/min) are in agreement with MacDougall et al and Hill et al. This may be the result a combination of factors including a large vasodilated muscle mass that was previously mechanically occluded and subsequent venous pooling and reduction of preload, as well as a transient pressure undershoot initiated by arterial and cardiopulmonary baroreflexes initiated during RE.

Immediately following the initial rapid drop in pressure to a “local min,” pressure rose slightly to a “local max”. This suggests the existence of a post-exercise pressure recovery overshoot phenomenon. While this phenomenon has not been previously described, its consistent appearance in our pilot studies and in the present data (Figure 2.1, 3.1, 3.6, 3.11, 3.16-3.18) suggests that this is a repeatable physiologic event. Although out of the scope of this study, it can be hypothesized that the increase to a local max reflects a reflexive post-exercise recovery pressure overshoot subsequent to a drastic drop in pressure, possibly mediated by arterial and cardiopulmonary baroreceptor activity. Consistent with this is our finding of a direct relationship between sympathetic activation immediately after RE (LFnu and LF/HF ratio at R0-5) and the magnitude and
rate of SBP recovery from local min to local max (Figure 4.1, Tables 3.28-3.31, and Section 4.5 for addition comments).

$$(r = 0.44; p = 0.0001)$$

**Figure 4.1** Relationship Between LFnu at Rec 0-5 and Mean Rate of SBP Recovery After RE in Young and Older Adult Women

In the assessments of hemodynamic variables throughout the long-term (i.e., for 1-hour after RE) recovery period, post-exercise arterial BP drops below pre-exercise level appeared to occur across all subjects (Figure 3.2). SBP dropped below pre-exercise level (~4 mm Hg) by about 10’ into the recovery period, and did not appear to change appreciably thereafter (R60’: ~5 mm Hg). DBP dropped below pre-exercise level (~3 mmHg) by about 1’ into the recovery period and continued to drop (R60’: ~6 mmHg) at a slow rate throughout the recovery period. MAP followed a pattern nearly similar to DBP dropping below pre-exercise level (~3 mm Hg) by about 5’ into the recovery period and continued to drop (R60’: ~6 mm Hg) at a slow rate throughout the recovery period. These results are in agreement with Hill et al, Brown et al, and MacDonald et al, but appear to disagree with O’Connor et al and Koltyn et al who found elevation in blood pressure following resistance exercise. Once again, the disparate findings of these latter
studies may be reconciled as differences in experimental design and measurement. Participants in the O’Connor study were engaged in activities of daily living during recovery (post-exercise ambulatory blood pressure) and Koltyn et al. reported SBP readings only at 3 and 5 minutes following the RE bout. 55-56

In general, the mechanisms underlying post-exercise arterial BP drops below pre-exercise level following exercise are not completely understood. However, neural (e.g., sympato-inhibition) 24-26, 46, 49-51, 56 and local (e.g., impairment of vascular responsiveness to $\alpha$ adrenergic receptor stimulation, and/or vasodilator substances) 24-26, 46, 49-51, 56 alterations in sympathetic vascular regulation are suggested as potentially mediating a sustained decrease in regional and systemic vascular resistance. 5, 24, 26, 46, 51, 56

In addition, other contributing factors may include 1.) vasodilation underlying post-exercise BP drop below pre-exercise level is not restricted to the sites of active skeletal muscles (i.e., the legs) but also involves inactive regions as well (e.g., the arms); 5, 24, 26, 46, 51 2.) the associated rise in arterial blood inflow through the vasodilated regions contributes to an increase in venous pooling of blood due to the absence of “muscle pump” during passive recovery from exercise; 26 3.) an increase in venous pooling, in conjunction with the loss of plasma volume associated with exercise, leads to a reduction in central venous pressure and cardiac filling pressure (preload); 26, 51 and 4.) despite the fall in cardiac preload, stroke volume is maintained due to the reduction in cardiac afterload and a probable increase in cardiac contractility 26, 46, 51; thus, the end result of these influences on the blood vessels and heart is that cardiac output is elevated (heart rate is higher and stroke volume is unchanged compared with pre-exercise condition).
Interestingly, our correlation analysis revealed a unique relationship between resting FVO and the magnitude of post-exercise SBP and DBP drop below pre-exercise level (Figure 4.2, Table 3.24, and Section 4.5 for addition comments). This relationship indicates that participants with greater resting FVO have a larger magnitude of arterial BP drop below pre-exercise level after RE, and suggests that venous function may be a contributing factor in the hemodynamic responses following exercise. However, the effect of venous function on the hemodynamic response following RE is out the scope of this study. Therefore, future research should examine the role of venous side on hemodynamic responses following RE (See Section 4.5 for addition comments).

![Figure 4.2 Relationship Between Resting Forearm Venous Outflow and Magnitude of SBP Drop After RE in Young and Older Women](image)

**Figure 4.2** Relationship Between Resting Forearm Venous Outflow and Magnitude of SBP Drop After RE in Young and Older Women
FVO = Forearm venous outflow. 20'-Magnitude PE SBP Drop = Magnitude of post-exercise systolic blood pressure drop 20’ after following resistance exercise.

The HRV data also revealed a general effect of resistance exercise (i.e., test condition effect). Of particular interest is the appearance of a lower than pre-exercise level LFnu and LF/HF ratio during the 5-minute (R1-6, R2-7, and R3-8) and 10 minute segments (R0-
10) immediately following RE, and the tendency thereafter for LFnu and LF/HF ratio to increase slightly throughout the recovery with the result being that both indicators of autonomic function were higher than pre-exercise level at the end of the long-term recovery period. Thus, the data from this investigation appear to suggest that a sympatho-inhibition of the heart period modulation after the cessation of the activity might be associated with the decline in BP below pre-exercise level after exercise. This finding is in agreement with previous studies indicating that sympathetic nerve activity is inhibited during post-exercise BP drop below pre-exercise level in human 4-5, 18-19, 24-26, 46 and some animal model 24-26, 48, 133-135. For example, Floras et al 4 reported significant reduction (10 mm Hg) in SBP and muscle sympathetic nerve activity in borderline hypertensive subjects 60 minutes after treadmill exercise. Likewise, simultaneous reductions in arterial BP and sympathetic nerve discharge (renal and splanchnic) have been observed after prolonged sciatic nerve stimulation in spontaneous hypertensive rats 24, 134-135 and pre-hypertensive Dahl salt-sensitive rats 24, 133. However, other studies have also indicated that the role of sympatho-inhibition activity in causing post-exercise BP drop below pre-exercise level is both complex and limited in normotensive humans 5 and some animal model 138. For example, Hara and Floras 5 reported that diastolic and mean arterial blood pressures were reduced, whereas muscle sympathetic nerve activity and plasma norepinephrine levels remained unchanged from control levels after sub-maximal exercise, suggesting that post-exercise BP drop below pre-exercise level can occur in normotensive humans in the absence of reductions in sympathetic nerve activity. VanNess et al 138 using a ganglionic receptor antagonist to block sympathetic outflow in rats reported > 85% reduction in post-exercise BP drop below pre-exercise level.
Halliwill et al.\textsuperscript{5} using $\alpha$-adrenergic receptor antagonist to block sympathetically mediated vasoconstriction in normotensive humans reported no change in post-exercise drop in BP below pre-exercise level, although a $\sim 30\%$ of drop in systemic vascular resistance could be attributed to loss of sympathetic vasoconstriction after exercise. In this context, it is becoming evident that sympatho-inhibition cannot account for all magnitude in BP drop below pre-exercise level after exercise in normotensive humans nor in some animal models. However, under other circumstances and/or conditions (i.e., health status, age, exercise modality, and gender) inhibition of sympathetic activity appears to be a determining factor for the magnitude, rate, and duration of post-exercise BP drop below pre-exercise level.

Muscle sympathetic nerve activity is under regulation by the arterial baroreflexes and cardiopulmonary receptor reflexes under resting conditions. The baroreflex is reset to a higher operating point and sympathetic activity is increased during exercise. However, these reflexes are reset to lower pressures such that sympathetic outflow from the central nervous system is lower than pre-exercise levels\textsuperscript{24-26,46}. Earlier studies in animals suggested that this sympatho-inhibition might be the result of activation of endogenous opioid receptor pathways in the central nervous system\textsuperscript{26,136}. However, it does not appear to be the case during post-exercise BP drop below pre-exercise level, in that the blockade of opioid receptors with naloxone does not alter post-exercise sympathetic nerve activity or arterial pressure in human\textsuperscript{5}, and the more recent research on this mechanism in animal has had inconsistent results\textsuperscript{26,137}. Thus, the central nervous system mechanism involved in baroreflex resetting during exercise and post-exercise BP drop below pre-exercise level are unknown.\textsuperscript{26}
Lastly, the FVR data were also subject to a significant effect of test condition. In general, post-exercise FVR were higher than pre-exercise level. While there is some evidence that in most persons, a post-exercise drop in arterial BP below pre-exercise level is due to a persistent drop in systemic vascular resistance, Hagberg et al suggest that the primary cause is a decrease in stroke volume, attributed to either reduced venous return or reduced myocardial contractility (systemic vascular resistance was elevated). Therefore, the present data can be reconciled as a drop in blood pressure due to a decrease in cardiac output that is not completely compensated for by increased vascular resistance. The present data are limited, however, in that a measure of cardiac output is not available, and therefore, future research designs should look to include measures of central CV function.

4.3. Moderating Effect of Resistance Exercise Condition (5-RM vs. 15-RM)

The exercise condition (i.e., 5-RM vs. 15-RM) appeared to influence the hemodynamic responses to the exercise. These effects are manifest primarily as main effects of the exercise condition; however, there were significant (MAP and HRV) and some nearly significant (SBP and DBP) exercise condition by test condition interactions.

In comparison to the 5-RM condition, the 15-RM condition essentially resulted in higher HR and SBP (effects on DBP and MAP were not quite significant) at peak exercise and in the early phases of recovery. These findings are consistent with those of MacDougall et al who documented that pressures reach progressively higher levels with subsequent repetitions, such that the highest peak recordings occur during the last completed repetition prior to the point of failure. Moreover, data indicate that pressures reach higher values when lifts at sub-maximal load are continued to failure than when a single maximum lifts (100% 1-RM) is performed.
It has been suggested that the moderating influence of the number of repetitions on arterial blood pressure during dynamic resistance exercise might be due to a combination of factors such as a greater voluntary effort, increased recruitment of additional motor units and accessory muscle to overcome fatigue, increased use of the Valsalva maneuver; and feedback from muscle ergoreceptors and nociceptors to the cardiovascular control areas in the medulla. 63, 97, 99-100

In addition to evoking greater pressure responses, 15-RM exercise was also followed by a greater magnitude of drop in DBP from peak exercise to local min. While the $p$-values for exercise condition effects on immediate decrease in MAP and SBP did not achieve significance, these are the first data to document the influence of resistance exercise condition on immediately post-exercise blood pressure drop. While direct evidence does not exist to support an hypothesis regarding the cause of the exercise condition effect, one might surmise that this might be due to a larger active muscle mass and subsequent greater venous pooling during recovery. 52, 63, 97

Similarly, these are also the first data to suggest a possible influence of different RE conditions on the post-exercise pressure recovery overshoot phenomenon. During post-exercise recovery, the 15-RM condition tended to result in a smaller magnitude of SBP “overshoot” ($p = 0.13$), and a larger magnitude of DBP “overshoot” ($p = 0.08$), from local min to local max. When one considers the larger drop in DBP from peak to local min following 15-RM, the finding of a nearly larger “overshoot” under these conditions supports the hypothesis that this phenomenon may be mediated by arterial and/or cardiopulmonary baroreceptors.
In the assessment of the hemodynamic variables during the long-term recovery period, three-way ANOVAs did not reveal significant main effects of exercise condition on post-exercise SBP, DBP, and MAP throughout the recovery period, or in the magnitude of drop below pre-exercise level in SBP, DBP, and MAP. However, there was some indication (pairwise comparisons) that the 15-RM condition resulted in greater blood pressure at R15’, R20’, and R50’. Thus, the pressure responses to 15-RM condition (in comparison to the 5-RM condition) can arguably be described as having a greater amplitude of change; that is the 15-RM was associated with greater peak responses during the exercise, a greater magnitude of drop immediately following the exercise, and generally higher pressures during the 1-hour recovery.

In contrast, the 5-RM exercise condition resulted in greater post-exercise SBP/peak ratios. This observation may be clinically relevant insofar as greater 1’ and 3’ post-exercise SBP/peak ratios are indicative of increased risk of cardiovascular disease. Therefore, one might surmise that 5-RM resistance exercise condition may involve greater cardiovascular risk as compared to 15-RM condition.

In addition, the three-way ANOVAs in the assessment of the hemodynamic variables during the long-term recovery period revealed significant (MAP and HRV) and nearly significant (SBP and DBP) indications of exercise condition by test condition interactions on the hemodynamic variables following RE. Of particular interest is that 5-RM exercise condition was followed by a more persistent and longer drop in MAP below pre-exercise level during the recovery period. While the p-values for exercise condition by test condition interaction on SBP and DBP did not quite reach statistical significance, these are also the first data to document the influence of RE condition on post-exercise BP drop
below pre-exercise level during a long-term (1-hour) recovery period. Although direct evidence does not exist to support an hypothesis regarding the cause of the RE condition effect on post-exercise BP drop below pre-exercise level, one might assume that greater neural (sympatho-inhibition)\(^{24-26, 46, 49-51, 56}\) and local (e.g., impairment of vascular responsiveness to \(\alpha\) adrenergic receptor stimulation, and/or vasodilator substances)\(^{24-26, 46, 49-51, 56}\) alterations in sympathetic vascular regulation, as well as larger active muscle mass and subsequent greater venous pooling during recovery\(^{52, 63, 97}\) might be suggested as potentially mediating a sustained decrease in regional and systemic vascular resistance following high intensity low volume RE (e.g., 5-RM condition) in comparison to low intensity high volume RE (e.g., 15-RM condition).

With regard to HRV, of particular interest was the exercise condition by test condition interaction on LFnu and LF/HF ratio indicating that the 15-RM condition was associated with lower than pre-exercise LFnu and LF/HF ratio (approximately during the first 20 minutes) and thereafter with higher than pre-exercise LFnu and LF/HF ratio. Conversely, the 5-RM condition was associated with higher than pre-exercise LFnu and LF/HF ratio at the end of the long-term recovery period (~50-minutes after the cessation of the activity). Thus, the data in this investigation seem to suggest that while the 15-RM condition was associated with an inhibition of the modulation of the heart early after the cessation of the exercise, the 5-RM condition was associated with a sympatho-inhibition of the heart period mostly all the way through the long-term recovery period. While the scope of the present investigation does not allow us to make strong inferences regarding whether, and the extent to which certain mechanisms may be involved in the moderating effects of exercise condition by test condition interaction on post-exercise sympathetic...
modulation of the heart, the present data are certainly consistent with an inhibition of the sympathetic activity following exercise, thereby resulting in greater magnitude of post-exercise BP drop below pre-exercise level.

4.4. Moderating Effect of Age

The age group of the participants was a mitigating factor in many of the hemodynamic responses to RE. These effects were noted as significant main effects of age and age by test condition interactions. There were also some nearly significantly age by exercise condition interactions, and possible three-way interactions (test condition x exercise condition x age).

As expected, there were significant main effects of age group in SBP, DBP, MAP, HR, HRV, and FVR. The older women had higher SBPs throughout the testing period, but had smaller magnitudes of immediate pressure drops (SBP, DBP, and MAP) manifest as, higher 1’ and 3’ recovery ratios. These findings are suggestive of heightened cardiovascular risk in the older women. Equally interesting is the observation that the older women eventually experienced earlier onset and greater magnitude of post-exercise drops in arterial BP below pre-exercise levels. Thus, one might hypothesize that while the older women appeared to be at greater cardiovascular risk in comparison to the young group, they also seemed to benefit more in terms of the potential clinical utility of the post-exercise hypotensive effect of exercise.

There was also some indication that age modified the main effects of the exercise condition (5RM vs. 15RM); however, the interaction terms did not quite reach statistical significance. Nonetheless, there were tendencies for higher SBP \( (p = 0.08) \), DBP \( (p = 0.07) \), and MAP \( (p = 0.06) \) during the 15-RM exercise condition (in the short-term assessment) and larger systolic \( (p = 0.16) \) and diastolic \( (p = 0.19) \) blood pressure drop
below pre-exercise level following 5-RM condition (in the long-term assessment) to be present only in older women (Figure 3.16-3.21). These nearly significant findings seem noteworthy and warrant further investigation, and were also reflected in the nearly significant 3-way interaction suggesting that the older women tended to have greater magnitudes and rates of change in pressure through the exercise period and in the early phase of recovery following the 15-RM condition (Figure 3.16-3.18), and a greater and earlier onset of post-exercise BP drop below pre-exercise level following the 5-RM condition (Figure 3.19-3.21). In contrast, hemodynamic responses among the young women were independent of the exercise condition.

Therefore, the hemodynamic data indicate that older women have higher blood pressure than younger women, and experience an earlier onset and greater magnitude of post-exercise drop in arterial BP below pre-exercise level. In addition, the hemodynamic responses in older women tend to be influenced by exercise condition such that 15 RM results in higher blood pressure during exercise (peak exercise) and in the early phase of recovery (local min and local max), while 5RM results in larger magnitudes of post-exercise drop in arterial BP below pre-exercise level, but the hemodynamic response of the younger women do not appear to be dependent upon exercise condition as defined in the present investigation.

To our knowledge, the appearances of age-related differences in the hemodynamic responses during and immediately following dynamic RE, as well as in the magnitude and rate of post-exercise drop in arterial BP below pre-exercise level have not been documented prior to this report. Thus, direct evidence does not exist to support an hypothesis concerning the cause of age-related effects. However, age-related changes in
hemodynamic responses were not unexpected in light of changes in central and local cardiovascular control mechanisms with age. Such changes include: 1.) altered autonomic control characterized by diminished autonomic tone, impaired responsiveness to beta-adrenergic stimulation (i.e., changes in adrenergic receptor sensitivity), and/or changes in the intrinsic sinus node rate; 79, 105 2.) increased end-diastolic volume and stroke volume that might compensate for age-associated reduction in HR and thus averting a decline in cardiac output; 107 3.) increased stiffness of large arteries due to intrinsic (biochemical) alterations and/or extrinsic (neurohumoral) factors; 79, 105-106, 108-111, 119 4.) increased peripheral vascular resistance as a result of less responsiveness of the arterial wall smooth muscle to β-adrenergic stimulation, while alpha-adrenergic responsiveness remains intact; 104, 118 5.) changes in the cardiovascular-acting hormones (plasma epinephrine concentration, rennin-angiotensin-aldosterone system, vasopressin, and atrial natriuretic peptide) involved in short- and long-term regulation of blood pressure; 112 6.) impaired arterial and cardiopulmonary baroreceptor functions; 112 perhaps due to decreased vagal and sympathetic components; and 7.) impaired venous function due to a decrease in venous capacitance and venous outflow. 109, 115-117

More interestingly, our correlation analysis revealed an unique relationships between FVO (at rest and after arterial occlusion) and the magnitude of post-exercise SBP drop below pre-exercise condition among the older women (Figure 4.3, and Table 3.25). Such relationship would suggest that older women with greater venous function exhibited larger magnitudes of SBP drop below pre-exercise after RE (Figure 4.3, Table 3.25, and Section 4.5 for additional comments). However, the effect of venous function in the magnitude of BP drop below pre-exercise level is out the scope of the present
investigation, and additional research is required to understand the moderating effect of venous side on the hemodynamic response following RE

Figure 4.3  Relationship Between Forearm Venous Outflow After Arterial Occlusion and Magnitude of SBP Drop After RE in Older Women
FVOocc = Forearm venous outflow after arterial occlusion. 20’- Magnitude PE SBP Drop = Magnitude of post-exercise systolic blood pressure drop 20’ after following resistance exercise.

Similarly, it is of particular interest that older women seem to have less increase in BP response during the exercise period with the 5-RM condition as compared to the 15-RM condition and the young group. Accordingly, previous studies have suggested the moderating influence of the number of repetitions on arterial BP during dynamic RE, which might be a result of a combination of factor, such as a greater voluntary effort; increased recruitment of additional motor units, and accessory muscle to overcome fatigue; increased use of the Valsalva maneuver; and feedback from muscle ergoreceptors and nociceptors to the cardiovascular control areas in the medulla. However, given that (1) there were no differences in the rate of perceived exertion (RPE) reported for both age groups during both muscle strength tests (indicating similar effort for both
group), and (2) the difference in BP response between both exercise conditions during the exercise period might be a true hemodynamic response to RE in older women, it might be hypothesized that the greater magnitude of BP response would be either a physiological reason or a more conservative approach by the tester. A more conservative approach by the tester was not the case in this study, so that it is hypothesized that a reduced pressor response as a result of a decline in the generation or conduction of the stimulus involved in the pressor response potentially mediating the hemodynamic response to RE might be the potential reason by which older women exhibited a diminished arterial BP response to the 5-RM condition in comparison to the 15-RM condition and young group.

While the scope of the present investigation does not allow us to make strong inferences regarding whether, and the extent to which certain mechanisms may be involved in the moderating effects of age on post-exercise drop in blood pressure, the present data are certainly consistent with a lower reactivity of blood vessels in older women following exercise, thereby resulting in smaller increases in vascular resistance, a greater magnitude of post-exercise BP drop, and a slower and smaller adjustment during blood pressure recovery “overshoot” from local min to local max. Linear regression analysis revealed that lnLFnu was related to recovery of MAP (r = 0.66 and 0.83; p<0.001 in young and older women, respectively) and FVR (r = 0.93 and 0.95; p<0.001 in young and older women, respectively); however, the beta values were higher among young (MAP beta= 0.95, FVR beta= 13.14) in comparison to older women (MAP beta= 0.51, FVR beta= 9.82), such that the magnitudes of change in arterial BP and FVR to the corresponding change in sympathetic activation of the heart were approximately
50% greater among the young group as compared to older adult women (Figure 4.4-4.6, and Table 3.32 and 3.41).

Thus, these findings support the notion that local (e.g., impairment of vascular responsiveness to $\alpha$-adrenergic receptor stimulation, and/or vasodilator substances) alterations in sympathetic vascular regulation might potentially mediating a sustained decrease in regional and systemic vascular resistance in older women. Thereby, the age group differences in post-exercise drop in arterial BP below pre-exercise level after RE, characterized by a greater drop in BP in older adults, may be attributed to smaller increases in vascular resistance in this age group.

**Figure 4.4** Relationship Between LFnu at Rec 0-5 & Magnitude of MAP Recovery After RE in Young and Older Adult Women

In LFnu.0-5 = Natural log of low frequency power of normalized unit during the first 5 minute of recovery period immediately after RE. Magnitude of MAP Rec = Magnitude of mean arterial pressure recovery from local min to local max.
Lastly, regardless of the physiologic causes of these age-related differences, the relevance of this investigation goes to risks associated with RE in older adults. The present data appear to suggest a greater risk to RE among the older women to the extent
that the recovery ratios are reasonable risk markers of cardiovascular status and recovery efficiency following exercise. Findings from this investigation revealed greater recovery ratios among the older women as compared to young group. Thus, it should be of additional investigation considerations about the RE among the older women.

4.5. Relationships Between Indicators of Autonomic and Vascular Function and Hemodynamic Responses During and Following RE

4.5.1. Autonomic Function & Hemodynamic Responses During and Following RE.
Bivariate correlations revealed significant relationships between indicators of autonomic function (i.e., SDNN, pnn50, LFnu, and LF/HF ratio) and patterns of SBP before and immediately after RE. There was a significant relationship between resting SDNN and mean pre-exercise SBP (Extended Results Section, Figure B.21), and pnn50 and mean pre-exercise SBP (Table 3.28) such that those participants with smaller SDNN and pnn50 exhibited a higher mean pre-exercise SBP. This finding is in agreement with previous studies indicating that participants with smaller variation of the heart period exhibited higher resting SBPs. Likewise, the results revealed a significant correlation between resting LFnu and LF/HF ratio and the magnitude and rate of SBP recovery immediately after RE (Table 3.8, and Extended Results Section, Figure B.22). More interesting, the results revealed significant relationships between LFnu and LF/HF ratio at Rec0-5 and the magnitude and mean rate of SBP recovery immediately after RE (Figure 4.1, Table 3.8, and Extended Results Section, Figure B.25-B.25). As previously mentioned, such relationships would suggest a direct association between sympathetic activation of the heart immediately after RE and the magnitude and rate of SBP recovery during the BP recovery overshoot phenomenon (Table 3.28-3.31) indicating that participants with greater sympathetic activation exhibited larger and faster magnitude and rate of SBP
recovery immediately after the cessation of activity. Therefore, this association appears also to indicate that BP recovery overshoot phenomenon would be a reflexive response to the drastic drop in BP immediately after the cessation of exercise. These data are unique in that no published studies have addressed this specific question.

Similarly, linear regression analysis revealed that logarithmic transformation (natural log function) of LFnu and LF/HF ratio at Rec0-5 were related to magnitude and rate of recovery of SBP, DBP, and MAP in young and older adult women. However, the beta values were higher among the young group in comparison to the older adult women such that the ratios of magnitude of change in SBP, DBP, and MAP to the corresponding magnitude of change in sympathetic activation of the heart were greater among the young group (~50%) as compared to older adult women (Figure 4.4- 4.5, and Table 3.22).

4.5.2. Vascular Function Indices & Hemodynamic Responses During and Following RE.

Bivariate correlations revealed significant relationships between vascular function indices (i.e., FBF, FVC, and FVO) and patterns of arterial BP (i.e., SBP, and DBP) response before, immediately after, and during the long-term recovery period following RE. Pooling both young and older adult data, the results revealed a unique relationship between resting FVO and the magnitude of post-exercise SBP drop below pre-exercise level. More interestingly, splitting the data by age group, only the older women exhibited this unique relationship between FVO (at rest and following arterial occlusion) and the magnitude of post-exercise SBP drop below pre-exercise level. Such relationship would suggest that older participants with greater venous function exhibited larger magnitude of post-exercise SBP drop below pre-exercise level. Thus, this unique relationship between venous measurements and BP response following RE appears to suggest venous function
as a potential contributing factor in post-exercise BP drop below pre-exercise among the older women. However, while out the scope of this study and the lack of previous studies, assumptions about the role of venous function on the hemodynamic response after RE require additional investigation.

Additionally, the results among the young group revealed significant associations between FVC following arterial occlusion and the magnitude of SBP increase with RE, and FVC following arterial occlusion and the magnitude of SBP decline immediately after RE. Such relationships would also suggest a direct relationship between venous function and hemodynamic response (magnitude of SBP increase and decline) during and immediately following RE. However, the effect of venous function on the hemodynamic responses during and immediately after the cessation of the activity is out the scope of this study, so that additional research is required to understand the role of venous side on the hemodynamic response during and following RE.

Similarly, bivariate correlations pooling both age group data together revealed significant associations between resting FVO and magnitude of DBP increase, resting FVO and magnitude of DBP decline, and resting FVO and mean rate of DBP decline. Such relationships would suggest that participants with larger resting FVO exhibited larger magnitudes of DBP increase during exercise period, and larger and faster magnitude and rate of DBP decline after the cessation of the activity. These relationships would suggest also direct associations between venous function and the hemodynamic responses during and following RE.

Likewise, the results indicated an unique relationship between resting FVO and the magnitude of post-exercise DBP drop below pre-exercise level, such that participants
with larger resting FVO exhibited larger magnitudes of post-exercise DBP drop below pre-exercise level. Once again, this unique relationship between venous measurements and BP response after RE appears to suggest venous function as a potential contributing factor in post-exercise BP drops below pre-exercise level among the older women. However, additional information is required to figure out the role of venous system on the magnitude of BP drop below pre-exercise level following resistance exercise.

4.5.3. Autonomic Function & Vascular Function Indices. Bivariate correlations pooling both young and older group data revealed significant associations between SDNN and FVO at rest and following arterial occlusion, SDNN and FBF following arterial occlusion, and SDNN and FVR following arterial occlusion, such that participants with smaller SDNN (mostly older adult women) exhibited smaller FVO at rest and following arterial occlusion, lower FBF following arterial occlusion, and higher FVR following arterial occlusion. Further, the results revealed a relationship between pnn50 and FBF after arterial occlusion, and pnn50 and FVR after arterial occlusion such that participants with smaller pnn50 exhibited smaller FBF and larger FVR after arterial occlusion.

In addition, splitting the data by age group, the results among the older women revealed significant associations between SDNN and resting FBF, pnn50 and resting FBF, and pnn50 and FVO at rest and after arterial occlusion. Such associations would suggest that older adult women with smaller SDNN exhibited lower resting FBF, and with smaller pnn50 lower resting FBF and FVO at rest and after arterial occlusion. Further, the results among the young group revealed associations between (1) SDNN and FBF and FVO following arterial occlusion such that young participants with smaller SDNN exhibited smaller FBF and FVO following arterial occlusion, (2) tendency
towards associations between pnn50 and resting FVC, pnn50 and FBF after arterial occlusion, and pnn50 and FVO after arterial occlusion, such that young participants with smaller pnn50 exhibited smaller resting FVC, and FBF and FVO after arterial occlusion, and (3) significant associations between LFnu and FVR at rest and after arterial occlusion, and between LFnu and FBF and FVC after arterial occlusion such that young participants with higher LFnu exhibited higher FVR at rest and after arterial occlusion, smaller FBF after arterial occlusion, and higher FVO after occlusion, respectively.

Similarly, linear regression analysis revealed that logarithmic transformation (natural log function) of LFnu and LF/HF ratios at recovery period 0-5 were related to FVR in young and older adult women. However, the beta values were higher among the young group in comparison to the older adult women such that the magnitudes of change in FVR to the corresponding change in sympathetic activation of the heart was greater among the young group (~50%) as compared to older adult women (Table 3.41). Thus, the present data appear to be consistent with a lower reactivity of blood vessels in older women following exercise, thereby resulting in smaller increases in vascular resistance, a greater magnitude of post-exercise BP drop, and a slower and smaller adjustment during blood pressure recovery “overshoot” from local min to local max.

4.6. Limitations of the Study

The potential limitations of this investigation are (1) the uncertainty of the pre-exercise hemodynamics reflecting a true baseline measure, (2) the use of resting forearm vascular resistance is valid and reliable, and (3) the assumption that HRV findings from a nonstationary heart period (the 5-minute segment immediately after exercise) is valid indicator of autonomic modulation of the heart.
As to the first limitation, it is a known common occurrence that arterial BP elevates right before exercise due to an anticipatory response. Therefore, the pre-exercise measurements in the current study may not represent a “true baseline condition.” Accordingly, in this study several strategies were adopted to minimize such an effect. First, participants were familiarized with the personnel and procedure over the course of the first three visits as suggested by Kaufman et al. Second, the pre-exercise data collection started following approximately 20 minutes of seated quiet rest, and ended at least 2 minutes prior the exercise period, and prior to the participant receiving any information as to the upcoming work bout. Third, the pre-exercise condition was standardized by asking the participants to engage in reading magazines having no violent or controversial content. Last, the testing procedures were performed in a private room with temperature adjustment control.

To further address the issue of anticipatory effects on pre-exercise blood pressure, we examined the 12 minutes of pre-exercise data, post facto, by splitting the 10-minute resting period into two 5-minute segments and comparing the blood pressures and heart rates with those observed during the 2-minute period just prior to exercise. The data reveal an interesting age by exercise condition on the several hemodynamic variables, which are presented in Table 4.1. On the basis of the results of this analyses the hemodynamic data farthest removed from the start of exercise was used as our pre-exercise measurement. However, it cannot be stated with absolute certainty that the pre-exercise data are truly “resting blood pressures”.

93
Table 4.1  Pre-exercise Hemodynamic Variables Assessment

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th></th>
<th>Older</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>5-RM</td>
<td>15-RM</td>
<td>Average</td>
<td>5-RM</td>
</tr>
<tr>
<td>SBP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$5_1'$</td>
<td>106.2 ± 3.8</td>
<td>106.3 ± 3.8</td>
<td>106.2 ± 2.7</td>
<td>121.1 ± 3.8</td>
</tr>
<tr>
<td>$5_2'$</td>
<td>108.3 ± 3.7</td>
<td>107.6 ± 3.7</td>
<td>108.0 ± 2.6</td>
<td>120.5 ± 3.7</td>
</tr>
<tr>
<td>$2_{\text{prior}}$</td>
<td>117.3 ± 3.7 *‡</td>
<td>108.9 ± 3.7</td>
<td>113.1 ± 2.6 *‡</td>
<td>123.6 ± 3.7</td>
</tr>
<tr>
<td>DBP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$5_1'$</td>
<td>58.4 ± 2.9</td>
<td>58.9 ± 2.9</td>
<td>58.6 ± 2.0</td>
<td>59.3 ± 2.9</td>
</tr>
<tr>
<td>$5_2'$</td>
<td>60.8 ± 2.6</td>
<td>60.3 ± 2.6</td>
<td>60.6 ± 1.8</td>
<td>58.7 ± 2.6</td>
</tr>
<tr>
<td>$2_{\text{prior}}$</td>
<td>63.8 ± 2.7</td>
<td>60.7 ± 2.7</td>
<td>62.3 ± 1.9</td>
<td>61.2 ± 2.7</td>
</tr>
<tr>
<td>HR</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$5_1'$</td>
<td>70.8 ± 3.3</td>
<td>71.1 ± 3.3</td>
<td>71.0 ± 2.4</td>
<td>62.4 ± 3.3</td>
</tr>
<tr>
<td>$5_2'$</td>
<td>73.3 ± 3.2</td>
<td>72.7 ± 3.2</td>
<td>73.0 ± 2.3</td>
<td>63.0 ± 3.2</td>
</tr>
<tr>
<td>$2_{\text{prior}}$</td>
<td>76.9 ± 3.5 *‡</td>
<td>78.9 ± 3.5</td>
<td>77.9 ± 2.5 *‡</td>
<td>64.4 ± 3.5</td>
</tr>
</tbody>
</table>

Values are mean ± SE. SBP = Systolic blood pressure (mm Hg). DBP = Diastolic blood pressure (mm Hg). HR = Heart rate (beat/min). * = Different from the first 5-minute period ($p < 0.05$). ‡ = Different from the second 5-minute period ($p < 0.05$).

A second limitation is the use of forearm vascular resistance as an indirect measure of the true resistance. Forearm vascular function indices were derived using a noninvasive procedure (i.e., mercury strain-gauge phethysmography technique) based on the assumption that alterations of pressures in strategically placed cuff allows the assessment of the rate of change of limb volume, which is thought to reflect indices of vascular function, such as forearm blood inflow, vascular resistance, venous capacitance, and venous outflow. However, the volumes that make up the arm include various compartments (e.g., arterial, venous, and interstitial) that are fairly steady at baseline condition, but following a dynamic state (e.g., exercise) the above-mentioned compartments have and may be continuing to change. Further, the between-day resting
forearm blood inflow measures are quite unreliable as indicated in our laboratory, other laboratory (Sinoway and Mush), and this study data. Accordingly, our forearm vascular resistance data cannot be used with a high degree of certainty as a measure of vascular resistance. As a result, our findings that post-exercise BP drop below pre-exercise level following exercise is associated to a diminished systemic and peripheral vascular resistance, while consistent with previous studies, should be considered with caution and require additional investigation.

The final limitation of this study that will be discussed is the assumption that HRV data immediately after RE (Rec0-5) are valid, despite the nonstationary behavior of the data. Certainly, one of the underlying assumptions of the HRV analyses is the stationarity of heart period data since the presence of slow or irregular trends in the series can potentially distort analyses and lead to misinterpretation. However, while more research is necessary to clarify the consequences of violations of stationarity and determinants of the magnitude of resulting biases, data suggest that moderate violations of stationarity do not necessary affect respiratory sinus arrhythmia measures. Moreover, several previous studies have used a comparable approach for examining patterns of HRV profiles during similar conditions. Accordingly, we recognize that our findings on modulation of heart period indices during a nonstationary condition (Rec0-5) should be also considered with caution.

4.7. Summary and Conclusions

The specific aims of this investigation were (1) to examine the effects of test condition, exercise condition, and age group, as well as the age by exercise condition interaction effect on the magnitude and rate of post-exercise BP drop below pre-exercise level after a single bout of 5-RM and 15-RM bilateral knee extension RE in young and older adult
women; (2) to examine the effects of test condition, exercise condition, and age group, as well as the age by exercise condition interaction effect on indicators of autonomic and vascular function before and after a single bout of 5-RM and 15-RM bilateral knee extension RE in young and older adult women; and (3) to describe associations between indicators of autonomic (i.e., heart rate variability profiles) and vascular function (i.e., FBF, FVR, FVC, and FVO) and pattern of post-exercise hemodynamic parameters (i.e., magnitude and rate of arterial BP drops below pre-exercise levels).

With respect to the first aim, data from the current study demonstrate that test condition, exercise condition and age group had main effects on the hemodynamic response during and following RE (during, immediately after, and throughout 1-hour recovery period). However, there was some indication that age group tended to modify the effects of test condition and exercise condition, such that the hemodynamic response through all testing period was exercise condition dependent among the older women but not among the young group, and the magnitude and rate of change of the hemodynamic variables were smaller and slower among the older women. It was of particular interest that the earlier and greater magnitudes of post-exercise SBP and DBP drops below pre-exercise level were intensity-dependent among the older women, but not among the young women. Also noteworthy was the identification of a pressure recovery overshoot phenomenon following the immediate drop in pressure after exercise. While we can only hypothesize as to the physiologic explanation of the hemodynamic responses, the clinical relevance of this study is meted out in the higher 1’ and 3’ post-exercise pressure recovery ratios that appear to serve as markers of cardiovascular disease.
With respect to the second aim, data from the current investigation also demonstrate that test condition, exercise condition, and age group had main effects on indicators of autonomic (HRV profiles) and vascular (FVR) function following RE. Of particular interest is the appearance of a lower than pre-exercise level sympathetic modulation of the heart during the first 10 minutes following the cessation of exercise, which is followed for a recovery towards and above pre-exercise level through the rest of recovery period. However, there were indications that exercise condition by test condition, and age group by test condition interactions modified the effect of test condition such that the recovery of sympathetic modulation of the heart towards and above pre-exercise level after exercise was earlier following the 15-RM condition (~20 min) and among the young group (~20 min) as compared to the 5-RM (~50 min) and their older adult counterparts (~40 min). However, there was neither an indication of an age by exercise condition interaction nor age by exercise condition by test condition interaction on the indicators of sympathetic modulation on the heart. The time domain data, as previously mentioned, are more difficult to interpret in so far as the increase in SDNN and pnn50 are representative of non-stationary time signals that occur with exercise. Otherwise, pre-exercise did not appear to be different from recovery. With regard to FVR, there was a main effect of test period indicating that post-exercise FVRs were higher than pre-exercise level throughout the recovery period, and a main effect of age group indicating that young women had higher post-exercise FVR in comparison to the older women. However, it was of particular interest a significant three-way (test condition x age group x exercise condition) interaction on post-exercise FVR, such that FVRs were influenced by exercise condition in the young adults, but not in the older adults. In general, post-exercise FVRs
after the 5-RM condition were higher than pre-exercise level at all recovery time points (except R7’) among the young group, but only in a few testing time (R4’, R20’, and R40’) among the older group. Further, post-exercise FVRs after the 15-RM condition were higher than pre-exercise level at R4’, R7’, R30’, R40’, and R60’ among the young group, but lower than pre-exercise level at R7’ and R30’ among the older women. Consequently, data from this investigation appear to support the notion that the greater magnitude of post-exercise arterial BP drop below pre-exercise level is mediated by smaller increases in systemic and peripheral resistance, in addition to an apparent decrease in heart rate through the recovery time. Thus, the greater post-exercise arterial BP drop below pre-exercise level among the older women may be mediated by a lack of regional responsiveness (due to a modification of the sensitivity or number of beta-adrenergic receptor) to increase systemic and peripheral vascular resistance. Moreover, the similar behavior of lnLFnu across age groups, paired with age-related differences in the LFnu vs. MAP and FVR slopes may be consistent with older adults having stiffer and less reactive blood vessels, possibly contributing to a greater magnitude of post-exercise BP drop in older women.

Finally, the third aim revealed significant association between indicators of autonomic and vascular function and patterns of hemodynamic response during and following RE. Of particular interest were: (1) the direct association between sympathetic modulation of the heart at Rec0-5 and the magnitude and mean rate of SBP recovery from local min to local max suggesting that the BP overshoot recovery phenomenon described in this investigation would be a reflexive response to the drastic drop in BP immediately after the cessation of exercise, and that the ratios of magnitudes of change in
arterial BP to the corresponding magnitude of change in sympathetic activation of the heart were greater among the young group (~50%) as compared to older adult women; (2) the unique association between FVO and post-exercise BP drop below pre-exercise level (venous measurements and BP response following RE) suggesting venous side as a potential contributing factor in post-exercise SBP drops below pre-exercise level; and (3) the associations between sympathetic modulation of the heart (i.e., LFnu and LF/HF ratios) and FVR such that participant with higher sympathetic activity exhibited higher FVR; and (4) the direct association between sympathetic modulation of the heart and FVR following RE such that the magnitudes of change in FVR to the corresponding change in sympathetic activation of the heart was greater among the young group (~50%) as compared to older adult women. Thus, this last direct association also support the notion that greater magnitudes of post-exercise arterial BP drop below pre-exercise level is due to a smaller increase in systemic and peripheral resistance, in addition to an apparent decrease in heart rate through the recovery time.

We summarize these results as meaning that RE resulted in an increase in BP and HR. This was followed by a drop in BP below pre-exercise level throughout the recovery period. The 15-RM condition resulted in higher SBP at peak exercise and in the early phase of recovery; however, the 5-RM condition resulted in greater 1’ and 3’ post-exercise SBP recovery ratios. ANOVA also revealed that older women had higher SBP throughout the testing period, and higher 1’ and 3’ recovery ratios. However, the older women experienced earlier and greater drops in BP during the recovery period. Forearm vascular resistance after RE was subject to a test condition by age interaction, increasing above pre-exercise only in the young group. Pearson correlation revealed that low-
frequency variations in HR (LFnu) were related to recovery of arterial BP and FVR; however, the beta value was higher among young in comparison to older women. Age-group difference in post-exercise BP drop, characterized by a greater decline in BP in older adults, might be attributed to smaller increases in vascular resistance in older women.

Future research should address: (1) the present study limitations (anticipatory response, validity and reliability of the measure of vascular resistance, and magnitude and determinants of resulting biases from non-stationary heart period data); (2) the potential mechanisms and determinant factors mediating BP recovery overshoot phenomenon immediately following RE; (3) the effect of other potential determinant factors (i.e., venous function, gender, body position during recovery position, exercise duration, participant’s fitness and health status, and exercise training) on the magnitude, rate, and duration of post-exercise hemodynamic responses following RE; and (4) the usefulness of post-exercise arterial BP (SBP, DBP, and MAP) recovery ratios as a marker of cardiovascular events following resistance exercise, and presence of cardiovascular disease.
REFERENCES


APPENDIX A. BACKGROUND AND SIGNIFICANCE

A.1. Previous Work.

There has long been an interest in examining hemodynamic responses during the application of physical\textsuperscript{1-26} and mental\textsuperscript{27-29, 44} stressors. Over the past decade, clinical scientists have also expressed a burgeoning interest in the examination of the occurrence, magnitude, and duration of specific changes on cardiovascular regulatory system after the cessation of specific stressor applications\textsuperscript{1-29}. Of specific interest here is the sudden and prolonged reduction of arterial BP below pre-exercise level after a single bout of resistance exercise.\textsuperscript{1-26}

Underscoring this area of investigation is (1) the observation that sudden BP drop below pre-exercise level after exercise may be related to increased risk of cardiovascular events after acute exercise;\textsuperscript{24-26, 29, 31} (2) the delayed BP recovery has been implicated as a risk factor for the development of cardiovascular disease\textsuperscript{30-32} and hypertension;\textsuperscript{33-34, 43} and (3) the suggestion that post-exercise BP drop below pre-exercise level (as part of cardiovascular recovery), at sufficient magnitude and duration, might be of some clinical value in the non-pharmacological treatment of hypertension.\textsuperscript{24-26}

The rate of the decrease in systolic blood pressure (SBP) during recovery is usually fairly rapid after dynamic aerobic exercise.\textsuperscript{74, 75} However, in studies examining post-exercise SBP after similar exercises in patients with or without significant coronary artery disease (CAD)\textsuperscript{30-31, 76-77} and/or hypertension\textsuperscript{34, 77} some patients showed either a lesser decrease in SBP during the recovery,\textsuperscript{30} or higher SBPs in the recovery period than those in the peak-exercise pressures.\textsuperscript{30, 76-77} The patients who exhibit this phenomenon often had severe CAD, as determined by selective coronary artery angiography.\textsuperscript{30-31, 76-77}
In addition, these studies derived SBP recovery ratios by dividing the SBPs at 1, 2, and 3 minutes after exercise by the peak exercise SBPs. The 1, 2, and 3 min ratios in the subjects without CAD or hypertension declined steadily (e.g., 0.85±0.07, to 0.79±0.06 and to 0.73±0.06, respectively), while the ratios in the patients with CAD remained elevated (e.g., 0.97±0.12 to 0.97±0.11 to 0.93±0.13 \(^{75}\)). Further, recovery ratios at the optimal cutoff point (e.g., greater than 1.0, 0.9, and 0.8 at 1, 2, and 3 min of recovery, respectively) were used to identify patient with CAD, which in some cases was performed with a degree of sensitivity of 95%, and a specificity of 90%. \(^{75}\) Similarly, the SBP ratio was greater in patients who had a greater number of diseased coronary arteries, and was highest in patients with a narrowing of the left main coronary artery. Thus, it was suggested that abnormal SBP response after exercise in patients with CAD is indicative of severe myocardial ischemia during exercise, and may be caused by an increase in stroke volume due to recovery from myocardial ischemia and increased systemic vascular resistance secondary to exaggerated sympathetic nervous activity. \(^{30}\)

Post-exercise BP drop below pre-exercise level has been well documented in normotensive \(^{6-7}\) and hypertensive individuals \(^{1-4}\) in response to several types of dynamic exercise (e.g., walking and running, \(^{1-9}\) leg exercise, \(^{10-16}\) and swimming \(^{17}\)), as well as in several animal models (e.g., spontaneously hypertensive rats, and electrical stimulation of somatic and muscle afferents –post-stimulation hypotension-). \(^{20,24-26}\) The investigation of post-exercise BP drop below pre-exercise condition has focused on different attributes of the recovery curve, namely the occurrence or existence, magnitude, and duration of blood pressure drop. The decline in systolic and/or diastolic BP below pre-exercise levels after aerobic exercise has shown to be independent of exercise intensity, \(^{1-6,8-10,12,14,18-21}\)
exercise duration, 1-6, 9-12, 14, 18-23 exercising muscle mass, 35 body recovery posture, 26, 36-37 and thermal conditions. 26, 39 For example, post-exercise BP drop below pre-exercise level was observed in humans and rats following exercise at intensities between 40% and 70% of maximal oxygen consumption (VO$_2$$_{max}$). 1-6, 8-10, 12, 14, 18-20, 26 Further, arterial BP in humans is reduced after maximal treadmill and leg cycling exercise to exhaustion. 7, 11, 15-16 In animal model studies, post-stimulation hypotension occurs in response to electrical stimulation of the gastrocnemius and biceps femoris muscles at current intensities between four and twenty-five times the twitch threshold (between 3 and 25 mA). 24

Post-exercise BP drop below pre-exercise level was also observed with exercise durations as short as 3 to 10 minutes 13, 17 and as long as 170 minutes, 7 as well in numerous studies using a more typical exercise duration between 20 and 60 minutes. 1-3, 6, 9, 13-14, 20, 23-26 Post-exercise reductions in SBP and DBP were observed after arm (smaller-muscle mass) and leg (larger-muscle mass) ergometry activity at similar relative intensity and duration, 35 as well during supine, seated, and upright recovery position after aerobic exercise. 24-26, 36-37

In contrast to the occurrence of PEH, magnitude (the difference between post-exercise arterial BP value, and pre-exercise arterial BP values) and duration (the time period throughout recovery in which arterial BP values are maintained below pre-exercise level) of PEH after aerobic exercise appear to exist as a function of health history and age of the participants, 1-4, 6, 12, 15, 24-26, 38, 41-43 exercise intensity, 1, 14, 21, 24-26, 40-41 and duration, 20, 22, 24-26 exercising muscle mass; 24-26, 35 and the thermal environment conditions. 24-26, 39 The magnitudes in systolic and diastolic blood pressure (SBP and DBP, respectively) reductions after a single bout of aerobic exercise in humans were
observed from 18 to 20 and 7 to 9 mm Hg, respectively, in patients with established essential hypertension (sustained mild to moderate), \(^1\text{-}^3, 6, 8\text{-}9, 12, 14, 16\text{-}19, 26\) and 8 to 10 and 3 to 5 mm Hg, respectively, in young and middle-aged normotensive humans. \(^3, 5\text{-}8, 10\text{-}12, 14, 16\text{-}19, 21, 24\text{-}26, 41\)

Early observations revealed that PEH is related to exercise intensity \(^1, 40\) and exercise duration \(^20\) in hypertensive humans \(^1, 40\) and rats. \(^20\) For example, Hagberg et al \(^1\) reported that the magnitude and duration of PEH were greater after three bouts of 15-min treadmill exercise at 70\% versus 50\% of VO\(_{2\text{max}}\) in older (mean age, 64 years) human with essential hypertension. Overton et al \(^20\) reported similar observations (greater magnitude and duration) after 40-min as opposed to 20 min of treadmill exercise at the same intensity, in spontaneous hypertensive rats. \(^20\) Piepoli et al \(^40\) reported decrements in BP only after maximal cycle exercise (5-min stages of 25 Watt increments) as compared to moderate (5-min stages of 12.5 Watt increments) and minimal (constant 50 Watt) intensities of exercise in normotensive volunteers.

In contrast, recent studies reveal no differences in the magnitude and duration of PEH at different exercise intensities, \(^14, 21, 41\) and exercise durations \(^22\) in normotensive \(^21, 41\) and hypertensive \(^14\) participants. Pescatello et al \(^14\) reported in hypertensive participants no difference in the magnitude of PEH following 30-min bouts of cycle ergometry at 40\% and 70\% of maximal exercise capacity. MacDonald et al \(^21\) documented that both mild (50\% VO\(_{2\text{peak}}\)) and moderate (70\% VO\(_{2\text{peak}}\)) intensities of cycle ergometer exercise elicit similar magnitudes of PEH in a normotensive population. In addition, Forjaz et al \(^41\) observed no PEH differences after 45-min of cycle ergometer exercise at 30, 50, & 80\% VO\(_{2\text{max}}\) in normotensive active participants.
The potential factors that might explain the discrepancy in PEH observations after aerobic exercise include: (1) the use of intermittent auscultatory measurement techniques (in comparison to continuous BP monitoring), which might be unable to detect the oscillatory nature of BP in normotensive subjects; (2) the use of exercise protocols with different levels of effort and exercise modalities (e.g., treadmill and cycle ergometer) that might elicit differences in the hemodynamic responses; (3) the effect of body position (e.g., standing, upright seated) during pre-exercise and post-exercise BP measurements, which might add some orthostatic stress, and (4) the conditions in which the dependent variable is measured (controlled and quiet setting vs. free-living conditions/ambulatory BP), which might elicit differences among research findings.

In addition, other studies have examined the effects of exercising muscle mass and thermal environment conditions on the magnitude and duration of PEH following aerobic exercise. For example, a study examining the magnitude and duration of PEH after aerobic exercise in regard to the ratio of working-muscle mass (arm or leg exercise) showed a similar magnitude of PEH, but the duration of PEH was prolonged following the leg exercise. A study examining the effect of thermal environment conditions on PEH after cycling exercise observed that the magnitude and duration of PEH were greater in a warmer (35°C) than in a cooler (23°C) environment, and post-exercise reductions in DBP were greater than those observed for SBP in the warmer environment.

Lastly, studies investigating the duration of PEH have observed that post-exercise arterial BP reductions are maintained throughout different time periods. For example, in humans with mild to moderate essential hypertension, reductions in SBP and
DBP were maintained during the period between 2 and 3 hours after moderate cycling exercise.\(^{19}\) In older adults with essential hypertension, SBP was reduced below control values for 3 hours after exercise at 70% of VO\(_{2\text{max}}\).\(^{1}\) In borderline hypertensive humans, mean arterial pressure (MAP) was reduced for at least 4 hours after sub-maximal cycling exercise.\(^{13}\) In mildly hypertensive men, SBP was reduced over 8.7 hours and DBP over 12.7 hours after sub-maximal exercise.\(^{14}\)

Given that arterial blood pressure is a function of cardiac output time total peripheral resistance, post-exercise BP drop below pre-exercise level following aerobic exercise, or post-stimulation hypotension following electrical stimulation of somatic and muscle afferents, might result from decreases in cardiac output, total peripheral resistance, or both.\(^{24-26}\) Accordingly, it is generally accepted that, in most subjects, PEH following aerobic exercise is due to a persistent drop in regional and systemic vascular resistance, which is not completely offset by increases in cardiac output.\(^{11, 18-19, 24-26, 38, 40, 46-47, 51}\)

Findings from that viewpoint suggested that sustained vasodilation is associated with two alterations in sympathetic vascular regulation, which has been defined as a “neural” and a “vascular” or local component.\(^{24-26, 46-47, 51}\) The neural component of this vasodilation is a reduction in the outflow of sympathetic vasoconstrictor nerve activity to skeletal muscle vascular beds (i.e., sympatheoinhibition); and the vascular component is the attenuation of vascular responses to sympathetic vasoconstriction, as well the potential influence of local and circulating vasodilator substances.\(^{24-26, 46-47, 51}\) However, research in older humans with essential hypertension indicate that mechanisms underlying PEH (decrease in cardiac output) after aerobic exercise may be divergent from that of other population groups.\(^{1, 24-26}\) In this older adult group, PEH was mediated by a
decrease in cardiac output (systemic vascular resistance was elevated). The fundamental cause appeared to be a decrease in stroke volume that was attributed to either reduced venous return or reduced myocardial contractility. Nevertheless, it is suggested that this group was studied during recovery in a seated position, so that the apparent discrepancy in results may be related to interactions among aging, hypertension, and the superimposed stress of seated recovery position (i.e., orthostatic stress). In summary, the general view is that PEH arises from a reduction in systemic vascular resistance and not from a fall in cardiac output, but it appears to be that exceptions have been found.

While there is considerable evidence about PEH after aerobic activity, little and inconclusive information is available regarding the hemodynamic responses following dynamic resistance exercise (RE) in humans. Only a few studies have examined arterial BP responses after an acute bout of RE, monitoring arterial BP either in a controlled setting (inside of the laboratory-quiet and comfortable resting conditions) or under free-living conditions outside of the laboratory (ambulatory blood pressure). These interventions were made primarily using intermittent auscultatory BP method, limited to healthy younger adults (age range= 18-33 yrs), and starting BP recordings 1-minute after performing a RE workout session.

Studies using intermittent auscultatory BP method or continuous intra-arterial BP monitoring to examine post-exercise arterial BP response in a controlled setting observed the occurrence of post-exercise BP drop below pre-exercise level following an acute bout of RE at different time points within the recovery-period (e.g., ~10-seconds immediately after, or after 10-min following RE), with different exercise...
testing protocols, and during different body positions (e.g., standing, and upright seated ). However, this was not true for other studies involving similar testing procedures. For example, Hill et al examined standing BP response using intermittent auscultatory BP method in healthy younger men (age range=22 to 33 yrs; n=6) immediately after (~10 seconds), and at 1, 2, 15, 30 and 60-minutes following 3 sets of as many repetitions as possible on 4 free-weight exercises at 70% of 1-RM. In this intervention, reductions in both SBP and DBP were observed within the first 10-seconds of recovery. Thereafter, SBP and DBP abruptly returned toward control levels but remained slightly (systolic) to moderately (diastolic) depressed throughout 1-hour post-exercise recovery period. Brown et al assessed upright seated BP response using also auscultatory intermittent BP method in healthy younger men and women (mean age=20.5±1.5 yrs; n=7) at 2, 5, 10, 15, 30, and 60 minutes following 3 sets x 5 exercises at 40% 1-RM (20-25 repetitions), 70% 1-RM (8-10 repetitions), and 25-minute of submaximal dynamic exercise at 70% of heart rate reserve, during 3 separate days. Brown et al observed that upright seated SBP fell slightly below pre-exercise levels only after 10-min of recovery, and remained low throughout 60-min of recovery. For both exercise modalities (RE and dynamic exercise), DBP recovered to pre-exercise values, but remained slightly lower than pre-exercise values for up to 60-min of recovery for both exercise modalities. Recently, MacDonald et al examined post-exercise upright seated arterial BP using continuous intra-arterial BP monitoring in healthy younger men (mean age=24.3±2.4 yrs; n=13) at 1.5, 3, 5, 10, 15, 30, 45, and 60-minutes following 15-min of unilateral leg press exercises at 65% of 1-RM, and ~15 minutes of cycle ergometer at 65% of VO$_{2\text{max}}$. In this intervention, upright seated SBP was ~20 mm
Hg lower than before exercise after 10-minute of recovery, and MAP was ~7 mm Hg lower from 30-min onwards. Additionally, Brown et al, 56, and MacDonald et al 55 observed that the magnitude of upright seated PEH after RE was similar as compared to PEH following aerobic exercise.

In summary, post-exercise hypotenstion was observed in healthy younger adults after an acute bout of RE performed at “repetition-limit” for exercise-intensity (e.g., as many repetitions as possible at 70% of 1-RM; 52 20-25 repetitions, and 8-10 repetitions at 40% and 70% of 1-RM, respectively; 56 and 15-minutes at 65% of 1-RM 55). At exception of Hill et al’s study, 52 reductions on arterial BP below pre-exercise levels were observed after 10-min of recovery, and remained low throughout 60-min of standing and upright seated recovery. However, it should be noted that these exercise-testing protocols allow different numbers of repetition for exercise and participant for exercise-intensity. Thus, it is difficult to identify the workload examined, and to specify the load eliciting post-exercise BP drop below pre-exercise level after RE.

In contrast to the studies referred above, O’Connor et al 53 and Koltyn et al 57 also using intermittent auscultatory BP method reported elevated BP response after RE. O’Connor et al 53 examined arterial BP response 1-min immediately after exercise (in a controlled setting), and for up 120-min ambulatory BP (under free-living condition) in healthy younger women (mean age= 22.6±3.9 yrs; n=14) following 3-sets of 10-reps on six different REs at 40%, 60%, and 80% of the 10-RM, performed during 3 separate days. In this intervention, elevated SBP and DBP were observed immediately after the exercise (1-minute after exercise) with a rapid return toward pre-exercise control levels during the remainder of the 2-hours post-exercise measurement period. In contrast to the
finding of Hill et al, these findings revealed an increase in SBP that occurred 1-min after exercise at 60% of 1-RM, and 1- and 15-min after exercise at 80% of 1-RM. However, differences in BP assessment, gender, exercise intensity, and duration are all factors that may explain the lack of the post-exercise BP drop below pre-exercise level in these investigations.

Similarly, Koltyn et al using also intermittent auscultatory BP method evaluated BP responses 5-min after a weight training session (50-min of self-selected weight lifting activities) comparing 25 college students (men=13; women=12; mean age=19±3 yrs) enrolled in a beginner’s weight training class to a control group (25 college students enrolled in a lecture class; men=10; women=15; mean age=18±1.5 yrs). In this intervention, an increase in SPB (pre=114.7±10.9; post=119.4±16.1) was observed in the students enrolled in the weight training class, while a decrease in SBP (pre=120.0±13.5; post=115.2±14.5) was observed in the lecture class students at that time-point of recovery (5-minutes after exercise). The weight training class students’ increase in SBP was 4.7 mmHg, which represents a 4.1% raise.

Additional studies examining post-exercise arterial BP under free-living conditions showed no differences in SBP, DBP, or MAP after a single bout of RE. As described previously, O’Connor et al observed that after initial elevations in SBP and DBP immediately following exercise, ambulatory BP return toward pre-exercise control levels during the remainder of the 2-hours post-exercise measurement period. However, pre-exercise BP measurements were recorded while the subjects assumed an upright-seated position, and post-exercise BP measurements with the subjects mostly in the upright standing position and walking (both standing position and walking have well-documented
effects on blood pressure). In addition, the average pre-exercise SBP reported for the group was relatively low (i.e., 110 mm Hg), which might have made post-exercise BP drop below pre-exercise level less likely. Recently, Roltsch et al.,\textsuperscript{54} using an intermittent auscultatory BP method, examined 24-h ambulatory BP in sedentary (men=6, women=6; mean age=20.3±1.8 yrs), resistance-trained (men=6, women=6; mean age=23.4±2.3 yrs), and endurance-trained (men=6, women=6; mean age=23.5±3.4 yrs) men and women (age range=18-26 yrs) after 2 sets of 8-RM (with upper body exercises) and 12-RM (with lower body exercises) on 12 weight machines, and in comparison to 48-hrs without exercise. BP recordings started 25-30 minutes after performing RE protocol, or following an equivalent time of non-exercise condition (control day). In this intervention, SBP, DBP, MAP, & HR were neither increased nor decreased in the hours immediately after and for up to 24 h after a acute session of RE bout compared with during a non-exercise control day in young, normotensive men and women. Neither were gender differences observed for any of the BP or HR parameters, nor in the ambulatory BP response after the single session of RE based on the training status of the subjects.

It should be noted that Roltsch et al.,\textsuperscript{54} in contrast to Hill et al (3 sets of 4 different REs at 70% of 1-RM, as many repetitions as possible),\textsuperscript{52} O’Connor et al (3 sets of six different REs at 40, 60, and 80% of 10-RM, and 10-repetitions held constant),\textsuperscript{53} and Brown et al (3 sets of 5 different REs at 40% and 70% of 1-RM, 20-25 repetitions and 8-10 repetitions, respectively),\textsuperscript{54} used an 8-RM for each upper-body exercise, and a 12-RM for each lower-body exercise to determine the weight to be used during the acute RE session, and provide the same number of RM for all exercises and for all subjects (a more individualized protocol). The 8-RM, and 12-RM tests represented the maximal resistance
that could be moved throughout the full range of motion for 8 and 12 repetitions for exercise, respectively.

Thus, it appears to be that post-exercise BP drop below pre-exercise level has not been conclusively established after an acute bout of RE in humans under controlled setting, or under free-living conditions outside of laboratory. Differences in exercise testing protocol (e.g., number of exercise, modality, exercise intensity and duration, and number of repetitions), gender, assessing post-exercise BP response, and body positions (standing, seated, or supine) during pre-exercise and post-exercise BP assessments have been suggested as potential reasons that might explain the discrepancies in the results.

Moreover, it is not surprising that even less is known about the influence of exercise condition on post-exercise BP drop below pre-exercise level. Only two studies (using both intermittent auscultatory BP methods, but different exercise testing protocol) in healthy younger adults have addressed this issue, and the observations of these studies are equivocal. Brown et al \(^{56}\) reported similar BP responses (no differences in magnitude and duration for all conditions) after RE at different exercise intensities (SBP fell below pre-exercise values after 10-min, DBP recovered to pre-exercise values after 15-min, and both thereafter remained so after 60-min of recovery). However, O’Connor et al \(^{53}\) reported differential BP responses (increased SBP) after RE at different exercise intensities (unchanged SBP across control and 40% of 10-RM exercise condition, but SBP changes were found across 60 and 80% of 10-RM exercise conditions).

To address these issues, two separate pilot studies from our laboratory using continuous BP monitoring indicate a difference in post-exercise BP drop below pre-exercise level following consecutive incremental dynamic RE protocols (e.g., 20, 30, 40,
50, 60, 70, 80, 90% of 5-RM; and 20, 40, 60 and 80% of 5-RM) in younger ($p=0.03$) and older adults ($p=0.0001$), within a 4-minute upright seated recovery-period, respectively. In the pilot-study with younger adults (mean age=26±3.2 yrs; n=16), post-exercise DBP were lower when compared to pre-exercise ($p=0.001$) at 2-min for all intensities, while post-exercise SBP was lower at all recovery periods (from 30-second to 3-min recovery period) compared to pre-exercise. Furthermore, there was an intensity x test period interaction ($p=0.03$) indicating that post-exercise SBPs were lower following higher exercise intensities. In the pilot study with older adults (mean age=60±8.5 yrs; n=8), post-exercise SBP was lower at 2.5 and 3 min compared to pre-exercise ($p=0.0001$) for all intensities, while post-exercise DBP was lower at all recovery periods (from 30-second to 3-min recovery period) compared to pre-exercise. Furthermore, there was an intensity x test period interaction ($p=0.0001$) indicating that post-exercise DBPs were lower following higher exercise intensities.

Even with the above results, it should be noted that Brown et al $^{56}$ used different number of repetitions for both exercise intensities (which might represent similar effort between both combinations of exercise intensity and repetition), as well different “repetition-limit” for exercise and participant at each exercise-intensity (which might represent different exercise load examined for participant). Moreover, O’Connor et al $^{53}$ used loads equivalent to relative intensities of 10-RM (which might represent a very low exercise intensity), the number of repetitions was held constant for all exercise intensities (which might indicate that the participants reached a limit effort only at higher exercise intensities), and post-exercise ambulatory BP (mostly in upright standing and walking condition) was compared to seated pre-exercise BP measurements. In our case, we did
not account for an order effect of the consecutive incremental protocols used with younger (12-reps of consecutive knee-extension RE at 20, 30, 40, 50, 60, 70, 80, and 90% of 5-RM, separate for 4-minute recovery-periods) and older adults (15-reps of consecutive knee-extension RE at 20, 40, 60 and 80% of 5-RM, separated for 4 minutes recovery-periods), so that the design of these studies only provide weak inferences with regard to the influence of exercise condition on post-exercise BP drop (due to the possibility of an order effect of the consecutive incremental protocol). In addition, both pilot study designs provide limited information about the duration of post-exercise BP drop below pre-exercise level after RE (hemodynamic responses observed throughout 4-minute recovery-period between loads, and after the last one), and the number of repetitions for exercise intensities was held constant, so that the participants reached a limit-effort only at higher intensities.

Lastly, it should be noted that to date no study that we could find has examined post-exercise hemodynamic response after RE in older adults, even though age-related changes within the cardiovascular system (e.g., increase in the stiffness of large arteries \textsuperscript{74-76}), and in neural and local cardiovascular control mechanisms (e.g., diminished autonomic tone, and/or impairment of vascular responsiveness to beta-adrenergic receptor stimulation \textsuperscript{75}) have been revealed. It is possible that those age-related changes might influence the post-exercise hemodynamic responses in this population. In fact, age-related differences in post-exercise BP drop below pre-exercise level after aerobic exercise have been observed. In addition, guidelines for exercise prescription for older adults \textsuperscript{65,73} recommend including RE as part of a well-rounded physical fitness program for this population. Thus, it is important to investigate the hemodynamic response to RE
in older adults as they might have implications for risk, and influence exercise prescription.

In summary, it is difficult to formulate hypotheses about the effects of exercise condition on the hemodynamic response after RE in young and older adults based on the existing information. Additional evidence is required to discern the role of exercise condition, as well the effect of age on the magnitude, rate and duration of post-exercise BP drop below pre-exercise level following RE. Therefore, using continuous BP monitoring, the main purpose of this investigation was to examine the acute hemodynamic response (SBP, DBP, MAP, PP, HR, RPP) in healthy young adult women, and 15 healthy older adult women before, during, immediately after, and for 1 hour following 5-RM, and 15-RM of bilateral knee-extension RE. In particular, this study design examined the effects of age (younger and older adults) and exercise intensity (5-RM, and 15-RM), as well the age x exercise intensity interaction on the magnitude (peak exercise BP, local minimum BP, magnitude of drop from peak to local minimum, local minimum minus pre-exercise, and 1-minute and 3-minute post-exercise BP ratios), rate (time from peak exercise BP to local minimum, and decay of BP drop), duration (60 min recovery BP minus pre-exercise), and recovery overshoot of post-exercise BP (the difference between local maximum and local minimum, the time from local minimum to local maximum, and the rate) accordingly with the parameters of interest characterizing each one of them.


Preliminary studies include the pilot studies conducted by the department of Kinesiology at LSU examining (a) the reliability and stability of tonometric blood pressure readings at
rest, during, and following incremental bilateral knee-extension RE, and (b) the post-exercise acute hemodynamic responses to consecutive incremental bilateral knee-extension RE in older adults.

**Reliability Study:** Over recent months, we have integrated continuous non-invasive BP monitoring in our non-invasive cardiovascular laboratory here at LSU. Among the protocols employed has been the examination of continuous arterial BP at rest, and during and after dynamic incremental bilateral knee-extension RE. The purpose of this early pilot study was to establish the test-retest reliability of the tonometric BP readings under different conditions.

The validity of tonometric BP readings in comparison to intra-arterial measures of arterial BP is documented for various investigations (Intraclass R’s in the range of 0.81-0.91). In addition, tonometric BP readings are considered to be reliable at rest. However, the reliability of the tonometric BP readings during and after dynamic incremental bilateral knee-extension RE has not been reported. Pilot data from our own lab for the 2-week test-retest reliability of the BP responses as measured by the Colin 7000 prior to (at rest), during and after dynamic incremental bilateral knee-extension RE in 16 adults of all ages are reported in Tables 1 through 6.

The incremental test protocol required the participants to perform knee-extensions at consecutive workloads equivalent to 20, 30, 40, 50, 60, 70, 80, and 90% of the 5-RM. Each set was performed to 12 repetitions or to fatigue, which ever occurred first. The first work bout was preceded by a minimum of 10 minutes of quiet resting, each repetition was performed in 4 seconds (2-seconds for concentric phase and 2-seconds for eccentric phase), and each work bout was followed by a 4-minute recovery period. During
exercise, the participants were instructed to breath out lifting the weight, and breath in lowering the weight. While seated on the Med-X knee-extension device, the participant’s left wrist was fitted with the Colin 7000 tonometric sensor (Colin Medical Instruments, San Antonio, TX). The participants were instructed to maintain a static position in the measured arm during the rest period and during each incremental work bout. This was facilitated by the use of an armrest plus straps that stabilized the arm at heart level. Continuous pressure data were collected throughout the test. Sixteen of twenty-two participants completed three tests in three consecutive weeks.

Resting data from this pilot-study support previous observation of the reliability of the tonometric BP readings at rest. Average values at rest and 1-min immediately prior to each exercise-intensity in all three combined trials are presented in the Table A.1.

Table A.1 Blood Pressures at Rest and Immediately Prior to Exercise.

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>IP20%</th>
<th>IP30%</th>
<th>IP40%</th>
<th>IP50%</th>
<th>IP60%</th>
<th>IP70%</th>
<th>IP80%</th>
<th>IP90%</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP</td>
<td>120.7</td>
<td>126.1</td>
<td>127.9</td>
<td>128.3</td>
<td>128.8</td>
<td>129.0</td>
<td>129.4</td>
<td>129.1</td>
<td>128.5</td>
</tr>
<tr>
<td></td>
<td>±8.0</td>
<td>±11.7</td>
<td>±12.3</td>
<td>±11.4</td>
<td>±11.4</td>
<td>±10.2</td>
<td>±10.4</td>
<td>±10.0</td>
<td>±11.6</td>
</tr>
<tr>
<td>DBP</td>
<td>65.7</td>
<td>66.7</td>
<td>65.9</td>
<td>65.9</td>
<td>66.1</td>
<td>64.7</td>
<td>64.7</td>
<td>62.3</td>
<td>65.2</td>
</tr>
<tr>
<td></td>
<td>±7.9</td>
<td>±10.2</td>
<td>±8.1</td>
<td>±8.1</td>
<td>±10.1</td>
<td>±8.9</td>
<td>±8.6</td>
<td>±7.4</td>
<td>±9.6</td>
</tr>
<tr>
<td>MAP</td>
<td>84.1</td>
<td>86.5</td>
<td>86.6</td>
<td>86.7</td>
<td>87.0</td>
<td>86.1</td>
<td>86.3</td>
<td>84.4</td>
<td>84.0</td>
</tr>
<tr>
<td></td>
<td>±7.4</td>
<td>±10.1</td>
<td>±8.2</td>
<td>±8.2</td>
<td>±9.9</td>
<td>±8.5</td>
<td>±8.5</td>
<td>±7.6</td>
<td>±9.6</td>
</tr>
</tbody>
</table>

Values are in mmHg, and represent the mean and standard deviation (mean ±SD) of peak-values for intensity of the 16 participants across all three trials. SBP = systolic blood pressure. DBP = diastolic blood pressure. MAP = mean arterial blood pressure

Table A.2 presents the reliability indicators of tonometric BP reading (coefficient of variation, and intraclass correlation coefficients) for SBP, DBP, and MAP during rest, and 1-min immediately prior to each exercise-intensity. The coefficients of variation...
(CV) ranged between 6.1 to 10.0% and the intraclass correlation coefficients (ICC) ranged from 0.69 to 0.86 (except data after 50% and 90% of 5-RM).

Table A.2  Reliability Indicators For Tonometric Blood Pressure Readings at Rest and Immediately Prior to Exercise

<table>
<thead>
<tr>
<th>Rest, &amp; IPE</th>
<th>Systolic peak Pressure</th>
<th>Diastolic peak Pressure</th>
<th>Mean Arterial Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CV</td>
<td>ICC</td>
<td>CV</td>
</tr>
<tr>
<td>Rest</td>
<td>6.1</td>
<td>0.78</td>
<td>10.8</td>
</tr>
<tr>
<td>IP20%</td>
<td>8.6</td>
<td>0.70</td>
<td>13.1</td>
</tr>
<tr>
<td>IP30%</td>
<td>9.1</td>
<td>0.75</td>
<td>11.7</td>
</tr>
<tr>
<td>IP40%</td>
<td>8.3</td>
<td>0.77</td>
<td>11.4</td>
</tr>
<tr>
<td>IP50%</td>
<td>8.8</td>
<td>0.57</td>
<td>13.8</td>
</tr>
<tr>
<td>IP60%</td>
<td>7.7</td>
<td>0.61</td>
<td>12.6</td>
</tr>
<tr>
<td>IP70%</td>
<td>7.8</td>
<td>0.69</td>
<td>12.1</td>
</tr>
<tr>
<td>IP80%</td>
<td>7.7</td>
<td>0.73</td>
<td>9.5</td>
</tr>
<tr>
<td>IP90%</td>
<td>9.1</td>
<td>0.59</td>
<td>14.0</td>
</tr>
</tbody>
</table>

CV = coefficient of variation as a %. ICC = Intra-class correlation coefficient. IPE = immediately prior to exercise.

Table A.3 shows the average peak values (SBP, DBP, MAP and HR) for the three trials during exercise. Figure A.1 shows the behavior of each dependent variable (SBP, DBP, MAP) during each of the three trials. The pressures are very close to those reported by Bermon, although somewhat low in comparison to the values reported by McCartney, & MacDougal.
Table A.3  Blood Pressure and Heart Rate Responses to Incremental Work by Trial
Workload (% of 5RM)

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>20%</th>
<th>30%</th>
<th>40%</th>
<th>50%</th>
<th>60%</th>
<th>70%</th>
<th>80%</th>
<th>90%</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP</td>
<td>120.7</td>
<td>153.5</td>
<td>162.9</td>
<td>169.6</td>
<td>171.4</td>
<td>179.2</td>
<td>188.1</td>
<td>190.6</td>
<td>190.2</td>
</tr>
<tr>
<td></td>
<td>±6.1</td>
<td>±14.3</td>
<td>±18.6</td>
<td>±18.4</td>
<td>±20.9</td>
<td>±19.6</td>
<td>±24.3</td>
<td>±18.8</td>
<td>±21.3</td>
</tr>
<tr>
<td>DBP</td>
<td>65.8</td>
<td>90.2</td>
<td>97.9</td>
<td>101.3</td>
<td>103.4</td>
<td>110.2</td>
<td>121.2</td>
<td>123.3</td>
<td>120.4</td>
</tr>
<tr>
<td></td>
<td>±7.6</td>
<td>±11.8</td>
<td>±10.6</td>
<td>±10.6</td>
<td>±12.9</td>
<td>±12.3</td>
<td>±18.1</td>
<td>±13.4</td>
<td>±16.7</td>
</tr>
<tr>
<td>MAP</td>
<td>84.1</td>
<td>111.3</td>
<td>119.6</td>
<td>124.1</td>
<td>126.1</td>
<td>133.2</td>
<td>143.5</td>
<td>145.7</td>
<td>135.5</td>
</tr>
<tr>
<td></td>
<td>±7.7</td>
<td>±12.0</td>
<td>±12.2</td>
<td>±12.3</td>
<td>±14.7</td>
<td>±13.2</td>
<td>±19.4</td>
<td>±14.7</td>
<td>±18.2</td>
</tr>
<tr>
<td>HR</td>
<td>75.9</td>
<td>102.3</td>
<td>109.0</td>
<td>116.2</td>
<td>122.7</td>
<td>130.0</td>
<td>138.8</td>
<td>144.0</td>
<td>145.1</td>
</tr>
<tr>
<td></td>
<td>±5.6</td>
<td>±5.3</td>
<td>±4.9</td>
<td>±5.6</td>
<td>±6.7</td>
<td>±6.9</td>
<td>±6.3</td>
<td>±7.2</td>
<td>±10.8</td>
</tr>
</tbody>
</table>

Values are in mmHg, and represent the mean and standard deviation (mean ± SD) of peak-values for intensity of the 16 participants across all three trials. SBP = systolic blood pressure. DBP = diastolic blood pressure. MAP = mean arterial blood pressure. HR = heart rate.

Figure A.1  Blood Pressure Response to Incremental Work-load Trials
Values are in mm Hg, and represent mean ± SD. SBP = systolic blood pressure. DBP = diastolic blood pressure. MAP = mean arterial blood pressure.
Table A.4 presents the coefficients of variation (CVs), and intraclass correlation coefficients (ICCs) for SBP, DBP, MAP and HR for the three trials during exercise. The ranges of ICCs and CVs are similar to ICCs and CVs for resting BP previously reported.

### Table A.4 Reliability Indicators for Tonometric Blood Pressure Readings During Exercise

<table>
<thead>
<tr>
<th>Intensity (% 5-RM)</th>
<th>Systolic peak Pressure</th>
<th>Diastolic peak Pressure</th>
<th>Mean Arterial Pressure</th>
<th>Heart Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CV</td>
<td>ICC</td>
<td>CV</td>
<td>ICC</td>
</tr>
<tr>
<td>Resting</td>
<td>8.6</td>
<td>0.64</td>
<td>10.2</td>
<td>0.59</td>
</tr>
<tr>
<td>20</td>
<td>8.2</td>
<td>0.75</td>
<td>11.8</td>
<td>0.74</td>
</tr>
<tr>
<td>30</td>
<td>9.9</td>
<td>0.55</td>
<td>9.3</td>
<td>0.67</td>
</tr>
<tr>
<td>40</td>
<td>9.5</td>
<td>0.73</td>
<td>9.3</td>
<td>0.75</td>
</tr>
<tr>
<td>50</td>
<td>10.4</td>
<td>0.58</td>
<td>11.4</td>
<td>0.62</td>
</tr>
<tr>
<td>60</td>
<td>9.9</td>
<td>0.72</td>
<td>9.9</td>
<td>0.60</td>
</tr>
<tr>
<td>70</td>
<td>11.4</td>
<td>0.77</td>
<td>12.4</td>
<td>0.67</td>
</tr>
<tr>
<td>80</td>
<td>8.5</td>
<td>0.87</td>
<td>9.5</td>
<td>0.76</td>
</tr>
<tr>
<td>90</td>
<td>10.2</td>
<td>0.76</td>
<td>12.3</td>
<td>0.55</td>
</tr>
</tbody>
</table>

CV = coefficient of variation as a %

ICC = intraclass correlation coefficient

The magnitudes of post-exercise SBP and DBP (difference between post-exercise BP value, at a specific time-point, and pre-exercise arterial BP value) for six consecutive 30-second recovery periods (R30, R60, R90, R120, R150, and R180) are presented in Table A.5 and A.6, respectively. The negative values indicate that the magnitude of post-exercise SBP and DBP were lower than pre-exercise values at each time point on the test protocol.
Table A.5  Magnitude of Post-Exercise SBP Drop Following Incremental Workload  
(30-sec Recovery Periods)

<table>
<thead>
<tr>
<th></th>
<th>IPE</th>
<th>R30</th>
<th>R60</th>
<th>R90</th>
<th>R120</th>
<th>R150</th>
<th>R180</th>
</tr>
</thead>
<tbody>
<tr>
<td>20%</td>
<td>126.3 ±10.8</td>
<td>-6.2 ±7.2</td>
<td>-7.0 ±8.0</td>
<td>-7.9 ±9.4</td>
<td>-8.7 ±9.9</td>
<td>-10.5 ±6.5</td>
<td>-10.4 ±10.0</td>
</tr>
<tr>
<td>30%</td>
<td>124.0 ±8.3</td>
<td>-8.0 ±9.2</td>
<td>-3.8 ±8.1</td>
<td>-8.2 ±9.2</td>
<td>-10.0 ±8.9</td>
<td>-9.7 ±10.4</td>
<td>-8.9 ±7.2</td>
</tr>
<tr>
<td>40%</td>
<td>124.0 ±13.7</td>
<td>-5.3 ±6.3</td>
<td>-4.0 ±4.9</td>
<td>-6.4 ±8.0</td>
<td>-5.9 ±9.1</td>
<td>-8.6 ±9.0</td>
<td>-6.5 ±9.4</td>
</tr>
<tr>
<td>50%</td>
<td>127.3 +9.5</td>
<td>-12.9 ±7.1</td>
<td>-11.2 ±8.1</td>
<td>-11.2 ±8.2</td>
<td>-12.3 ±9.6</td>
<td>-15.9 ±11.6</td>
<td>-15.0 ±11.4</td>
</tr>
<tr>
<td>60%</td>
<td>125.2 ±10.1</td>
<td>-7.6 ±10.2</td>
<td>-9.3 ±10.1</td>
<td>-11.3 ±7.8</td>
<td>-12.7 ±10.9</td>
<td>-12.6 ±10.3</td>
<td>-12.8 ±8.5</td>
</tr>
<tr>
<td>70%</td>
<td>127.7 ±8.5</td>
<td>-9.9 ±9.5</td>
<td>-10.6 ±7.4</td>
<td>-11.9 ±5.4</td>
<td>-12.2 ±5.0</td>
<td>-14.9 ±6.9</td>
<td>-15.4 ±8.5</td>
</tr>
<tr>
<td>80%</td>
<td>128.3 ±10.4</td>
<td>-13.1 ±9.8</td>
<td>-10.5 ±5.69</td>
<td>-13.1 ±8.7</td>
<td>-16.0 ±8.5</td>
<td>-13.5 ±7.3</td>
<td>-14.3 ±6.1</td>
</tr>
</tbody>
</table>

Values are in mmHg, and represent the mean and standard deviation (mean ± SD) of the magnitude of post-exercise SBP of 10 participants across all three trials. 
SBP = systolic blood pressure

Table A.6  Magnitude of Post-Exercise DBP Drop Following Incremental Workload  
(30-second Recovery Period)

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>R30</th>
<th>R60</th>
<th>R90</th>
<th>R120</th>
<th>R150</th>
<th>R180</th>
</tr>
</thead>
<tbody>
<tr>
<td>20%</td>
<td>70.2 ±8.1</td>
<td>-9.2 ±4.6</td>
<td>-7.1 ±6.5</td>
<td>-7.3 ±6.5</td>
<td>-5.7 ±7.2</td>
<td>-5.5 ±7.7</td>
<td>-6.5 ±6.1</td>
</tr>
<tr>
<td>30%</td>
<td>68.5 ±6.6</td>
<td>-11.8 ±3.8</td>
<td>-9.43 ±4.0</td>
<td>-9.8 ±4.5</td>
<td>-9.1 ±5.2</td>
<td>-11.5 ±7.8</td>
<td>-9.3 ±6.6</td>
</tr>
<tr>
<td>40%</td>
<td>70.6 ±6.3</td>
<td>-13.3 ±7.3</td>
<td>-9.5 ±5.1</td>
<td>-8.2 ±5.2</td>
<td>-8.9 ±5.8</td>
<td>-9.8 ±6.0</td>
<td>-8.7 ±5.8</td>
</tr>
<tr>
<td>50%</td>
<td>69.6 ±8.7</td>
<td>-14.0 ±7.3</td>
<td>-9.8 ±5.1</td>
<td>-9.9 ±5.2</td>
<td>-9.7 ±5.8</td>
<td>-10.8 ±6.0</td>
<td>-8.8 ±5.8</td>
</tr>
<tr>
<td>60%</td>
<td>67.4 ±6.7</td>
<td>-12.9 ±8.3</td>
<td>-9.5 ±9.4</td>
<td>-9.5 ±8.1</td>
<td>-9.1 ±8.3</td>
<td>-9.9 ±8.9</td>
<td>-9.8 ±9.8</td>
</tr>
<tr>
<td>70%</td>
<td>68.7 ±7.2</td>
<td>-12.3 ±8.1</td>
<td>-10.3 ±6.8</td>
<td>-9.9 ±8.8</td>
<td>-10.0 ±9.3</td>
<td>-10.0 ±7.3</td>
<td>-9.1 ±7.3</td>
</tr>
<tr>
<td>80%</td>
<td>71.1 ±6.3</td>
<td>-7.8 ±10.6</td>
<td>-9.2 ±6.8</td>
<td>-7.7 ±6.7</td>
<td>-9.1 ±8.0</td>
<td>-9.4 ±9.2</td>
<td>-7.5 ±6.4</td>
</tr>
</tbody>
</table>

Values are in mm Hg, and represent the mean and standard deviation (mean ± SD) of the magnitude of post-exercise DBP of 10 participants across all three trials. 
DBP = diastolic blood pressure
Accordingly, Tables A.7 and A.8 show the ICCs and CVs for SBP and DBP (peak and minimum BP values) respectively, for all three trials following exercise (12-repetitions of knee-extension RE at consecutive incremental exercise-intensity) during the six consecutive 30-second recovery-periods (R\textsubscript{30}, R\textsubscript{60}, R\textsubscript{90}, R\textsubscript{120}, R\textsubscript{150}, and R\textsubscript{180}) observed. The ranges of ICCs and CVs are similar those ICCs and CVs for resting BP previously reported.

### Table A.7 Reliability Indicators for Tonometric Systolic BP Readings Immediately Prior to and Following Resistance Exercise

<table>
<thead>
<tr>
<th>Reliability Ind. by workload</th>
<th>Recovery Periods</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
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<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IPE</td>
<td>R\textsubscript{30}</td>
<td>R\textsubscript{60}</td>
<td>R\textsubscript{90}</td>
<td>R\textsubscript{120}</td>
<td>R\textsubscript{150}</td>
<td>R\textsubscript{180}</td>
<td>Average</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20% CV</td>
<td>8.6</td>
<td>12.8</td>
<td>11.0</td>
<td>13.3</td>
<td>11.2</td>
<td>10.6</td>
<td>12.3</td>
<td>11.4</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICC</td>
<td>0.78</td>
<td>0.67</td>
<td>0.64</td>
<td>0.71</td>
<td>0.79</td>
<td>0.81</td>
<td>0.80</td>
<td>0.74</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30% CV</td>
<td>8.14</td>
<td>13.84</td>
<td>11.48</td>
<td>9.84</td>
<td>10.31</td>
<td>13.26</td>
<td>13.06</td>
<td>11.42</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICC</td>
<td>0.89</td>
<td>0.84</td>
<td>0.79</td>
<td>0.68</td>
<td>0.48</td>
<td>0.57</td>
<td>0.60</td>
<td>0.69</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>ICC</td>
<td>0.74</td>
<td>0.73</td>
<td>0.82</td>
<td>0.79</td>
<td>0.74</td>
<td>0.63</td>
<td>0.75</td>
<td>0.74</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>50% CV</td>
<td>6.30</td>
<td>8.20</td>
<td>10.80</td>
<td>10.12</td>
<td>10.00</td>
<td>7.93</td>
<td>9.27</td>
<td>8.95</td>
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</tr>
<tr>
<td>ICC</td>
<td>0.92</td>
<td>0.79</td>
<td>0.70</td>
<td>0.76</td>
<td>0.83</td>
<td>0.82</td>
<td>0.52</td>
<td>0.76</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>60% CV</td>
<td>8.35</td>
<td>8.51</td>
<td>9.92</td>
<td>8.75</td>
<td>11.28</td>
<td>8.30</td>
<td>8.49</td>
<td>9.09</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>ICC</td>
<td>0.69</td>
<td>0.76</td>
<td>0.72</td>
<td>0.80</td>
<td>0.78</td>
<td>0.80</td>
<td>0.79</td>
<td>0.76</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>70% CV</td>
<td>5.50</td>
<td>9.23</td>
<td>9.42</td>
<td>7.94</td>
<td>9.71</td>
<td>10.42</td>
<td>8.98</td>
<td>8.74</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>ICC</td>
<td>0.78</td>
<td>0.74</td>
<td>0.85</td>
<td>0.82</td>
<td>0.84</td>
<td>0.68</td>
<td>0.72</td>
<td>0.78</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>80% CV</td>
<td>7.04</td>
<td>12.54</td>
<td>7.60</td>
<td>8.10</td>
<td>7.50</td>
<td>6.61</td>
<td>7.50</td>
<td>8.13</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>ICC</td>
<td>0.79</td>
<td>0.79</td>
<td>0.85</td>
<td>0.86</td>
<td>0.80</td>
<td>0.92</td>
<td>0.83</td>
<td>0.83</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td>7.73</td>
<td>11.06</td>
<td>10.25</td>
<td>9.97</td>
<td>9.90</td>
<td>9.91</td>
<td>10.47</td>
<td>9.90</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CV = coefficient of variation as a %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICC = intraclass correlation coefficient</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R\textsubscript{30} = recovery period in seconds</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>
The data reveal an influence of test stage (e.g., intensity) on the acute hemodynamic response following the exercise. There was also a main effect of test period (i.e. pre exercise vs. post exercise) on DBP ($p=0.0001$), while there was a significant intensity x test period interaction ($p=0.03$) such that recovery SBPs were lower following higher intensities. Thus, the following experiment (second project) was performed to examine whether the magnitude of post-exercise BP drop below pre-exercise level following a consecutive incremental protocol was also present in older adults.

---

<table>
<thead>
<tr>
<th>Reliability Ind. by workload</th>
<th>Recovery Periods</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>$R_{30}$</td>
<td>$R_{60}$</td>
<td>$R_{90}$</td>
<td>$R_{120}$</td>
<td>$R_{150}$</td>
<td>$R_{180}$</td>
</tr>
<tr>
<td>20% ICC</td>
<td>0.79</td>
<td>0.82</td>
<td>0.73</td>
<td>0.70</td>
<td>0.79</td>
<td>0.74</td>
<td>0.76</td>
</tr>
<tr>
<td>30% CV</td>
<td>9.81</td>
<td>13.39</td>
<td>14.50</td>
<td>12.02</td>
<td>14.89</td>
<td>16.60</td>
<td>15.36</td>
</tr>
<tr>
<td>30% ICC</td>
<td>0.81</td>
<td>0.80</td>
<td>0.80</td>
<td>0.82</td>
<td>0.64</td>
<td>0.68</td>
<td>0.70</td>
</tr>
<tr>
<td>40% ICC</td>
<td>0.84</td>
<td>0.73</td>
<td>0.81</td>
<td>0.77</td>
<td>0.85</td>
<td>0.76</td>
<td>0.80</td>
</tr>
<tr>
<td>50% CV</td>
<td>9.86</td>
<td>12.33</td>
<td>15.02</td>
<td>15.32</td>
<td>15.91</td>
<td>16.93</td>
<td>14.96</td>
</tr>
<tr>
<td>50% ICC</td>
<td>0.89</td>
<td>0.82</td>
<td>0.76</td>
<td>0.80</td>
<td>0.63</td>
<td>0.57</td>
<td>0.60</td>
</tr>
<tr>
<td>60% CV</td>
<td>10.14</td>
<td>16.60</td>
<td>15.64</td>
<td>14.21</td>
<td>11.47</td>
<td>12.82</td>
<td>14.43</td>
</tr>
<tr>
<td>60% ICC</td>
<td>0.62</td>
<td>0.80</td>
<td>0.78</td>
<td>0.80</td>
<td>0.68</td>
<td>0.76</td>
<td>0.69</td>
</tr>
<tr>
<td>70% CV</td>
<td>10.14</td>
<td>14.06</td>
<td>14.93</td>
<td>14.81</td>
<td>15.46</td>
<td>14.50</td>
<td>12.48</td>
</tr>
<tr>
<td>70% ICC</td>
<td>0.61</td>
<td>0.72</td>
<td>0.68</td>
<td>0.63</td>
<td>0.61</td>
<td>0.65</td>
<td>0.76</td>
</tr>
<tr>
<td>80% CV</td>
<td>7.34</td>
<td>14.59</td>
<td>12.79</td>
<td>10.12</td>
<td>9.87</td>
<td>9.78</td>
<td>13.25</td>
</tr>
<tr>
<td>80% ICC</td>
<td>0.80</td>
<td>0.82</td>
<td>0.86</td>
<td>0.89</td>
<td>0.75</td>
<td>0.81</td>
<td>0.63</td>
</tr>
<tr>
<td>Average ICC</td>
<td>0.77</td>
<td>0.79</td>
<td>0.77</td>
<td>0.77</td>
<td>0.70</td>
<td>0.72</td>
<td>0.70</td>
</tr>
</tbody>
</table>

CV = coefficient of variation as a %
ICC = intraclass correlation coefficient
$R_{30}$ = recovery period in seconds
Blood Pressure Responses During and Following RE in Older Adults: The second experiment consisted of a cross-sectional pilot study to ascertain whether older adults demonstrated the same basic changes in arterial BP during and following the activity. Therefore, we examined the influence of consecutive incremental knee-extension RE on the magnitude of post-exercise hemodynamic responses (SBP, DBP, MAP, HR, and RPP) in eight older adults (mean age=60±8.5 yrs) (Figure A.2, & A.3).

Figure A.2  Systolic Blood Pressure Recovery from Incremental Resistance Exercise in Older Adults.

The incremental test protocol required participants to perform 15 repetitions of bilateral knee-extension RE at consecutive workloads equivalent to 20%, 40%, 60%, and 80% of 5- RM, separated by 4-minutes rest periods between each workload. Similar to the first project, the first workload was preceded by a minimum of 10 minutes of quiet resting. Each repetition was performed in 4 seconds (2-seconds for concentric phase, and 2-seconds for eccentric phase). During exercise, the participants were instructed to breathe out lifting the weight, and breath in lowering the weight. Protocol was the same
as before. Continuous BP and ECG data were acquired throughout the test, and measurements of dependent variables were recorded during rest, peak exercise, and the peak minimum for each 30-second recovery period (Figure A.2, & A.3).

Data indicated that the older adults demonstrated similar changes in arterial BP during and following the activity to the young adult participants. The results indicated a clear influence of test stage (i.e., intensity) on the hemodynamic response following the exercise ($p=0.0001$). In particular, there was a main effect of test period (i.e. pre exercise vs. post exercise) on SBP ($p=0.0001$), and a significant intensity x test period interaction ($p=0.001$) such that recovery DBPs were lower following higher exercise intensities (see Figure A.2 and A.3).

![Figure A.3](image)

**Figure A.3** Diastolic Blood Pressure Recovery from Incremental Resistance Exercise in Older Adults.

However, we did not account for an order effect of the protocol used (15 repetitions of consecutive knee-extension RE at 20, 40, 60 and 80% of 5-RM, separated for 4 min-
recovery periods). Thus, the design of our pilot study only provides weak inferences with regard to the influence of exercise condition on post-exercise BP drop below pre-exercise level. Accordingly, the purpose of this follow-up experiment will be to examine the effect of different RE conditions on the magnitude, rate, and duration of the post-exercise BP drop below pre-exercise level, using continuous BP monitoring in a healthy younger and older adults during a recovery period of 60-minutes.

A.3. Clinical relevance and Contribution

The sustained reduction of arterial blood pressure after a single bout of exercise, at sufficient magnitude and duration, might be a significant non-pharmacological treatment for some types of coronary artery disease (e.g., hypertension). Therefore, post-exercise BP drop below pre-exercise level may be related to risk following acute exercise (e.g., syncope and cardiovascular events have been reported with more frequency after the cessation of exercise), and delayed recovery has been implicated as a risk factor for the development of cardiovascular disease and hypertension. In addition, it has been shown that people who are physically fit show the same reactivity to similar relative physical and psychological stressor as those who are not fit, but recovery from the stressor more rapidly

139
APPENDIX B. EXTENDED RESULTS SECTION

B.1. Participant Characteristics

Of the 40 respondents, 32 passed screening and elected to continue with the study. Of the 8 respondents who did not participate in the study, 2 young women withdrew before providing informed consent, and 6 older women did not meet the inclusion criteria. The latter either had signs or symptoms consistent with high risk for adverse responses to exercise according to the ACSM guidelines,\(^\text{65}\) such as orthopedic or neurological disease (n = 2), high blood pressure (n = 2), or were on medication that could affect the results of this study (n = 2). Therefore, the following data are from 32 apparently healthy White participants, 16 young women (mean age = 21 ± 2.3 yrs) and 16 older women (mean age = 69 ± 2.3 yrs). Descriptive statistics for the participant’s characteristics, pre-exercise autonomic indicators from 5-, 10-minute segments, resting vascular function indices, and pre-exercise vascular function indices are reported from Table 3.1 to 3.5, respectively.

The results of t-test comparisons revealed age-group differences in (1) participant’s characteristics: weight (p = 0.04), waist-hip ratio (p = 0.02), estimated VO\(_2\) max (p = 0.0001), 5-RM (p = 0.0001), and 15-RM (p = 0.002); (2) pre-exercise autonomic indicators: R-R interval (p = 0.004/0.0001), SDNN (p = 0.0001/0.0001); and pnn50 (p = 0.0001/ 0.0001); (3) resting vascular function indices: SBPrest (p = 0.0001), MAPrest (p = 0.0001) FVRrest (p = 0.05), SBPocc (p = 0.05), DBPocc (p = 0.05), MAPocc (p = 0.05), and FVRocc (p = 0.006); and (4) pre-exercise vascular function indices: HR (p = 0.001), SBP (p = 0.0001), and MAP (p = 0.01) (Table 3.1-3.5, respectively).
Table B.1  Participant’s Characteristics

<table>
<thead>
<tr>
<th>Age</th>
<th>Young</th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>Age (year)</td>
<td>21.7 ± 1.6</td>
<td>69.8 ± 3.9 *</td>
</tr>
<tr>
<td>Height (inch)</td>
<td>63.4 ± 2.2</td>
<td>63.9 ± 2.9</td>
</tr>
<tr>
<td>Weight (lbs)</td>
<td>127.6 ± 19.8</td>
<td>143.7 ± 22.9 *</td>
</tr>
<tr>
<td>Waist/Hip ratio</td>
<td>0.74 ± 0.03</td>
<td>0.79 ± 0.06 *</td>
</tr>
<tr>
<td>Est. VO₂ (ml/kg/min)</td>
<td>39.8 ± 9.0</td>
<td>25.9 ± 3.9 *</td>
</tr>
<tr>
<td>5-RM (ft-lbs)</td>
<td>200.5 ± 40.8</td>
<td>128.9 ± 39.6 *</td>
</tr>
<tr>
<td>15-RM (ft-lbs)</td>
<td>119.4 ± 29.4</td>
<td>92.4 ± 13.3 *</td>
</tr>
<tr>
<td>Right Leg circumference (cm)</td>
<td>57.0 ± 5.1</td>
<td>56.4 ± 5.0</td>
</tr>
<tr>
<td>Left Leg circumference (cm)</td>
<td>56.6 ± 4.7</td>
<td>55.8 ± 5.0</td>
</tr>
</tbody>
</table>

Values are mean ± SD. 5-RM = 5 repetition maximum. 15-RM = 15 repetition maximum. * = Diff. from young group (p ≤ 0.05). + = trend for age-group differences (p = 0.11).

Table B.2  Pre-exercise Autonomic Indicators From 5-minute Segments

<table>
<thead>
<tr>
<th>Age group</th>
<th>R-R Interval</th>
<th>SDNN</th>
<th>Pnn50</th>
<th>LFnu</th>
<th>LF/HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5-RM</td>
<td>855.8 ± 130.5</td>
<td>55.9 ± 17.3</td>
<td>19.3 ± 15.7</td>
<td>0.61 ± 0.15</td>
<td>2.11 ± 1.64</td>
</tr>
<tr>
<td>15-RM</td>
<td>873.3 ± 139.9</td>
<td>64.1 ± 19.0</td>
<td>24.8 ± 19.1</td>
<td>0.60 ± 0.15</td>
<td>1.92 ± 1.35</td>
</tr>
<tr>
<td>Average</td>
<td>864.6 ± 133.3</td>
<td>59.9 ± 18.3</td>
<td>22.1 ± 17.5</td>
<td>0.60 ± 0.15</td>
<td>2.01 ± 1.48</td>
</tr>
<tr>
<td>Older</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5-RM</td>
<td>960.8 ± 127.6*</td>
<td>32.6 ± 12.2*</td>
<td>4.9 ± 9.1 *</td>
<td>0.58 ± 0.17</td>
<td>1.93 ± 1.56</td>
</tr>
<tr>
<td>15-RM</td>
<td>960.8 ± 122.5*</td>
<td>31.6 ± 13.0*</td>
<td>4.8 ± 9.2 *</td>
<td>0.58 ± 0.18</td>
<td>1.87 ± 1.38</td>
</tr>
<tr>
<td>Average</td>
<td>963.8 ± 127.6*</td>
<td>32.6 ± 12.2*</td>
<td>4.9 ± 9.1 *</td>
<td>0.58 ± 0.17</td>
<td>1.93 ± 1.56</td>
</tr>
</tbody>
</table>

Values are mean ± SD. * = Different from young group (p ≤ 0.05).

Table B.3  Pre-exercise Autonomic Indicators From 10-minute Segments

<table>
<thead>
<tr>
<th>Age group</th>
<th>R-R Interval</th>
<th>SDNN</th>
<th>Pnn50</th>
<th>LFnu</th>
<th>LF/HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>05-RM</td>
<td>846.9 ± 132.5</td>
<td>66.8 ± 20.1</td>
<td>18.9 ±15.2</td>
<td>0.65 ± 0.13</td>
<td>2.40 ± 1.70</td>
</tr>
<tr>
<td>15-RM</td>
<td>849.7 ± 126.7</td>
<td>71.8 ± 22.8</td>
<td>21.0 ±15.6</td>
<td>0.63 ± 0.14</td>
<td>2.16 ± 1.42</td>
</tr>
<tr>
<td>Average</td>
<td>848.3 ± 127.5</td>
<td>69.2 ± 21.3</td>
<td>22.1 ±17.5</td>
<td>0.64 ± 0.13</td>
<td>2.29 ± 1.55</td>
</tr>
<tr>
<td>Older</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>05-RM</td>
<td>960.8 ± 130.7*</td>
<td>46.4 ± 26.4*</td>
<td>8.2 ± 15.0 *</td>
<td>0.61 ± 0.16</td>
<td>2.19 ± 1.82</td>
</tr>
<tr>
<td>15-RM</td>
<td>954.7 ± 120.7*</td>
<td>43.2 ± 14.7*</td>
<td>5.2 ± 9.0  *</td>
<td>0.60 ± 0.18</td>
<td>2.20 ± 1.69</td>
</tr>
<tr>
<td>Average</td>
<td>957.8 ± 123.9*</td>
<td>45.0 ± 21.7*</td>
<td>6.8 ± 12.6 *</td>
<td>0.61 ± 0.17</td>
<td>2.19 ± 1.73</td>
</tr>
</tbody>
</table>

Values are mean ± SD. * = Different from young group (p ≤ 0.05).
Table B.4 Resting and After Occlusion HR, BP, & Vascular Function Indices

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Young</th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Resting</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR&lt;sub&gt;rest&lt;/sub&gt; (beat/min)</td>
<td>62.1 ± 13.4</td>
<td>59.9 ± 5.2</td>
</tr>
<tr>
<td>SBP&lt;sub&gt;rest&lt;/sub&gt; (mm Hg)</td>
<td>101.1 ± 5.7</td>
<td>124.3 ± 13.4 *</td>
</tr>
<tr>
<td>DBP&lt;sub&gt;rest&lt;/sub&gt; (mm Hg)</td>
<td>60.4 ± 6.7</td>
<td>64.4 ± 7.1 +</td>
</tr>
<tr>
<td>MAP&lt;sub&gt;rest&lt;/sub&gt; (mm Hg)</td>
<td>74.0 ± 6.0</td>
<td>84.3 ± 7.7 *</td>
</tr>
<tr>
<td>FBF&lt;sub&gt;rest&lt;/sub&gt; (mL/100mL/min)</td>
<td>1.90 ± 0.78</td>
<td>1.66 ± 0.70</td>
</tr>
<tr>
<td>FVR&lt;sub&gt;rest&lt;/sub&gt; (U)</td>
<td>44.5 ± 16.2</td>
<td>60.2 ± 25.4 *</td>
</tr>
<tr>
<td>FVC&lt;sub&gt;rest&lt;/sub&gt; (mL/100mL/min)</td>
<td>3.95 ± 0.73</td>
<td>3.89 ± 1.07</td>
</tr>
<tr>
<td>FVO&lt;sub&gt;rest&lt;/sub&gt; (mL/100mL/min)</td>
<td>48.2 ± 9.82</td>
<td>41.1 ± 8.70 +</td>
</tr>
<tr>
<td><strong>After Occlusion</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR&lt;sub&gt;occ&lt;/sub&gt; (beat/min)</td>
<td>59.0 ± 8.6</td>
<td>62.7 ± 16.5</td>
</tr>
<tr>
<td>SBP&lt;sub&gt;occ&lt;/sub&gt; (mm Hg)</td>
<td>104.6 ± 7.6</td>
<td>131.8 ± 27.7 *</td>
</tr>
<tr>
<td>DBP&lt;sub&gt;occ&lt;/sub&gt; (mm Hg)</td>
<td>60.9 ± 4.8</td>
<td>68.0 ± 8.1 *</td>
</tr>
<tr>
<td>MAP&lt;sub&gt;occ&lt;/sub&gt; (mm Hg)</td>
<td>75.5 ± 5.2</td>
<td>89.3 ± 13.8 *</td>
</tr>
<tr>
<td>FBF&lt;sub&gt;occ&lt;/sub&gt; (mL/100mL/min)</td>
<td>21.9 ± 5.54</td>
<td>18.3 ± 5.43 +</td>
</tr>
<tr>
<td>FVR&lt;sub&gt;occ&lt;/sub&gt; (U)</td>
<td>3.61 ± 0.86</td>
<td>5.37 ± 2.00 *</td>
</tr>
<tr>
<td>FVC&lt;sub&gt;occ&lt;/sub&gt; (mL/100mL/min)</td>
<td>2.87 ± 0.92</td>
<td>2.80 ± 1.18</td>
</tr>
<tr>
<td>FVO&lt;sub&gt;occ&lt;/sub&gt; (mL/100mL/min)</td>
<td>43.0 ± 12.5</td>
<td>43.1 ± 13.3</td>
</tr>
</tbody>
</table>

Values are mean ± SD. HR = Heart rate. SBP = Systolic blood pressure. DBP = Diastolic blood pressure. MAP = Mean arterial blood pressure. FBF = Forearm blood inflow. FVR = Forearm vascular resistance. FVC = Forearm venous capacitance. FVO = Forearm venous outflow.

* = Different from young group (p ≤ 0.05). + = trend towards an age group difference (p > 0.05).

Table B.5 Pre-exercise HR, BP, & Vascular Function Indices

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th>Older</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5-RM</td>
<td>15-RM</td>
</tr>
<tr>
<td>HR</td>
<td>72.5 ±12.0</td>
<td>71.2 ±12.0</td>
</tr>
<tr>
<td>SBP</td>
<td>104.4 ± 9.0</td>
<td>105.0 ± 6.4</td>
</tr>
<tr>
<td>DBP</td>
<td>59.3 ±10.2</td>
<td>58.9 ± 8.3</td>
</tr>
<tr>
<td>MAP</td>
<td>74.3 ± 9.6</td>
<td>74.3 ± 7.2</td>
</tr>
<tr>
<td>FBF</td>
<td>1.93 ± 0.4</td>
<td>1.93 ± 0.6</td>
</tr>
<tr>
<td>FVR</td>
<td>38.3 ± 6.5</td>
<td>39.0 ± 9.9</td>
</tr>
</tbody>
</table>

Values are mean ± SD. HR = Heart rate (beat/min). SBP = Systolic blood pressure (mm Hg) DBP = Diastolic blood pressure (mm Hg). MAP = Mean arterial blood pressure (mm Hg). FBF = Forearm blood inflow (mL/100mL/min). FVR = Forearm Vascular Resistance (Units).

* = Different from young group (p ≤ 0.05).
B.2. Systolic Blood Pressure

Systolic blood pressure responses during (peak SBP) and immediately after RE (local min and local max) were evaluated using a 4x2x2 (time x age x exercise condition) mixed model ANOVA with repeated measures on time (pre-exercise, peak, local min, local max) (Figure B.1, and Table B.6).

Figure B.1  Systolic Blood Pressure Before, During & Immediately After Resistance Exercise by Age & Exercise Condition in Women
Values are mean ± SD. Systolic blood pressure (SBP) before (Pre-), during (Peak), and immediately (local min and local max) after RE. * = Diff from 5-RM condition value in the older group ($p < 0.006$). † / ‡ = 5- / 15-RM Old group values diff from pre-exercise level ($p < 0.05$). $ / # = 5- / 15-RM young group values diff from pre-exercise level ($p < 0.05$).
Table B.6  Systolic Blood Pressure Before, During, and Immediately After RE

<table>
<thead>
<tr>
<th></th>
<th>Young 5RM</th>
<th>Young 15RM</th>
<th>Old 5RM</th>
<th>Old 15RM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-</td>
<td>104.4 ± 8.0</td>
<td>105.0 ± 6.4</td>
<td>121.3 ± 11.7</td>
<td>123.7 ± 15.1</td>
</tr>
<tr>
<td>Peak</td>
<td>138.7 ± 15.0 $</td>
<td>147.0 ± 25.1 #</td>
<td>145.9 ± 17.2†</td>
<td>164.3 ± 22.6‡*</td>
</tr>
<tr>
<td>Local min</td>
<td>115.6 ± 12.9 $</td>
<td>113.0 ± 17.3</td>
<td>125.2 ± 7.9</td>
<td>141.0 ± 18.8‡*</td>
</tr>
<tr>
<td>Local max</td>
<td>119.4 ± 11.9 $</td>
<td>116.6 ± 17.9 #</td>
<td>128.2 ± 9.6</td>
<td>143.5 ± 19.1‡*</td>
</tr>
</tbody>
</table>

Values are mm Hg and represent mean ± SD. Systolic blood pressure (SBP) before (Pre-), during (Peak), and immediately (Local min and local max) after RE. * = Diff from 5-RM condition in the older group ($p < 0.006$). † / ‡ = 5- / 15-RM Old group values diff from pre-exercise level ($p < 0.05$). $ / # = 5- / 15-RM young group values diff from pre-exercise level ($p < 0.05$).

The results revealed: 1.) a significant main effect of test period effect ($p = 0.0001$) such that pre-exercise value was lower than peak SBP, local min, and local max, but peak SBP was higher than all other test periods; 2.) a significant main effect of age group ($p = 0.0001$) such that SBP was higher among the older women across the test conditions; and 3.) a significant main effect of exercise condition ($p = 0.047$) indicating that SBP values at peak exercise, local min, and local max were higher with the 15-RM condition (Figure B.1, and Table B.6)

There was also a tendency towards an effect of age x exercise condition interaction ($p = 0.08$) and test condition x exercise condition interaction ($p = 0.08$). Therefore, inferences from main effects (test condition, exercise condition, and age group) may be tempered by a nearly significant three-way (test period x age x exercise condition) interaction ($p = 0.10$). Such an interaction would suggest that the effects of exercise condition on SBP during different test periods are different in the older adults compared to younger counterparts. More specifically, the post-exercise SBPs (local min and max) appear to be influenced by exercise condition in the older adults, but not in the younger adults. Figure B.1 illustrates the tendency for 15-RM exercise condition to result in
higher SBP during local min and local max in the older group (in comparison to the 5-RM exercise condition), but not in the young participants.

Given that the higher pre-exercise SBP values among the older women might explain the higher SBP values during and immediately after RE in this group, 2x2 univariate age by exercise condition ANOVAs were used to analyze the magnitudes of SBP changes (magnitude of increase, magnitude of drop, and magnitude of recovery) (Table B.7).

<table>
<thead>
<tr>
<th>Table B.7</th>
<th>Magnitude of Systolic Blood Pressure Changes During and Immediately After RE by Age and Exercise Condition in Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Young</td>
</tr>
<tr>
<td></td>
<td>5-RM</td>
</tr>
<tr>
<td>Increase</td>
<td></td>
</tr>
<tr>
<td>Young</td>
<td>34.3±12.6</td>
</tr>
<tr>
<td>Old</td>
<td>24.6±13.1</td>
</tr>
<tr>
<td>Decline</td>
<td>-23.2±10.9</td>
</tr>
<tr>
<td>Young</td>
<td>-23.2±10.9</td>
</tr>
<tr>
<td>Old</td>
<td>-23.3±15.2</td>
</tr>
<tr>
<td>Recovery</td>
<td>3.9±3.5</td>
</tr>
<tr>
<td>Young</td>
<td>3.9±3.5</td>
</tr>
<tr>
<td>Old</td>
<td>3.0±3.6</td>
</tr>
</tbody>
</table>

Values are mean ± SD, and represent mm Hg. * = Different from 5-RM condition within each age group (p < 0.05).

The results revealed neither significant age or exercise condition main effects nor age by exercise condition interaction effect on the magnitude of increase (peak minus pre-exercise) and magnitude of SBP drop from peak exercise to local min. However, follow up pairwise comparisons revealed a significant exercise condition effect on the magnitude of SBP increase indicating that the 15-RM exercise condition was related to a greater SBP increase from pre-exercise condition to peak exercise (p = 0.002). Similarly, t-test comparisons within each age group revealed that the magnitude of SBP drop from peak exercise to local min was greater following the 15-RM exercise condition among the young women in comparison to the 5-RM exercise condition. The magnitude of SBP drop among the older women, however, was similar under both exercise conditions. In
regards to the SBP change from local min to local max, ANOVA revealed a nearly significant age-related effect ($p = 0.056$) and a trend for exercise condition effect ($p = 0.13$) on the magnitude of SBP recovery between both time points suggesting a smaller magnitude of SBP recovery among the older women and following the 5-RM exercise condition, respectively.

In regard to the time and mean rate of SBP changes from peak to local min, and from local min to local max, 2x2 univariate age by exercise condition ANOVAs were used. The results revealed no significant age-related or exercise condition differences in any of these parameters. However, follow up pairwise comparisons revealed a tendency towards a slower mean rate of SBP decline among the older women (Table B.8).

### Table B.8  Systolic Blood Pressure - Time & Mean Rate of Change From Peak to Local Minimum, & Local Minimum to Local Maximum After RE by Age and Exercise Condition in Women

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th></th>
<th>Old</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5-RM</td>
<td>15-RM</td>
<td>5-RM</td>
<td>15-RM</td>
</tr>
<tr>
<td>Time Peak-Local Min</td>
<td>19.3 ± 8.7</td>
<td>22.0 ± 13.9</td>
<td>20.3 ± 7.04</td>
<td>21.0 ± 11.8</td>
</tr>
<tr>
<td>(sec)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time Local Min-Local</td>
<td>10.7 ± 5.9</td>
<td>9.9 ± 7.6</td>
<td>9.8 ± 3.6</td>
<td>7.7 ± 1.0</td>
</tr>
<tr>
<td>Max (sec)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Rate Decline</td>
<td>1.3 ± 0.7</td>
<td>1.7 ± 1.2</td>
<td>1.1 ± 0.8</td>
<td>1.2 ± 0.7</td>
</tr>
<tr>
<td>(mm Hg/sec)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Rate Recovery</td>
<td>0.4 ± 0.3</td>
<td>0.3 ± 0.5</td>
<td>0.3 ± 0.3</td>
<td>0.3 ± 0.4</td>
</tr>
<tr>
<td>(mm Hg/sec)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ± SD. Local Min = Local minimum. Local Max = Local Maximum.

Similarly, a 12x2x2 (time x age x exercise condition) mixed model ANOVA with repeated measured on time (pre-exercise, peak, R1’, R3’, R5’, R10’, R15’, R20’, R30’, R40’, R50’, and R60’) was employed to evaluate the SBP behavior before, during, and for 1 hour after RE. The results revealed a test period effect ($p = 0.0001$) such that pre-exercise value was lower than peak SBP, R1’, and R3’, but SBP values were lower from
R5’ to R60’ compared to R1’ (see Figure B.2). Further, peak SBP was higher than pre-exercise level and all recovery periods (Figure B.2, and Table B.9).

The results revealed a significant main effect of age \( (p = 0.0001) \) such that pre-exercise SBP and all SBP recovery periods were higher among the older women. However, the mentioned main effects might be modified for a trend towards a test condition by exercise condition interaction effect on SBP \( (p = 0.06) \) indicating that the 15-RM exercise condition appeared to be associated with higher SBP at peak, 1’, 3’, 15’, 20’, 40’, and 50’ in comparison to the 5-RM exercise condition.

![Figure B.2 Systolic Blood Pressure Before, During & For 1 Hour After RE by Age and Exercise Condition in Women](image)

Values are mean ± SD. Systolic blood pressure (SBP) before (Pre-), during (Peak), and for 1 hour after RE. * = Diff from the 5-RM condition value in the older group \( (p < 0.05) \). + = tended to be diff from the 5-RM condition in the older group \( (p < 0.15) \). † / ‡ = 5- / 15-RM older group values diff from pre-exercise level \( (p < 0.05) \). $ / # = 5- / 15-RM young group values diff from pre-exercise level \( (p < 0.05) \).
Table B.9  Age Group by Exercise Condition Interaction Effect on SBP Responses During, & For 1 Hour After RE in Women

<table>
<thead>
<tr>
<th></th>
<th>Young 5-RM</th>
<th>Young 15-RM</th>
<th>Old 5-RM</th>
<th>Old 15-RM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-</td>
<td>104.4 ± 8.9</td>
<td>105.0 ± 6.4</td>
<td>121.3 ± 11.7</td>
<td>123.7 ± 15.1</td>
</tr>
<tr>
<td>Peak</td>
<td>138.7 ± 15.0 $</td>
<td>146.9 ± 25.1 #</td>
<td>145.9 ± 17.2 †</td>
<td>164.3 ± 22.6 ‡ *</td>
</tr>
<tr>
<td>R1'</td>
<td>116.2 ± 10.9 $</td>
<td>110.0 ± 15.3</td>
<td>122.1 ± 8.4</td>
<td>132.3 ± 20.9 ‡ *</td>
</tr>
<tr>
<td>R3'</td>
<td>112.5 ± 10.6 $</td>
<td>107.2 ± 15.4</td>
<td>118.7 ± 8.5</td>
<td>127.9 ± 18.7 *</td>
</tr>
<tr>
<td>R5'</td>
<td>110.9 ± 9.3 $</td>
<td>105.5 ± 12.8</td>
<td>118.1 ± 11.5</td>
<td>121.2 ± 19.1</td>
</tr>
<tr>
<td>R10'</td>
<td>106.9 ± 12.5</td>
<td>102.5 ± 8.7</td>
<td>113.6 ± 11.3 †</td>
<td>115.5 ± 15.9 ‡</td>
</tr>
<tr>
<td>R15'</td>
<td>102.7 ± 12.5</td>
<td>105.0 ± 11.9</td>
<td>112.7 ± 13.1 †</td>
<td>117.3 ± 13.3 ‡ +</td>
</tr>
<tr>
<td>R20'</td>
<td>104.6 ± 9.6</td>
<td>105.6 ± 12.4</td>
<td>113.5 ± 15.9 †</td>
<td>120.2 ± 16.1 +</td>
</tr>
<tr>
<td>R30'</td>
<td>104.6 ± 9.3</td>
<td>103.1 ± 14.1</td>
<td>116.0 ± 13.5 †</td>
<td>117.5 ± 15.5 ‡</td>
</tr>
<tr>
<td>R40'</td>
<td>105.3 ± 9.8</td>
<td>105.4 ± 14.7</td>
<td>112.6 ± 15.6 †</td>
<td>119.5 ± 15.4 +</td>
</tr>
<tr>
<td>R50'</td>
<td>104.6 ± 9.7</td>
<td>106.2 ± 12.1</td>
<td>111.5 ± 16.3 †</td>
<td>118.8 ± 14.9 +</td>
</tr>
<tr>
<td>R60'</td>
<td>106.4 ± 11.2</td>
<td>102.9 ± 15.6</td>
<td>110.6 ± 16.7 †</td>
<td>115.5 ± 16.3 ‡</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Systolic blood pressure before (Pre-), during (Peak), and for 1 hour after RE. * = Diff. from the 5-RM condition in the older group ($p < 0.05$). + = tended to be diff from the 5-RM condition in the older group ($p < 0.15$). † / ‡ = 5- / 15-RM older group values diff from pre-exercise ($p < 0.05$). $ / # = 5- / 15-RM young group values diff from pre-exercise ($p < 0.05$).

Likewise, an 11x2x2 (time x age x exercise condition) mixed model ANOVA with repeated measures on time (local min, R1’, R3’, R5’, R10’, R15’, R20’, R30’, R40’, R50’, and R60’) was employed to evaluate post-exercise SBP drops below pre-exercise level (calculated post-exercise SBP minus pre-exercise SBP) from local min to 60 minutes of recovery (R60’) following RE. The results revealed a significant test period effect on the magnitude of post-exercise SBP drops after RE ($p = 0.0001$) with a decrease from local min toward and below pre-exercise level during the first 5 minutes of recovery. Thereafter, post-exercise SBP dropped below pre-exercise level from R10’ to R60’ maintained approximately 3 or 4 mmHg below pre-exercise level. Further, there was an age-related main effect ($p = 0.021$) indicating larger post-exercise SBP drop below pre-exercise level among the older women (Figure B.3).
However, the main effect of age group on the magnitude of SBP drop below pre-exercise after RE appeared to be tempered by an age x exercise condition ($p = 0.16$), and a test condition x exercise condition ($p = 0.08$) interaction effects.

Lastly, a 2x2x2 (time x age x exercise condition) mixed model ANOVA with repeated measures on time (1’ and 3’ SBP/peak ratios) was used to evaluate post-exercise 1’ and 3’ SBP/peak ratios (calculated post-exercise SBP at 1, and 3 minutes divided by peak SBP). The results revealed a main effect of test period ($p = 0.0001$) such that 1-min SBP/peak ratio was greater than 3-min SBP/peak ratio. Further, there was a main effect of exercise condition ($p = 0.001$), such that post-exercise SBP ratios were greater following the 5-RM exercise condition. Further, there was a trend towards an age group difference ($p = 0.13$), which would suggest that post-exercise SBP ratios were greater among the older women than their younger counterparts (Table B.10).

Figure B.3  Magnitude of Systolic Blood Pressure Drop After RE by Age and Exercise Condition in Women
Magnitude of systolic blood pressure drop below pre-exercise level after resistance exercise (RE). Values are mean $\pm$ SE.
Table B.10  SBP / Peak Ratio at 1 Minute and 3 Minutes After RE by Age and Exercise Condition in Women

<table>
<thead>
<tr>
<th></th>
<th>Young 5-RM</th>
<th>Young 15-RM</th>
<th>Old 5-RM</th>
<th>Old 15-RM</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-min SBP/ peak ratio</td>
<td>0.84 ± 0.06 *</td>
<td>0.76 ± 0.09</td>
<td>0.84 ± 0.06 *</td>
<td>0.81 ± 0.07</td>
</tr>
<tr>
<td>3-min SBP/ peak ratio</td>
<td>0.81 ± 0.07 *</td>
<td>0.74 ± 0.08</td>
<td>0.82 ± 0.06 *</td>
<td>0.78 ± 0.07</td>
</tr>
</tbody>
</table>

Values are mean ± SD. * = Different from 15-RM condition within each age group (p < 0.05).

B.3. Diastolic Blood Pressure

Diastolic blood pressure responses during and immediately after RE were evaluated using a 4x2x2 (time x age x exercise condition) mixed model ANOVA with repeated measures on time (pre-exercise, peak, local min, local max) (Figure B.4, & Table B.11).

![Figure B.4 Diastolic Blood Pressure Before, During and Immediately After RE by Age and Exercise Condition in Women](image)

Values are mean ± SD. Diastolic blood pressure (DBP) before (Pre-), during (Peak), and immediately (local min and local max) after RE. * = Diff from 5-RM condition value in older group (p ≤ 0.04). † / ‡ = 5- / 15-RM older group values diff from pre-exercise level (p ≤ 0.05). $ / # = 5- / 15-RM young group values diff from pre-exercise level (p ≤ 0.05).
Table B.11  Diastolic Blood Pressure Before, During, & Immediately After RE

<table>
<thead>
<tr>
<th></th>
<th>Young 5RM</th>
<th>Young 15RM</th>
<th>Old 5RM</th>
<th>Old 15RM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-</td>
<td>59.3 ± 10.2</td>
<td>58.9 ± 8.3</td>
<td>57.9 ± 8.6</td>
<td>59.9 ± 9.5</td>
</tr>
<tr>
<td>Peak</td>
<td>90.4 ± 14.3 $</td>
<td>92.6 ± 15.4 #</td>
<td>73.8 ± 9.9 †</td>
<td>87.9 ± 15.4‡*</td>
</tr>
<tr>
<td>Local min</td>
<td>55.8 ± 11.6</td>
<td>53.3 ± 10.4 #</td>
<td>52.3 ±10.0 †</td>
<td>62.0 ± 15.5 *</td>
</tr>
<tr>
<td>Local max</td>
<td>59.6 ± 10.5</td>
<td>57.5 ± 10.4</td>
<td>54.4 ± 9.5</td>
<td>64.6 ± 15.4 *</td>
</tr>
</tbody>
</table>

Values are in mm Hg and represent mean ± SD. Diastolic blood pressure (DBP) before (Pre-), during (Peak), and immediately (local min and local max) after resistance exercise (RE). * = Diff from the 5-RM condition value in the older group ($p < 0.04$). † / ‡ = 5- / 15-RM older group values diff from pre-exercise level ($p < 0.05$). $ / # = 5- / 15-RM young group values diff from pre-exercise level ($p \leq 0.05$).

The results revealed a main effect of test condition ($p = 0.0001$) such that local min DBP was lower than pre-exercise, peak exercise, and local max; and peak exercise DBP was higher than all test periods. This main effect is accompanied by a nearly significant age by exercise condition interaction ($p = 0.067$) such that exercise condition tended to have a greater influence on the older as compared to younger adults (Figure B.4, & Table B.11).

Once again, 2x2 univariate age by exercise condition ANOVAs were used to analyze, in this case, the magnitudes of DBP changes (magnitude of increase, magnitude of drop, and magnitude of recovery) (Table B.12).

Table B.12  Magnitude of Diastolic Blood Pressure Changes During and Immediately After RE by Age and Exercise Condition in Women

<table>
<thead>
<tr>
<th></th>
<th>Young 5-RM</th>
<th>Young 15-RM</th>
<th>Old 5-RM</th>
<th>Old 15-RM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Magnitude of increase</td>
<td>30.8 ± 11.9</td>
<td>33.7 ± 16.4</td>
<td>17.6 ± 5.5</td>
<td>29.1 ± 12.2</td>
</tr>
<tr>
<td>Magnitude of drop</td>
<td>-34.6 ± 11.0</td>
<td>-39.3 ± 14.7</td>
<td>-21.5 ± 9.8</td>
<td>-25.9 ± 13.3</td>
</tr>
<tr>
<td>Magnitude of recovery</td>
<td>3.9 ± 2.1</td>
<td>4.2 ± 6.0</td>
<td>2.1 ± 1.3</td>
<td>2.5 ± 2.2</td>
</tr>
</tbody>
</table>

Values are in mm Hg, and represent mean ± SD.
With regard to the increase in DBP from pre-exercise to peak exercise (magnitude of increase), the results of the ANOVA revealed no significant main effects of age nor exercise condition, but there was a trend towards an age by exercise condition interaction effect on the magnitude of DBP increase ($p = 0.15$) suggesting a tendency for a greater magnitude of DBP increase with 15-RM exercise among the older women, but no effect on the magnitude of DBP increase among the young women. Follow up t-tests indicate that the magnitude of DBP increase from pre-exercise level to peak is exercise condition dependent in the older subjects ($p = 0.005$) but not in the young subjects ($p = 0.58$).

The results of magnitude of DBP drop revealed significant main effects of age group ($p = 0.007$), and exercise condition ($p = 0.019$) indicating a smaller magnitude of DBP drop among the older women, and a greater magnitude of drop in DBP with 15-RM exercise. In contrast, the results of magnitude of DBP recovery revealed a significant main effect of age group such that a smaller magnitude of DBP recovery was observed among the older women compared to their younger counterparts ($p = 0.018$), and a trend towards an exercise condition effect ($p = 0.08$), that would suggest smaller magnitude of DBP recovery with 5-RM exercise condition.

Concerning the time and mean rate of DBP changes from peak to local min, and from local min to local max, 2x2 univariate age by exercise condition ANOVAs were used (Table B.13). The results revealed significant main effects of age ($p = 0.009$) and exercise condition ($p = 0.006$) on the time from peak to local min, such that this phase of recovery was longer among the older women, and following the 15-RM exercise condition. However, there were no significant main effects of age or exercise condition on the time from local min to local max (see Table B.13). Follow up t-tests indicate that the mean
rate of DBP recovery from local min to local max is exercise condition dependent in the older subjects ($p = 0.005$) but not in the young subjects ($p = 0.58$).

**Table B.13** Diastolic Blood Pressure - Time & Mean Rate of Change from Peak to Local Minimum, & Local Minimum to Local Maximum After RE by Age and Exercise Condition in Women

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5-RM</td>
<td>15-RM</td>
</tr>
<tr>
<td>Time Peak-Local Min (sec)</td>
<td>16.7 ± 5.1</td>
<td>19.6 ± 3.8</td>
</tr>
<tr>
<td>Time Local Min-Local Max (sec)</td>
<td>11.2 ± 5.5</td>
<td>15.8 ± 10.6</td>
</tr>
<tr>
<td>Mean Rate of Decline (mm Hg/sec)</td>
<td>2.2 ± 0.8</td>
<td>2.0 ± 0.8</td>
</tr>
<tr>
<td>Mean Rate of Recovery (mm Hg/sec)</td>
<td>0.4 ± 0.3</td>
<td>0.3 ± 0.3</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Local Min = Local minimum. Local Max = Local Maximum.

With regard to the mean **rate of change** in DBP from peak to local min, the results revealed only a trend toward age-related differences ($p = 0.07$) suggesting a tendency for a slower rate of DBP decline among the older women. In regards to the mean **rate of change** in DBP from local min to local max (mean rate of DBP recovery), the results revealed a trend towards an age by exercise condition interaction ($p = 0.15$) suggesting that the mean rate of DBP recovery tended to be slower among the older women and mostly following the 5-RM exercise condition, while the mean rate of DBP recovery tended to be faster among the young women and mostly following the 15-RM exercise condition (See Table B.8). However, follow up paired t-test comparison did not support that conclusion.

Likewise, a 12x2x2 (time x age x exercise condition) mixed model ANOVA with repeated measures on time (pre-exercise, peak, R1’, R3’, R5’, R10’, R15’, R20’, R30’, R40’, R50’, and R60’) was used to evaluate for time, age and exercise condition effects on DBP before, during, and for 1 hour after RE. The results reveal a significant test.
condition effect ($p = 0.0001$) such that pre-exercise DBP was higher than all recovery periods, but lower than peak DBP. Further, peak DBP was higher than pre-exercise level and all recovery periods; $R1'$ was higher than $R10'$ and from $R30'$ to $R60'$; $R3'$ was higher than $R30'$, $R50'$, and $R60'$; and $R5'$ was only higher than $R50'$. This also indicates that the recovery periods from $R10'$ through $R60'$ were lower or close to being lower than $R1'$, $R3'$, and $R5'$. There was also a significant age-related main effect ($p = 0.047$) such that peak, and all recovery periods from $R10'$ through $R60'$ were lower than pre-exercise level among the older women, except $R10'$ ($p = 0.13$) and $R30'$ ($p = 0.08$) (Figure B.5).

![Graph showing diastolic blood pressure (DBP) before, during, and after exercise by age and exercise condition in women.](image)

**Figure B.5** Diastolic Blood Pressure Before, During and For 1 Hour After RE by Age & Exercise Condition in Women
Values are mean ± SD. Diastolic blood pressure (DBP) before (Pre-), during (Peak), and for 1 hour after RE. * = Diff. from 5-RM condition in the older group ($p < 0.05$). + = tended to be diff from 5-RM condition in the older group ($p < 0.15$). † / ‡ = 5- / 15-RM older group values diff from pre-exercise ($p < 0.05$). $\# / \#$ = 5- / 15-RM young group values diff from pre-exercise ($p < 0.05$).
Table B.14  Age Group by Exercise Condition Interaction Effect on DBP
Before, During, & For 1 Hour After RE in Women

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th></th>
<th>Old</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5-RM</td>
<td>15-RM</td>
<td>5-RM</td>
<td>15-RM</td>
</tr>
<tr>
<td>Pre-</td>
<td>59.3 + 10.2</td>
<td>58.9 + 8.3</td>
<td>57.9 + 8.6</td>
<td>59.9 + 9.5</td>
</tr>
<tr>
<td>Peak</td>
<td>90.4 + 14.3 $</td>
<td>92.6 + 15.4 #</td>
<td>73.8 + 9.9 †</td>
<td>87.9 + 15.4‡*</td>
</tr>
<tr>
<td>R1’</td>
<td>59.3 + 11.7</td>
<td>55.1 + 10.4 #</td>
<td>53.0 + 9.7</td>
<td>57.5 + 14.5</td>
</tr>
<tr>
<td>R3’</td>
<td>58.8 + 11.2</td>
<td>54.3 + 10.9 #</td>
<td>51.7 + 9.5 †</td>
<td>57.1 + 13.5 +</td>
</tr>
<tr>
<td>R5’</td>
<td>58.1 + 11.4</td>
<td>53.7 + 8.8 #</td>
<td>52.2 + 10.2†</td>
<td>56.1 + 14.4</td>
</tr>
<tr>
<td>R10’</td>
<td>56.7 + 13.0</td>
<td>54.6 + 8.7 #</td>
<td>50.6 + 9.5 †</td>
<td>52.1 + 11.0‡</td>
</tr>
<tr>
<td>R15’</td>
<td>55.8 + 9.8</td>
<td>58.7 + 11.2</td>
<td>48.7 + 10.2†</td>
<td>54.4 + 8.1 ‡*</td>
</tr>
<tr>
<td>R20’</td>
<td>56.2 + 10.1</td>
<td>57.6 + 10.2</td>
<td>47.9 + 10.0†</td>
<td>55.2 + 9.2 ‡*</td>
</tr>
<tr>
<td>R30’</td>
<td>55.2 + 9.6</td>
<td>55.5 + 12.0</td>
<td>48.8 + 9.8 †</td>
<td>52.3 + 9.7 ‡</td>
</tr>
<tr>
<td>R40’</td>
<td>56.9 + 7.4</td>
<td>57.1 + 12.8</td>
<td>48.9 + 10.3†</td>
<td>52.7 + 12.3‡</td>
</tr>
<tr>
<td>R50’</td>
<td>55.0 + 8.0</td>
<td>56.2 + 11.5</td>
<td>45.6 + 10.4†</td>
<td>53.6 + 9.7 ‡*</td>
</tr>
<tr>
<td>R60’</td>
<td>56.9 + 10.6</td>
<td>55.9 + 13.1</td>
<td>46.2 + 7.9 †</td>
<td>52.1 + 8.9 ‡+</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Diastolic blood pressure before (Pre-), during (Peak), and for 1 hour after RE. * = Diff. from 5-RM condition in the older group (p < 0.05). + = tended to be diff from 5-RM condition in the older group (p < 0.15). † / ‡ = 5- / 15-RM older group values diff from pre-exercise (p < 0.05). $ / # = 5- / 15-RM young group values diff from pre-exercise (p < 0.05).

Moreover, follow-up pairwise comparisons by age and exercise condition revealed lower post-exercise DBPs in comparison to pre-exercise values among the older women for both exercise conditions, such that DBPs (mean differences) reached statistical significance throughout R3’ to R60’ following 5-RM exercise condition, and from R10’ to R60’ (except at R20’) following 15-RM exercise condition. On the other hand, the pairwise comparisons of post-exercise DBPs to pre-exercise level among the young women did not reach statistical significance following the 5-RM condition, and reached statistical significance only from R3’ to R10’ following the 15-RM condition.

Likewise, an 11x2x2 (time x age x exercise condition) mixed model ANOVA with repeated measures on time was employed to evaluate the drop in DBP compared to pre-exercise (i.e., magnitude of post-exercise DBP drops below pre-exercise level), and calculated as pre-exercise DBP subtracted from DBPs observed at local min, R1’, R3’,

155
R5’, R10’, R15’, R20’, R30’, R40’, R50’, and R60’. The results revealed a significant main effect of age ($p = 0.046$) such that DBP among the older adult women dropped further below pre-exercise level compared to their younger counterparts. The effect of time period did not achieve statistical significance; however, there appeared to be a tendency ($p = 0.078$) for DBP to continue to drop further below pre-exercise from R1’ to R60’ (Figure B.6).

![Figure B.6 Magnitude of Diastolic Blood Pressure Drop After RE by Age and Exercise Condition in Women](image)

Magnitude of diastolic blood pressure drop below pre-exercise level following resistance exercise (RE). Values are mean ± SE.

Last of all, a 2x2x2 (time x age x exercise condition) mixed model ANOVA with repeated measures on time (1-min DBP/peak, and 3-min DBP/peak ratios) was used to evaluate 1-, and 3-minute post-exercise DBP ratios (1-minute and 3-minute DBPs divided by peak DBP). The results revealed a significant main effect of age ($p = 0.013$) such that post-exercise DBP ratios were greater among the older women than among their young counterparts. Further, there was a significant main effect of exercise condition such that post-exercise DBP ratios were greater following the 5-RM exercise (Table B.15).
Table B.15  DBP / Peak Ratio at 1-Minute and 3-Minute After RE by Age and Exercise Condition in Women

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5-RM</td>
<td>15-RM</td>
</tr>
<tr>
<td>1-min DBP/ peak ratio</td>
<td>0.66 ± 0.09</td>
<td>0.60 ± 0.09</td>
</tr>
<tr>
<td>3-min DBP/ peak ratio</td>
<td>0.65 ± 0.08</td>
<td>0.59 ± 0.11</td>
</tr>
</tbody>
</table>

Values are mean ± SD.

B.4. Heart Rate

Heart rate responses during and immediately after RE were also evaluated using a 4x2x2 (time x age x exercise condition) mixed model ANOVA with repeated measures on time (pre-exercise, peak, local min, local max) (Figure B.7, & Table B.12).

![Figure B.7](image-url)  

**Figure B.7** Heart Rate Before, During and Immediately After RE by Age and Exercise Condition in Women  
Values are mean ± SD. Heart rate (HR) before (Pre-), during (Peak), and immediately after (local min and local max) resistance exercise. * = Diff from young group ($p < 0.05$). † / ‡ = 5- / 15-RM older group values diff from pre-exercise level ($p < 0.05$). + = tended to be diff from 5-RM condition value in the young group ($p < 0.05$). $ / # = 5- / 15-RM young group values diff from pre-exercise level ($p < 0.05$).
Table B.16 Heart Rate Before, During, & Immediately After RE

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5-RM</td>
<td>15-RM</td>
</tr>
<tr>
<td>Pre-exercise (beat/min)</td>
<td>72.5 ± 12.0</td>
<td>71.2 ± 12.0</td>
</tr>
<tr>
<td>Peak Exercise (beat/min)</td>
<td>118.0 ± 12.6$</td>
<td>124.2 ± 14.7##</td>
</tr>
<tr>
<td>Local Minimum (beat/min)</td>
<td>83.6 ± 16.8$</td>
<td>90.8 ± 23.2##</td>
</tr>
<tr>
<td>Local Maximum (beat/min)</td>
<td>88.4 ± 14.7$</td>
<td>97.0 ± 21.6##</td>
</tr>
</tbody>
</table>

Values are mean ± SD. Heart rate (HR) before (Pre-), during (Peak), and immediately after (local min and local max) resistance exercise. * = Diff from young group (p < 0.05). † / ‡ = 5- / 15-RM older group values diff from pre-exercise level (p < 0.05). + = tended to be diff from 5-RM condition value in the young group (p < 0.05). $ / # = 5- / 15-RM young group values diff from pre-exercise level (p ≤ 0.05).

The results revealed a main effect of test condition (p = 0.0001) such that pre-exercise HR was lower than peak, local min, and local max, but peak HR was higher than all other test periods (pre-exercise, local min, and local max). There was also a main effect of age (p = 0.0001) revealing lower HRs among the older women under all conditions (Figure B.7, & Table B.16).

Inasmuch as the lower pre-exercise HR values among the older women might also explain the lower HR values during and immediately after RE in this group, so that 2x2 univariate age by exercise condition ANOVAs were used to evaluate the magnitudes of HR changes (peak minus pre-exercise, local minimum minus peak, and local maximum minus local minimum). The results revealed a significant main effect of age, such that the magnitude of HR increment (peak minus pre-exercise) was lower among the older women than among the young women (p = 0.036). Further, there was a trend towards an exercise condition effect, which appears to reveal a greater magnitude of HR increase to 15-RM exercise condition as compared to 5-RM exercise condition (p=0.132). In contrast, the results of magnitude of HR drop (from peak to local minimum) revealed...
only a trend of age-related differences that seems to suggest a lower magnitude of drop among the older women \( (p = 0.058) \). Pairwise comparison on the magnitude of drop, however, suggests a smaller magnitude of HR drop among the older women in comparison to young women \( (p = 0.002) \) (Table B.17).

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5-RM</td>
<td>15-RM</td>
</tr>
<tr>
<td>Magnitude of increase</td>
<td>45.5 ± 13.3</td>
<td>53.0 ± 16.5</td>
</tr>
<tr>
<td>Magnitude of drop</td>
<td>-34.4 ± 16.0</td>
<td>-33.4 ± 22.1</td>
</tr>
<tr>
<td>Magnitude of recovery</td>
<td>4.8 ± 7.0</td>
<td>6.2 ± 7.5</td>
</tr>
</tbody>
</table>

Values are in beats/min/sec, and represent mean ± SD.

With regard to the magnitude of HR recovery (local max minus local min), the effect of age did not quite reach statistical significance \( (p = 0.15) \) indicating a tendency for smaller magnitude of HR recovery among the older group. Pairwise comparisons on the magnitude of recovery revealed a smaller magnitude of recovery among the older women \( (p = 0.02) \).

With respect to the time and mean rate of HR changes from peak to local min, and from local min to local max, 2x2 univariate age by exercise condition ANOVAs were used. The results of the time from peak to local min HR revealed a trend of age-related differences \( (p = 0.07) \) such that the older women tended to take longer reaching local min HR. However, there were no effects of age or exercise condition on the time from local min to local max. Regarding the mean rates of decline and recovery, the ANOVA revealed a trend towards an age-related difference on the mean rate of decline \( (p = 0.11) \), and a significant main effect of age on mean rate of recovery \( (p = 0.033) \) (Table B.12).
Table B.18  Heart Rate - Time & Mean Rate of Changes From Peak to Local Minimum, & Local Minimum to Local Maximum After RE by Age & Exercise Intensity in Women

<table>
<thead>
<tr>
<th></th>
<th>Young 5-RM</th>
<th>Young 15-RM</th>
<th>Old 5-RM</th>
<th>Old 15-RM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time Peak-Local Min (sec)</td>
<td>34.3 ± 12.6</td>
<td>44.0 ± 23.1</td>
<td>24.6 ± 13.1</td>
<td>42.5 ± 16.5</td>
</tr>
<tr>
<td>Time Local Min-Local Max (sec)</td>
<td>23.2 ± 10.9</td>
<td>33.9 ± 20.7</td>
<td>20.6 ± 13.1</td>
<td>23.3 ± 15.2</td>
</tr>
<tr>
<td>Mean Rate of Decline (bpm/sec)</td>
<td>3.9 ± 3.5</td>
<td>3.6 ± 5.2</td>
<td>3.0 ± 3.6</td>
<td>2.5 ± 3.5</td>
</tr>
<tr>
<td>Mean Rate of Recovery (bpm/sec)</td>
<td>3.9 ± 3.5</td>
<td>3.6 ± 5.2</td>
<td>3.0 ± 3.6</td>
<td>2.5 ± 3.5</td>
</tr>
</tbody>
</table>

Values are mean ± SD.

In summary, these results suggest that the older women tended to have a longer time from peak to local min, but a slower rate of change in HR during that time. The time from local min to local max did not differ between age groups, but the rate of change was significantly slower in the older women (Table B.12).

Likewise, a 12x2x2 (time x age x exercise condition) mixed model ANOVA with repeated measures on time (pre-exercise, peak, R1’, R3’, R5’, R10’, R15’, R20’, R30’, R40’, R50’, and R60’) was employed to evaluate the HR behavior before, during, and for 1 hour following RE. The results revealed a significant main effect of test condition \( (p = 0.0001) \) such that pre-exercise HR was lower than peak HR and R1’, and higher than R10’, but HRs from R3’ to R60’ were not different from pre-exercise level. Peak HR was higher than pre-exercise value and all recovery periods. Further, there was a significant main effect of age \( (p = 0.0001) \) such that pre-exercise, peak, and all recovery period HR values were lower among the older women than among young women (Figure B.8).
**Figure B.8** Heart Rate Before, During and For 1 Hour After RE by Age & Exercise Condition in Women.

Values are mean ± SD. Heart Rate (HR) before (Pre-), during (Peak) and for 1 hour after resistance exercise (RE). * = Diff. from 5-RM condition in the older group ($p \leq 0.05$). + = tended to be diff from 5-RM condition in the older group ($p \leq 0.15$). † / ‡ = 5- / 15-RM older group values diff from pre-exercise ($p \leq 0.05$). $/$ # = 5- / 15-RM young group values diff from pre-exercise ($p \leq 0.05$).

**Table B.19** Age Group by Exercise Condition Interaction Effect on Heart Rate Before, During, & For 1 Hour After RE in Women

<table>
<thead>
<tr>
<th></th>
<th>Young 5-RM</th>
<th>15-RM</th>
<th>Old 5-RM</th>
<th>15-RM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-</td>
<td>72.5 ± 12.0</td>
<td>71.2 ± 12.0</td>
<td>64.0 ± 8.8</td>
<td>62.3 ± 7.7</td>
</tr>
<tr>
<td>Peak</td>
<td>118.0 ± 12.6 $</td>
<td>$ 124.2 ± 14.7 $</td>
<td>$ +</td>
<td>87.5 ± 9.6 †</td>
</tr>
<tr>
<td>R1’</td>
<td>79.0 ± 12.7 $</td>
<td>$ 85.3 ± 12.7 $</td>
<td>$ +</td>
<td>67.4 ± 9.3 †</td>
</tr>
<tr>
<td>R3’</td>
<td>70.8 ± 13.9 $</td>
<td>$ 73.4 ± 12.6</td>
<td>63.7 ± 10.3</td>
<td>63.2 ± 8.4</td>
</tr>
</tbody>
</table>
| R5’   | 69.6 ± 10.7 $|$ 71.9 ± 15.0 | 62.0 ± 9.1 † | 62.3 ± 8.5 *
| R10’  | 69.4 ± 9.1 $|$ 71.0 ± 10.2 | 62.2 ± 8.5 † | 61.2 ± 8.0 * |
| R15’  | 72.2 ± 12.3 | 71.3 ± 11.4 | 64.6 ± 10.9 | 61.9 ± 7.9 *
| R20’  | 72.8 ± 10.5 | 71.7 ± 10.5 | 64.0 ± 9.1 | 61.7 ± 7.0 *+ |
| R30’  | 72.5 ± 10.6 | 70.7 ± 11.7 | 64.1 ± 9.5 | 61.1 ± 7.1 ‡ +* |
| R40’  | 73.0 ± 7.8 | 73.2 ± 10.0 | 63.6 ± 8.7 | 60.9 ± 7.8 ‡ +* |
| R50’  | 70.2 ± 8.0 | 71.9 ± 10.6 | 62.0 ± 8.7 † | 61.5 ± 7.7 * |
| R60’  | 75.4 ± 11.4 | 74.6 ± 15.3 | 62.4 ± 8.0 † | 61.9 ± 7.4 * |

Values are mean ± SD. Heart rate before (Pre-), during (Peak), and for 1 hour after RE. * = Diff. from 5-RM condition in the older group ($p \leq 0.05$). + = tended to be diff from 5-RM condition in the older group ($p \leq 0.15$). † / ‡ = 5- / 15-RM older group values diff from pre-exercise ($p \leq 0.05$). $/$ # = 5- / 15-RM young group values diff from pre-exercise ($p \leq 0.05$).
Likewise, an 11x2x2 (time x age x exercise condition) mixed model ANOVA with repeated measures on time was employed to evaluate post-exercise HR changes from local min to R60’ (local minimum, R1’, R3’, R5’, R10’, R15’, R20’, R30’, R40’, R50’, and R60’). The results revealed a significant main effect of test period ($p = 0.0001$) such that a progressive decline of HR toward and below pre-exercise level was observed during the first 5-min of recovery. Thereafter, post-exercise HR remained close to pre-exercise levels. There was also a significant main effect of age ($p = 0.011$) and exercise condition ($p = 0.03$) such that a smaller post-exercise delta HR was observed among the older women, and following the 5-RM exercise condition (Figure B.9).

![Figure B.9](image)

**Figure B.9  Post-exercise Heart Rate Changes After RE by Age and Exercise Condition in Women**

Magnitude of HR drop below pre-exercise level following resistance exercise (RE). Values are mean ± SE.

The results revealed a significant main effect of test period ($p = 0.0001$) such that a progressive decline of HR toward and below pre-exercise level was observed during the first 5-min of recovery. Thereafter, post-exercise HR remained close to pre-exercise levels (see Figure B.9). There was a significant main effect of age ($p = 0.011$) and
exercise condition \((p = 0.03)\) such that a smaller post-exercise delta HR was observed among the older women, and following the 5-RM exercise condition (see Figure B.9).

**B.5. Indicator of Autonomic Function**

B.5.1. **Autonomic Indicators from 5-min segments.** Indicators of autonomic function (i.e., R-R interval, SDNN, pnn50, LFnu, and LF/HF ratio) derived from 5-minute segments before and following RE were evaluated using a 2x2 (exercise condition x age group) mixed model ANOVA with repeated measures on time (pre-exercise, Rec0-5, Rec1-6, Rec2-7, Rec3-8, Rec4-9, and Rec5-10) (Figure B.10 through B.14).

The results of R-R interval changes following RE revealed a significant main effect of test period \((p = 0.001)\) such that R-R interval at Rec0-5 was smaller than RR interval at pre-exercise and all other recovery periods. Further, there was a significant main effect of age group \((p = 0.001)\) suggesting a larger R-R interval among the older women in comparison to their young counterparts (Figure B.10).

![Figure B.10 R-R Interval (5-minute Segments) Before and Following RE by Age and Exercise Condition in Women](image)

163
Similarly, the results of SDNN changes following RE also revealed a significant main effect of test period ($p = 0.0001$) such that SDNN at: 1.) Rec0-5 was larger than pre-exercise and all other recovery periods; 2.) Rec1-6 was smaller than Rec0-5, but larger than all other recovery periods; 3.) Rec2-7 was smaller than Rec0-5 and Rec1-6, but larger than all other recovery periods; 4.) Rec3-8 was smaller than Rec0-5, Rec1-6, and Rec2-7, but larger than Rec4-9 and Rec5-10; and 5.) Rec4-9 and Rec5-10 were smaller than Rec0-5, Rec1-6, Rec2-7, and Rec3-8. Besides, there was a main effect of age group ($p = 0.0001$) such that a smaller SDNN among the older women as compared to their young counterpart (Figure B.11).

![Figure B.11 SDNN (5-minute Segments) Before and Following RE by Age and Exercise Condition in Women](image)

The results of pnn50 changes following RE revealed a significant main effect of test period ($p = 0.0001$) such that: 1.) pre-exercise, Rec4-9, and Rec5-10 were smaller than Rec0-5, Rec1-6, Rec2-7, and Rec3-8; 2.) Rec1-6 and Rec2-7 were higher than Rec3-8, and 3.) Rec4-9 was higher than Rec5-10, but similar to pre-exercise value. There was also a significant main effect of age group such that a smaller pnn50 was observed among
the older women in comparison to their younger counterparts \((p = 0.0001)\) (Figure B.12).

The results of LFnu changes following RE revealed a main effect of test period \((p = 0.001)\) such that LFnu at: 1.) pre-exercise was higher than Rec1-6, Rec2-7, and Rec3-8; 2.) Rec0-5 was higher than Rec1-6, Rec2-7, Rec3-8, and Rec4-9; 3.) Rec1-6 was lower than Rec5-10; 4.) Rec2-7 and Rec3-8 were lower than Rec4-9 and Rec5-10; and 5.) Rec4-9 was lower than Rec5-10 (Figure B.13).

Figure B.12 PNN50 (5-minute Segments) Before and Following RE by Age and Exercise Condition in Women

Figure B.13 LFnu (5-minute Segments) Before and Following RE by Age and Exercise Condition in Women
The results of LF/HF ratio changes following RE revealed a main effect of test period ($p = 0.017$) such that pre-exercise and Rec0-5 were higher than Rec1-6, Rec2-7, and Rec3-8. Further, Rec5-10 was higher than Rec2-7, and Rec3-8. (Figure B.14).

![Figure B.14 LF/HF Ratio (5-minute Segments) Before and Following RE by Age and Exercise Condition in Women](image)

**Figure B.14 LF/HF Ratio (5-minute Segments) Before and Following RE by Age and Exercise Condition in Women**

B.5.2. Autonomic Indicators from 10-min segments. Indicators of autonomic function derived from 10-minute segments before and after RE were evaluated using a 2x2 mixed model ANOVA with repeated measures on time (pre-exercise, Rec0-10, Rec10-20, Rec20-30, Rec30-40, Rec40-50, and Rec50-60) (Figure B.15 through B.19).

The results of R-R interval changes following RE revealed a significant main effect of test period ($p = 0.001$) such that R-R interval at Rec0-10 was smaller than RR interval at pre-exercise and all other recovery periods. Further, there was a significant main effect of age group ($p = 0.001$) suggesting a larger R-R interval among the older women in comparison to their young counterparts (Figure B.15).
Figure B.15 R-R Interval (10-minute Segments) Before and Following RE by Age and Exercise Condition in Women

The results of SDNN changes following RE revealed a significant main effect of test period ($p = 0.0001$) such that SDNN at: 1.) Rec0-10 was larger than pre-exercise and all other recovery periods; 2.) Rec10-20 was smaller than pre-exercise, Rec0-10, Rec20-30, Rec30-40, and Rec50-60; and 3.) Rec20-30 was smaller than pre-exercise, and Rec0-10, and Rec50-60, but higher than Rec10-20. Besides, there was a main effect of age group ($p = 0.0001$) such that a smaller SDNN among the older women as compared to their young counterpart (Figure B.16).

The results of pnn50 changes following RE revealed a significant main effect of test period ($p = 0.02$) such that Rec0-10 was larger than pre-exercise and all recovery periods. There was also a significant main effect of age group such that a smaller pnn50 was observed among the older women in comparison to their younger counterparts ($p = 0.0001$) (Figure B.17).
Figure B.16 SDNN (10-minute Segments) Before and Following RE by Age & Exercise Condition in Women

Figure B.17 PNN50 (10-minute Segments) Before and Following RE by Age and Exercise Condition in Women
The results of LFnu changes after RE revealed a main effect of test period \( (p = 0.007) \) such that LFnu at Rec40-50 and Rec50-60 were higher than pre-exercise level. There was also a significant test condition by age group interaction effect \( (p = 0.03) \) and test condition by exercise condition interaction effect \( (p = 0.02) \) indicating that recovery of sympathetic modulation of the heart period above pre-exercise level after exercise was earlier following the 15-RM condition (~20 minutes) and among the young group (~20 minutes) as compared to the 5-RM condition (~50 minutes) and among their older adult counterparts (~40 minutes) (Figure B.18). However, the results of LF/HF ratio revealed only a main effect of test condition such that Rec0-10 was higher than pre-exercise and all recovery periods (Figure B.19).

![Figure B.18 LFNN (10-minute Segments) Before and Following RE by Age & Exercise Condition in Women](image-url)
B.6. Vascular Function Indices

B.6.1 Reliability of FBF and FVR readings. Within session and inter-sessions reliability for resting forearm blood inflow (FBF) and forearm vascular resistance (FVR) indices were derived for each age group using intraclass correlation coefficients. The results of within session reliability (before occlusion, and before both 5-RM and 15-RM exercise conditions) for resting forearm blood inflow (FBF\textsubscript{rest}) and vascular resistance (FVR\textsubscript{rest}) revealed ranges of ICCs for resting FBF from 0.71 to 0.81 among older women, and from 0.63 to 0.95 among their young counterparts; and ranges of ICCs for resting FVR from 0.73 to 0.79 among adult women, and from 0.63 to 0.92 among the young women (Table B.20).
Table B.20  Within Session Reliability For FBF and FVR

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th></th>
<th>Old</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>ICC</td>
<td>1</td>
</tr>
<tr>
<td>Before Occlusion</td>
<td>FBF</td>
<td>2.05±1.09</td>
<td>1.69±0.58</td>
<td>0.63</td>
</tr>
<tr>
<td></td>
<td>FVR</td>
<td>44.8±20.1</td>
<td>48.4±14.5</td>
<td>0.70</td>
</tr>
<tr>
<td>Before 5-RM</td>
<td>FBF</td>
<td>1.96±0.41</td>
<td>1.89±0.42</td>
<td>0.80</td>
</tr>
<tr>
<td></td>
<td>FVR</td>
<td>37.6±7.42</td>
<td>40.1±6.87</td>
<td>0.63</td>
</tr>
<tr>
<td>Before 15-RM</td>
<td>FBF</td>
<td>1.96±0.58</td>
<td>1.90±0.59</td>
<td>0.95</td>
</tr>
<tr>
<td></td>
<td>FVR</td>
<td>37.4±9.85</td>
<td>40.8±10.8</td>
<td>0.92</td>
</tr>
</tbody>
</table>

Values are mean ± SE. FBF = Forearm blood inflow; FVR = forearm vascular resistance; and ICC = intraclass correlation coefficient.

On the other hand, the results of inter-sessions reliability (before occlusion, and before 5-RM and 15-RM exercise conditions) for $\text{FBF}_{\text{rest}}$ and $\text{FVR}_{\text{rest}}$ revealed low ICCs for resting forearm blood inflow and forearm vascular resistance among the older women, and modest ICCs for resting forearm blood inflow and forearm vascular resistance among the young women (Table B.21).

Table B.21  Inter-Sessions Reliability (Three different days) for FBF and FVR Within Each Age Group

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th></th>
<th>Old</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-Occ.</td>
<td>Pre-5RM</td>
<td>Pre-15RM</td>
<td>ICC</td>
</tr>
<tr>
<td>FBF</td>
<td>1.8±0.7</td>
<td>1.9±0.4</td>
<td>1.9±0.6</td>
<td>0.69</td>
</tr>
<tr>
<td>FVR</td>
<td>45.0±14.7</td>
<td>38.7±6.4</td>
<td>39.1±10.1</td>
<td>0.63</td>
</tr>
</tbody>
</table>

Values are mean ± SE. Before-occ = Before-Occlusion. FBF = Forearm blood inflow; FVR = forearm vascular resistance. ICC = intraclass correlation coefficient.
Additionally, given that the participants assumed a supine position during the occlusion testing day and a seated position during the exercising testing days, inter-sessions reliability for resting forearm blood inflow and vascular resistance only during both exercise condition days were determined within each age group. The results revealed low ICCs for resting forearm blood inflow within both age groups, and modest ICCs within both age groups for resting forearm vascular resistance (Table B.22). As a result, only repeated measures assessments to examine the effect of test condition, exercise condition, and age group on FVR were performed.

Table B.22  Inter-Sessions Reliability (Two Different Days) For FBF and FVR

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th></th>
<th>Old</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5-RM</td>
<td>15-RM</td>
<td>ICC</td>
<td>5-RM</td>
</tr>
<tr>
<td>FBF rest</td>
<td>1.93±0.39</td>
<td>1.93±0.57</td>
<td>0.40</td>
<td>2.12±0.63</td>
</tr>
<tr>
<td>FVF rest</td>
<td>38.3±6.52</td>
<td>39.0±9.92</td>
<td>0.62</td>
<td>38.8±9.52</td>
</tr>
</tbody>
</table>

Values are mean ± SE. FBF = Forearm blood flow; FVR = forearm vascular resistance; and ICC = intraclass correlation coefficient.
B.6.2. **Repeated Measures Assessments of FVR.** Vascular function indices in the arterial side (i.e., forearm blood inflow and forearm vascular resistance) were evaluated before and for 1 hour after RE using a 2x2 (exercise condition x age group) mixed model ANOVA with repeated measures on time (pre-exercise, Rec4’, Rec7’, Rec10’, Rec15’, Rec20’, Rec30’, Rec40’, Rec50’, and Rec60’). The results revealed an age group by exercise condition interaction effect on forearm vascular resistance such that young women exhibited an increase in FVR following RE, but not among the older women (See Figure B.20, & Table B.23).

**Figure B.20 Age Group by Exercise Condition by Test Condition Interaction Effect on FVR Before & For 1 Hour After RE in Women**
Values are mean ± SD. Forearm vascular resistance (FVR) before (Pre-) and for 1 hour after RE. † / † = 5- / 15-RM older group values diff from pre-exercise ($p < 0.05$). $ / # = 5- / 15-RM young group values diff from pre-exercise ($p \leq 0.05$).
## Table B.23  Age Group by Exercise Condition by Test Condition Interaction

**Effect on Forearm Vascular Resistance Before and For 1 Hour After RE in Women**

<table>
<thead>
<tr>
<th></th>
<th>FBF (mL/100mL/min)</th>
<th>FVR (Units)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Young</td>
<td>Old</td>
</tr>
<tr>
<td>Pre-05</td>
<td>1.90 ± 0.35</td>
<td>2.01 ± 0.61</td>
</tr>
<tr>
<td>Pre-15</td>
<td>1.85 ± 0.46</td>
<td>1.97 ± 0.73</td>
</tr>
<tr>
<td>R4’-05</td>
<td>1.70 ± 0.63</td>
<td>1.80 ± 0.50</td>
</tr>
<tr>
<td>R4’-15</td>
<td>1.39 ± 0.45</td>
<td>2.11 ± 0.83</td>
</tr>
<tr>
<td>R7’-05</td>
<td>1.85 ± 0.95</td>
<td>1.81 ± 0.61</td>
</tr>
<tr>
<td>R7’-15</td>
<td>1.67 ± 1.05</td>
<td>2.05 ± 0.55</td>
</tr>
<tr>
<td>R10’-05</td>
<td>1.52 ± 0.52</td>
<td>1.99 ± 0.56</td>
</tr>
<tr>
<td>R10’-15</td>
<td>1.46 ± 0.49</td>
<td>1.67 ± 0.39</td>
</tr>
<tr>
<td>R15’-05</td>
<td>1.48 ± 0.53</td>
<td>1.85 ± 0.82</td>
</tr>
<tr>
<td>R15’-15</td>
<td>1.51 ± 0.77</td>
<td>1.79 ± 0.49</td>
</tr>
<tr>
<td>R20’-05</td>
<td>1.55 ± 0.65</td>
<td>1.61 ± 0.53</td>
</tr>
<tr>
<td>R20’-15</td>
<td>1.59 ± 0.77</td>
<td>1.86 ± 0.56</td>
</tr>
<tr>
<td>R30’-05</td>
<td>1.38 ± 0.43</td>
<td>1.78 ± 0.60</td>
</tr>
<tr>
<td>R30’-15</td>
<td>1.82 ± 0.89</td>
<td>2.32 ± 0.72</td>
</tr>
<tr>
<td>R40’-05</td>
<td>1.60 ± 0.65</td>
<td>1.57 ± 0.52</td>
</tr>
<tr>
<td>R40’-15</td>
<td>1.80 ± 0.65</td>
<td>1.68 ± 0.45</td>
</tr>
<tr>
<td>R50’-05</td>
<td>1.43 ± 0.56</td>
<td>1.71 ± 0.50</td>
</tr>
<tr>
<td>R50’-15</td>
<td>1.50 ± 0.65</td>
<td>1.74 ± 0.45</td>
</tr>
<tr>
<td>R60’-05</td>
<td>1.59 ± 0.67</td>
<td>1.51 ± 0.48</td>
</tr>
<tr>
<td>R60’-15</td>
<td>1.41 ± 0.72</td>
<td>1.70 ± 0.34</td>
</tr>
</tbody>
</table>

Values are mean ± SD. FBF = Forearm blood inflow (FVR) and forearm vascular resistance (FVR) before (Pre-) and for 1-hour after RE. † / ‡ = 5- / 15-RM older group values diff from pre-exercise ($p < 0.05$). $ / # = 5- / 15-RM young group values diff from pre-exercise ($p < 0.05$).

## B.7  Bivariate Correlation Between Indicators of Autonomic Function and SBP at Rest and Following RE within each Age Group.

Bivariate correlations were used to evaluate the relationship between indicators of autonomic function (SDNN, pnn50, LFnu, LF/HF ratio) and arterial blood pressure (SBP, and DBP) before, immediately after, (local min, local max, magnitude of drop, magnitude of recovery, mean rate of decline and mean rate of recovery) and for 1 hour after RE by age and exercise condition.
**Young and Older Adult Group Data.** The results pooling both group data revealed a significant correlation between SDNN at rest and mean pre-exercise SBP (Figure B.21), and pnn50 and mean pre-exercise SBP such that those participants with smaller SDNN and pnn50 at rest (mostly older women) exhibited a higher mean pre-exercise SBP (Table B.24).

![Figure B.21 Relationships Between Resting SDNN and Mean Pre-exercise SBP](image)

**Table B.24 Bivariate Correlation Between Indicators of Autonomic Function and SBP Before and After RE Pooling Young and Old Group Data**

<table>
<thead>
<tr>
<th>SBP</th>
<th>SDNN</th>
<th>Pnn50</th>
<th>LFnu.rest</th>
<th>LF/HF.rest</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Rest</td>
<td>Rest</td>
<td>Rec0-5</td>
</tr>
<tr>
<td>Mean pre-exercise</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magnitude of recovery</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean rate of recovery</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

|                       |      |       |           |            |         |        |
| Mean pre-exercise     | r = -0.47 * | r = -0.37 * |
| Magnitude of recovery | r = 0.40 * | r = 0.36 * | r = 0.29 * | r = 0.28 * |
| Mean rate of recovery | r = 0.46 * | r = 0.44 * | r = 0.38 * | r = 0.36 * |

* = \( p \leq 0.05 \).

Additionally, the results revealed a significant correlation between LFnu at rest and the magnitude of SBP recovery \( (r = 0.40, p = 0.003) \), LFnu at rest and the mean rate of
SBP recovery ($r = 0.46, p = 0.0001$), LF/HF ratio at rest and the magnitude of SBP recovery ($r = 0.29, p = 0.03$), and LF/HF ratio at rest and the mean rate of SBP recovery ($r = 0.38, p = 0.004$) (see Figure B.22 for resting LFnu and mean rate of SBP recovery). Such relationships suggest that participants with higher LFnu and LF/HF ratio at rest (regardless the age) exhibited a larger magnitude of SBP recovery and faster mean rate of SBP recovery from local min to local max after RE (Figure B.22).

**Figure B.22 Relationships Between LFnu at Rest and Mean Rate of SBP Recovery After RE in Young and Older Adult Women**

**Older Adult Group.** The results within this age group revealed a significant correlation between LFnu at rest and mean rate of SBP recovery ($p = 0.001$) (Figure B.23), LFnu at rest and magnitude of SBP recovery ($p = 0.005$), LF/HF ratio at rest and mean rate of SBP recovery ($p = 0.008$), and LF/HF ratio at rest and magnitude of SBP recovery ($p = 0.02$). Such relationships suggest that old participants with higher resting LFnu and LF/HF ratio exhibited after RE a faster mean rate SBP recovery and a larger magnitude of SBP recovery (regardless the exercise condition) from local min to local max. On the contrary, there was not significant correlation between LFnu at rest and mean pre-exercise SBP. However, there was a trend towards a relationship between
LF/HF ratio at rest and mean pre-exercise SBP suggesting that older adult participants with higher LF/HF ratio at rest exhibited lower pre-exercise SBP (Table B.25).

Figure B.23 Relationship Between LFnu at Rest and Mean Rate of SBP Recovery After RE in Older Adult Women

The results revealed also a significant correlation between LFnu at Rec0-5 and mean rate of SBP recovery \( (p = 0.006) \), and LFnu at Rec0-5 and magnitude of SBP recovery \( (p = 0.03) \) such that older adult participants with higher LFnu at Rec0-5 (regardless the exercise condition) exhibited after RE a faster mean rate of SBP recovery, and larger magnitude of SBP recovery from local min to local max (Table B.25, and Figure B.24 for LFnu at Rec 0-5 and Mean Rate of SBP Recovery after RE).
Figure B.24 Relationship Between LFnu at Rec 0-5 and Mean Rate of SBP Recovery After RE in Older Adult Women

Table B.25 Bivariate Correlation Between Indicators of Autonomic Function & SBP Before and After RE in Older Adult Women

<table>
<thead>
<tr>
<th></th>
<th>LFnu</th>
<th>LF/HF ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Rec0-5</td>
</tr>
<tr>
<td>Magnitude of Recovery (mmHg/sec)</td>
<td>r = 0.54 *</td>
<td>r = 0.44 *</td>
</tr>
<tr>
<td>Mean Rate of Recovery (mmHg)</td>
<td>r = 0.59 *</td>
<td>r = 0.53 *</td>
</tr>
</tbody>
</table>

* = significant correlation (p < 0.05). + = trend towards a relationship (p > 0.05).

In addition, there was a significant correlation between LF/HF ratio at Rec0-5 and the mean rate of SBP recovery (p = 0.05) such that the old participants with higher LF/HF ratio at Rec0-5 exhibited a faster mean rate of SBP recovery from local min to local max after RE. There was also a trend toward a relationship between LF/HF ratio at Rec0-5 and magnitude of SBP recovery (p = 0.011) suggesting that older participants with higher LF/HF ratio at Rec0-5 exhibited a larger magnitude of SBP recovery from local min to local max after RE (Table B.25).
Young Adult Group. The results of pre-exercise data within the young group revealed a significant correlation between LFnu at rest and mean pre-exercise SBP ($p = 0.04$) such that young participants with higher LFnu at rest exhibited a higher mean pre-exercise SBP. There was also a trend towards a relationship between LFnu at rest and mean rate of SBP recovery ($p = 0.10$) suggesting that young participants with higher LFnu at rest exhibited a faster mean rate of SBP recovery from local min to local max after RE. Further, the results revealed a significant correlation between LFnu at Rec0-5 and mean rate of SBP recovery ($p = 0.006$) (Figure B.25), and LFnu at Rec0-5 and magnitude of SBP recovery ($p = 0.03$) such that young participants with higher LFnu at Rec0-5 exhibited a faster mean rate of SBP recovery and a larger magnitude of SBP recovery from local min to local max after RE (Table B.26).

![Figure B.25 Relationship Between LFnu at Recovery 0-5 and Mean Rate of SBP Recovery after RE in Young Women](image)

The results revealed also a significant correlation between LF/HF ratio at Rec 0-5 and the mean rate of SBP recovery ($p = 0.02$) such that young participants with higher LF/HF
ratio exhibited a faster mean rate of SBP from local min to local max after RE. There was also a trend towards a relationship between LF/HF ratio at Rec0-5 and magnitude of SBP recovery suggesting that young participants with higher LF/HF ratio at Rec 0-5 exhibited a larger magnitude of SBP recovery from local min to local max after RE (Table B.26).

<table>
<thead>
<tr>
<th></th>
<th>LFnu</th>
<th>LF/HF ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Rec0-5</td>
</tr>
<tr>
<td>Mean Pre-exercise SBP (mmHg)</td>
<td>r = 0.37 *</td>
<td></td>
</tr>
<tr>
<td>Magnitude of Recovery (mmHg/sec)</td>
<td>r = 0.40 *</td>
<td>r = 0.32 +</td>
</tr>
<tr>
<td>Mean Rate of Recovery (mmHg)</td>
<td>r = 0.31 +</td>
<td>r = 0.50 *</td>
</tr>
</tbody>
</table>

* = significant correlation (p ≤ 0.05). + = trend towards a relationship (p <0.15)

**Young and Older Adult Group by Exercise Condition.** The results pooling both age group data by exercise condition revealed the same significant relationship at rest and Rec 0-5 between autonomic indicators (LFnu and LH/HF ratio) and post-exercise SBP readings (mean rate of SBP recovery and magnitude of SBP recovery from local min to local max) regardless the age and exercise condition (Table B.27). Such relationships suggest that participants with higher LFnu and LF/HF ratio at rest and Rec 0-5 exhibited a faster mean rate of SBP recovery and a larger magnitude of SBP recovery from local min to local max after both exercise conditions (Table B.27).
Table B.27  Bivariate Correlation Between Indicators of Autonomic Function and SBP Before and After RE by Exercise Condition

<table>
<thead>
<tr>
<th>Autonomic Indicators</th>
<th>Mean Rate of SBP Recovery</th>
<th>Magnitude of SBP Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>LFnu at rest (5-RM)</td>
<td>r = 0.49 *</td>
<td>r = 0.34 +</td>
</tr>
<tr>
<td>LFnu at rest (15-RM)</td>
<td>r = 0.42 *</td>
<td>r = 0.42 *</td>
</tr>
<tr>
<td>LFnu at Rec 0-5 (5-RM)</td>
<td>r = 0.46 *</td>
<td>r = 0.27 +</td>
</tr>
<tr>
<td>LFnu at Rec 0-5 (15-RM)</td>
<td>r = 0.42 *</td>
<td>r = 0.42 *</td>
</tr>
<tr>
<td>LF/HF ratio at rest (5-RM)</td>
<td>r = 0.39 *</td>
<td>r = 0.26 +</td>
</tr>
<tr>
<td>LF/HF ratio at rest (15-RM)</td>
<td>r = 0.37 *</td>
<td>r = 0.32 +</td>
</tr>
<tr>
<td>LF/HF ratio at Rec 0-5 (5-RM)</td>
<td>r = 0.38 *</td>
<td>r = 0.39 *</td>
</tr>
<tr>
<td>LF/HF ratio at Rec 0-5 (15-RM)</td>
<td>r = 0.41 *</td>
<td></td>
</tr>
</tbody>
</table>

* = significant correlation (p < 0.05). + = trend towards a relationship (p >0.05)

B.7  Bivariate Correlation Between Vascular Function Indices and Arterial Blood Pressure (SBP and DBP) Before and After RE by Age and Exercise Condition

Bivariate correlations were used to evaluate the relationship between vascular function indices (\(\text{FBF}_{\text{rest}}, \text{FVC}_{\text{rest}}, \text{FVO}_{\text{rest}}, \text{FBF}_{\text{occ}}, \text{FVC}_{\text{occ}}, \text{and FVO}_{\text{occ}}\)) and arterial blood pressure (SBP and DBP) before, during, and after RE conditions (mean pre-exercise BP, peak BP, local min, local max, magnitude of drop, magnitude of recovery, mean rate of decline, mean rate of recovery, post-exercise BP, and magnitude of post-exercise BP drop) within each age group.

**Older Adult Group - Vascular Function Indices and SBP Response.** The results within this age group (looking first the arterial side) revealed a significant correlation between resting forearm blood inflow (\(\text{FBF}_{\text{rest}}\)) and 1-min post-exercise SBP/peak ratio (\(p = 0.04\)) such that older adult participants with higher \(\text{FBF}_{\text{rest}}\) exhibited a smaller 1-min post-exercise SBP/peak ratio after RE (Figure B.26, & Table B.28). Further, there was a trend towards a relationship between resting forearm blood flow and 3-min post-exercise...
SBP/peak ratio ($p = 0.18$) suggesting that older adult participants with higher FBF$_{\text{rest}}$ exhibited a smaller 3-min post-exercise SBP/peak ratio following RE.

![Graph showing the relationship between resting forearm blood inflow and 1’ SBP/Peak Ratio after RE in Older Women by Exercise Condition](image)

**Figure B.26** Relationship Between Resting Forearm Blood Inflow and 1’ SBP/Peak Ratio after RE in Older Women by Exercise Condition

On the other hand (looking the venous side), the results revealed a trend towards a nearly significant correlation between forearm venous capacitance at rest (FVC$_{\text{rest}}$) and 3-min post-exercise SBP/peak ratio ($p = 0.052$), and between forearm venous capacitance following arterial occlusion (FVC$_{\text{occ}}$) and 3-min post-exercise SBP/peak ratio ($p = 0.10$) suggesting that older participants with larger FVC at rest and following arterial occlusion exhibited smaller 3-min post-exercise SBP/peak ratio (Table B.28).

Additionally, the results within the older adult group revealed a relationship between resting forearm venous outflow (FVO$_{\text{rest}}$) and magnitude of post-exercise SBP drop below pre-exercise level (post-exercise SBP minus pre-exercise SBP) at different time points, which reached statistical significance after 10’ of recovery (mostly at all testing time points), such as R10’ ($p = 0.07$), R20’ ($p = 0.03$), R30’ ($p = 0.08$), R40’ ($p = 0.05$), and R60’ ($p = 0.03$). There was also a similar relationship between forearm venous
outflow following arterial occlusion (FVO_{occ}) and magnitudes of post-exercise SBP drop below pre-exercise level (see Figure B.27 for the relationship between FVO_{occ} and 20’ post-exercise SBP), which reach statistical significance after 10’ of recovery, such as at R10’ (p = 0.13), R20’ (p = 0.01), R30’ (p = 0.02), R40’ (p = 0.09), R50’ (p = 0.08), and R60’ (p = 0.12). Such relationship would suggest that older adult participants with larger FVO at both rest and following arterial occlusion exhibited a larger magnitude of SBP drop below pre-exercise level after RE (Table B.28).

![Figure B.27 Relationship Between Forearm Venous Outflow Following Arterial Occlusion and 20’ Post-exercise SBP After RE in Older Women](image)

(Figure B.27 Relationship Between Forearm Venous Outflow Following Arterial Occlusion and 20’ Post-exercise SBP After RE in Older Women)
<table>
<thead>
<tr>
<th></th>
<th>FBF (Rest)</th>
<th>FBF (Occlusion)</th>
<th>FVC (Rest)</th>
<th>FVC (Occlusion)</th>
<th>FVO (Rest)</th>
<th>FVO (Occlusion)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1' SBP/peak ratio</td>
<td>r = -0.38 *</td>
<td></td>
<td>r = -0.36 +</td>
<td></td>
<td>r = 0.34 +</td>
<td></td>
</tr>
<tr>
<td>3' SBP/peak ratio</td>
<td>r = -0.25 +</td>
<td>r = -0.36 +</td>
<td>r = -0.31 +</td>
<td></td>
<td>r = 0.29 +</td>
<td></td>
</tr>
<tr>
<td>10' PE SBP drop</td>
<td>r = 0.34 +</td>
<td></td>
<td>r = 0.29 +</td>
<td></td>
<td>r = 0.34 +</td>
<td></td>
</tr>
<tr>
<td>15' PE SBP drop</td>
<td>r = 0.40 *</td>
<td>r = 0.46 *</td>
<td></td>
<td>r = 0.34 +</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20' PE SBP drop</td>
<td>r = 0.33 +</td>
<td>r = 0.43 *</td>
<td></td>
<td>r = 0.33 +</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30' PE SBP drop</td>
<td>r = 0.36 *</td>
<td>r = 0.33 +</td>
<td></td>
<td>r = 0.34 +</td>
<td></td>
<td></td>
</tr>
<tr>
<td>40' PE SBP drop</td>
<td>r = 0.42 *</td>
<td>r = 0.30 +</td>
<td></td>
<td>r = 0.34 +</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50' PE SBP drop</td>
<td>r = 0.36 *</td>
<td>r = 0.33 +</td>
<td></td>
<td>r = 0.34 +</td>
<td></td>
<td></td>
</tr>
<tr>
<td>60' PE SBP drop</td>
<td>r = 0.42 *</td>
<td>r = 0.30 +</td>
<td></td>
<td>r = 0.34 +</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = significant correlation (p < 0.05). + = trend towards a relationship (p > 0.05)

**Young Adult Group - Vascular function indices and SBP response.** The results within this age group revealed: 1.) a significant correlation between resting forearm venous outflow (FVO_{rest}) and peak SBP (p = 0.016), FVO_{rest} and local min (p = 0.01); and FVO_{rest} and local max (p = 0.03) such that young participants with higher FVO_{rest} exhibited after RE higher SBP at peak exercise, local min, and local max; 2.) a significant correlation between forearm venous capacitance following arterial occlusion (FVC_{occ}) and magnitude of SBP decline (p = 0.03) such that young participants with higher FVC_{occ} exhibited a larger magnitude of SBP drop after RE; and 3.) a significant correlation between forearm venous outflow following arterial occlusion (FVO_{occ}) and mean rate of SBP recovery (p = 0.03) (see Figure B.28), and FVO_{occ} and magnitude of SBP recovery (p = 0.04) such that young participants with larger FVO_{occ} exhibited a slower mean rate of SBP recovery and a smaller magnitude of SBP recovery from local min to local max.
Additionally, the results revealed a relationship between resting forearm venous outflow (FVO\textsubscript{rest}) and absolute values of SBP following RE, which reached either nearly a statistical significance or statistical significance at different time points during the recovery period, such as: at R1′ (\( p = 0.03 \)), R3′ (\( p = 0.11 \)), R5′ (\( p = 0.07 \)), R10′ (\( p = 0.03 \)), R15′ (\( p = 0.01 \)), R20′ (\( p = 0.05 \)), R30′ (\( p = 0.08 \)), R40′ (\( p = 0.04 \)), and R50′ (\( p = 0.046 \)) (Table B.29, and Figure B.29) . Such relationship would suggest that young participants with higher FVO\textsubscript{rest} exhibited a higher SBP following RE. However, in contrast to older adult women, the results within young adult group revealed no relationship between forearm venous outflow (at rest and after arterial occlusion) and magnitude of SBP drop below pre-exercise level after RE (see Figure B.29 for the relationship between resting FVO and 15′ SBP values following RE, and Table B.29).
Table B.29  Bivariate Correlation Between Vascular Function Indices and SBP Before and After RE in Young Adult Women

<table>
<thead>
<tr>
<th></th>
<th>FVC</th>
<th>FVO</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Occlusion</td>
</tr>
<tr>
<td>Peak SBP</td>
<td>r = 0.35 +</td>
<td>r = 0.50 *</td>
</tr>
<tr>
<td>Local min</td>
<td></td>
<td>r = 0.51 *</td>
</tr>
<tr>
<td>Local max</td>
<td></td>
<td>r = 0.45 *</td>
</tr>
<tr>
<td>Magnitude of SBP increase</td>
<td>r = 0.34 +</td>
<td></td>
</tr>
<tr>
<td>Magnitude of SBP Drop</td>
<td>r = 0.40 *</td>
<td>r = 0.39 +</td>
</tr>
<tr>
<td>1’ SBP/peak ratio</td>
<td>r = -0.36 +</td>
<td></td>
</tr>
<tr>
<td>3’ SBP/peak ratio</td>
<td>r = -0.35 +</td>
<td></td>
</tr>
<tr>
<td>Mean rate of SBP Recovery</td>
<td></td>
<td>r = -0.43 *</td>
</tr>
<tr>
<td>Magnitude of SBP Recovery</td>
<td></td>
<td>r = -0.45 *</td>
</tr>
<tr>
<td>1’ Post-exercise SBP</td>
<td>r = 0.46 *</td>
<td></td>
</tr>
<tr>
<td>3’ Post-exercise SBP</td>
<td>r = 0.34 +</td>
<td></td>
</tr>
<tr>
<td>5’ Post-exercise SBP</td>
<td>r = 0.39 +</td>
<td></td>
</tr>
<tr>
<td>10’ Post-exercise SBP</td>
<td>r = 0.45 *</td>
<td></td>
</tr>
<tr>
<td>15’ Post-exercise SBP</td>
<td>r = 0.53 *</td>
<td></td>
</tr>
<tr>
<td>20’ Post-exercise SBP</td>
<td>r = 0.41 *</td>
<td></td>
</tr>
<tr>
<td>30’ Post-exercise SBP</td>
<td>r = 0.35 +</td>
<td>r = 0.38 +</td>
</tr>
<tr>
<td>40’ Post-exercise SBP</td>
<td>r = 0.39 *</td>
<td>r = 0.32 +</td>
</tr>
<tr>
<td>50’ Post-exercise SBP</td>
<td>r = 0.34 +</td>
<td>r = 0.36 *</td>
</tr>
</tbody>
</table>

* = significant correlation ($p < 0.05$). + = trend towards a relationship ($p > 0.05$)
Older Adult Group-Vascular Function Indices and DBP Response. The results of bivariate correlation between vascular function indices and DBP response before, during, after RE within the older adult group revealed (at the arterial side) a significant correlation between resting forearm blood inflow (FBF$_{\text{rest}}$) and mean rate of DBP decline ($p = 0.02$), and FBF$_{\text{rest}}$ and mean rate of DBP recovery ($p = 0.006$) such that older adult participants with higher FBF$_{\text{rest}}$ exhibited a faster mean rate of DBP decline from peak exercise to local min, and a faster mean rate of DBP recovery (Figure B.30) from local min to local max after both RE conditions. Further, there was a trend towards a relationship between forearm blood inflow (at rest and following arterial occlusion) and magnitude of DBP decline ($p = 0.32$, and $p = 0.26$, respectively) suggesting that older adult participants with higher forearm blood inflow at rest and following arterial occlusion exhibited a larger magnitude of DBP decline from peak exercise to local min after both RE conditions. There was also a trend towards a relationship between resting forearm inflow (FBF$_{\text{rest}}$) and DBP local min ($p = 0.16$) suggesting that older participants with higher (FBF$_{\text{rest}}$) exhibited lower diastolic local min after RE (Table B.30).

At the venous side, the results within this age group revealed a trend towards a relationship between: 1) forearm venous outflow at rest (FVO$_{\text{rest}}$) and DBP local min ($p = 0.15$) suggesting that older adult participants with larger FVO$_{\text{rest}}$ exhibited lower DBP local min following RE; 2.) forearm venous outflow at rest (FVO$_{\text{rest}}$) and DBP local max ($p = 0.11$), which would suggest that older adult participants with larger FVO$_{\text{rest}}$ exhibited a lower DBP local max after RE; 3.) forearm venous outflow following arterial occlusion (FVO$_{\text{occ}}$) and magnitude of DBP drop ($p = 0.16$), which would suggest that older adult participants with larger FVO$_{\text{occ}}$ exhibited a larger magnitude of DBP decline.
from peak exercise to local min after RE; and 4.) forearm venous capacitance at rest (FVC\textsubscript{rest}) and magnitude of DBP decline ($p = 0.06$) suggesting that older adult participants with larger FVC\textsubscript{rest} exhibited larger magnitudes of DBP drop from peak exercise to local min after both RE conditions (Table B.30).

$$(r = 0.51; p = 0.006)$$

**Figure B.30 Relationship Between Resting Forearm Blood Inflow and Mean Rate of DBP Recovery After RE in Older Adult Women**

**Table B.30  Bivariate Correlation Between Vascular Function Indices and DBP Before, during, after RE in Older Adult Women**

<table>
<thead>
<tr>
<th>DBP</th>
<th>FBF</th>
<th>FVC</th>
<th>FVO</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Occlusion</td>
<td>Rest</td>
</tr>
<tr>
<td>Local minimum</td>
<td>$r = -0.26$ +</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Local maximum</td>
<td></td>
<td>$r = -0.30$ +</td>
<td></td>
</tr>
<tr>
<td>Magnitude of decline</td>
<td>$r = 0.32$ +</td>
<td>$r = 0.26$ +</td>
<td>$r = 0.36$ +</td>
</tr>
<tr>
<td>Mean rate of decline</td>
<td>$r = 0.43$ *</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean rate of recovery</td>
<td>$r = 0.51$ *</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = significant correlation ($p < 0.05$). + = trend towards a relationship ($p > 0.05$)

**Young Adult Group-Vascular Function Indices and DBP Response.** The results of bivariate correlation between vascular indices and DBP response before, during and
after RE within this age group revealed 1) a significant relationship between resting forearm venous outflow (FVO_{rest}) with peak exercise \((p = 0.001)\), local min \((p = 0.05)\), local max \((p = 0.02)\), and magnitude of increase \((p = 0.03)\) such that young participants with higher FVO_{rest} exhibited a higher DBP reading at peak exercise, local min, and local max, and a larger magnitude of DBP increase; 2) a significant relation between forearm venous outflow following arterial occlusion (FVO_{occ}) with local min \((p = 0.04)\), and local max \((p = 0.02)\) such that young participants with higher FVO_{occ} exhibited higher local min and local max DBP after both RE; and 3) a trend towards a relationship between resting forearm venous outflow (FVO_{rest}) and the magnitude of DBP decline \((p = 0.02)\) suggesting that young participants with higher FVO_{rest} exhibited a larger magnitude of DBP decline after both RE conditions.
APPENDIX C. REVIEW: CARDIOVASCULAR ADAPTATIONS TO STRENGTH TRAINING IN OLDER ADULTS

TABLE OF CONTENTS

LIST OF TABLES .............................................................................................................................191

LIST OF FIGURES ..........................................................................................................................192

CHAPTER 1. INTRODUCCION ........................................................................................................193
  1.1 Demography and Health Cost of Age ..............................................................................195
  1.2 Disability in Age ..............................................................................................................198
  1.3 Research Design Issues in the Elderly ..........................................................................201
  1.4 Purpose of the Review .....................................................................................................203

CHAPTER 2. AGING OF THE CARDIOVASCULAR SYSTEM ...........................................205
  2.1 Changes on Cardiovascular System with Aging Process at Rest ................................205
    2.1.1 Central Function ......................................................................................................206
    2.1.2 Peripheral Function/Afterload ..............................................................................233
  2.2 Cardiovascular Performance during Aerobic and Anaerobic Work .........................259
    2.2.1 Acute Cardiovascular Responses to Aerobic Work ..............................................269
    2.2.2 Acute Cardiovascular Responses to Anaerobic Work .........................................308
  2.3 General Comments about Efficacy of Exercise Training .............................................320
    2.3.1 Chronic Cardiovascular Adaptations to Aerobic Exercise Training ...................321
    2.3.2 Chronic Cardiovascular Adaptations to Anaerobic Exercise Training ...............341

CHAPTER 3. STRENGTH TRAINING AND THE CARDIOVASCULAR SYSTEM IN THE ELDERLY .......................................................351
  3.1 List of Benefits of Strength Training ..............................................................................352
  3.2 Risks of Strength Training ..............................................................................................356
  3.3 CV benefits of strength training in the elderly ...............................................................361

CHAPTER 4. CURRENT TRENDS IN STRENGTH TRAINING PRESCRIPTION FOR THE ELDERLY .......................................................369
  4.1 Historical Evolution of Physical Activity Recommendations ...................................369
  4.2 Current Physical Activity Recommendations ............................................................373
  4.3 Exercise Prescription for Older Adults .........................................................................385

CONCLUSION ..................................................................................................................................390

REFERENCES ....................................................................................................................................392
# LIST OF TABLES

1. Structural Changes in the Heart due to Aging .................................................................207
2. Arterial Changes with Aging ..........................................................................................236
3. Physiological Adaptations to Resistance Exercise and Aerobic Training ..............349
4. Chronic Cardiovascular Adaptations to Resistance Exercise in Older Adults ....365
5. ACSM Position Stand, 1978 ..........................................................................................370
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Determinant Factors of Cardiac Output</td>
<td>214</td>
</tr>
<tr>
<td>2</td>
<td>Myocardial Inotropic Contractile Properties</td>
<td>228</td>
</tr>
<tr>
<td>3</td>
<td>Cardiac Output and Exercise Intensity Relationship</td>
<td>272</td>
</tr>
<tr>
<td>4</td>
<td>Heart Rate and Exercise Intensity Relationship</td>
<td>275</td>
</tr>
<tr>
<td>5</td>
<td>Stroke Volume and Exercise Intensity Relationship</td>
<td>276</td>
</tr>
<tr>
<td>6</td>
<td>Typical Blood Pressure Response to Dynamic Exercise</td>
<td>282</td>
</tr>
<tr>
<td>7</td>
<td>Oxygen Uptake and Age Relationship</td>
<td>287</td>
</tr>
</tbody>
</table>
CHAPTER 1. INTRODUCTION

The aging process has traditionally been viewed as a progressive decline in health and function, leading to disability and death. However, emerging views of the aging process differentiate between the decline in function due to biological aging from the decline in function associated with life-styles (e.g., physical inactivity, alcohol, smoking) and disease (e.g., coronary artery disease, cerebrovascular disease, hypertension, diabetes, cancer) (DiPrieto, & Seals, 1995; Spirduso, 1996). For example, studies hypothesizing that physical inactivity causes many of the functional losses attributed to aging have found that many of the physiological changes ascribed to aging (e.g., muscle atrophy, decreased endurance, and a loss of flexibility) are similar to those induced by enforced inactivity (i.e., bed-rest, and immobilization) or during prolonged space flight (Bloomfield, & Coyle, 1993; Bortz, 1982, Saltin et al., 1968; Shephard, 1993; Vorhies, & Riley, 1993). Therefore, even in healthy young people, serious muscle atrophy and marked declines in physiological functioning (sometimes equivalent to as much as 20 years worth of aging) have been observed following only a few weeks of immobility (Bloomfield, & Coyle, 1993; Bortz, 1982, Saltin et al., 1968; Shephard, 1993; Rikli, & Jones, 1997).

Similar results have been also reported in chronic disease patients, which implicate disease as a potential and important source of disability that may impair physical ability in a manner similar to disuse. For example, heart disease (Merz, & Forrester, 1997), cerebrovascular disease (Kiely et al., 1997), hypertension (Joint National Committee, 1993), diabetes (Clark, & Lee, 1995), osteoporosis (Cooper, 1997), arthritis (Fisher et al., 1991), visual impairment (Elliot et al., 1997), and dementia (Barberger-Gateau, &
Correspondingly, a number of research studies have provided evidence that physical activity, even if initiated late in life, is associated with improved physiological measures and functional performance, regardless of disease status (Cress et al., 1991; Fiatarone et al., 1990, 1994; Nichols, Hitzelberger, Sherman, & Patterson, 1995; Pyka, Linderberger, Charette, & Marcus, 1994; Rikli, & Edwards, 1991). In these studies, where the incidence of chronic conditions was similar for both exercising and control subjects, the exercising subjects demonstrated significantly higher levels of strength, endurance, and motor function compared to non-active subjects. This suggests that apparent age-related declines in function due to inactivity could be attenuated or possibly reversed by physical activity or structured exercise.

These results are also consistent with the notion that sedentary lifestyle contributes significantly to what has previously been considered the result of age-related functional decline. (Rikli, & Jones, 1997). Briefly, a general summation of the research in this area would suggest that: (1) physical activity can retard a decline in the determinants of physical ability (muscle strength, endurance, flexibility, neuromuscular coordination, and balance) in older adults, (2) increasing the level of physical activity might reverse some individuals' functional limitations, and (3) sustained physical activity throughout life could prevent the onset of some functional limitations (Carlson et al., 1999; Chiriboga et al., 1999; DiPrieto, 1996; Ostir et al., 1999; Rikli, & Jones, 1997).

The benefits or adaptations to physical activity have been identified by system (e.g., cardiovascular system), and according to two major subsets of physical activity: normal
physical activity (activity intended to complete normal daily activities) and exercise (activity intended to improve fitness and/or health). The latter, typically has involved what have been classified as resistance training, endurance or aerobic training, flexibility training, and/or balance training activities. While there is a vast expanse of literature in this general area of investigation, the purpose of this review is to provide a synthesis of the scientific literature concerning cardiovascular adaptations to resistance/strength training in older adults. To this end, the demography of aging and disablement, as well as design issues in "aging research" will first be addressed. The second chapter summarizes information concerning the aging of the cardiovascular system, acute cardiovascular response, and chronic cardiovascular adaptations to aerobic and anaerobic conditions. This is followed by a discussion of the existing information concerning cardiovascular adaptations to resistance training in older adults. Finally, information about exercise prescription and current trends in resistance/strength training prescription in the elderly are discussed.

1.1 Demography and Health Cost of Age

The demographics of an aging society, and the concomitant increase in health-care costs have prompted much of the interest in aging research. Life expectancy (i.e., the number of years a particular group can expect to live from a given point of time until death) has increased in recent years. Life expectancy in the U.S. in 1995 was 72.5 years for men and 78.9 years for women (National Center for Health Statistics, 1997), as opposed to 25 and 30 years for men and women respectively in 1900 (National Center for Health Statistics, 1997). Further, the 1995 life expectancy represents a 5 years increase from just 30 years earlier, when life expectancy was 67.1 years and 74.7 years for men and women,
respectively (For a review, see DiPrieto, & Seals, 1995; Ostir, et al., 1999). Initially, increases in life expectancy were attributed to decreasing in infant mortality, better sanitation and living conditions, and pharmacological treatment of parasitic diseases (e.g., malaria) and bacterial diseases (e.g., influenza, pneumonia, and tuberculosis). More recently, increases in life expectancy have also been attributed to better medical management of chronic disease (especially cardiovascular disease), changes in lifestyle (behavioral and social changes emphasizing health promotion), and improved nutrition (Schwartz et al., 1995; Wadsworth, 1997; Ostir et al., 1999). Paired with the heightened birth rate of the mid-1900s, this change in life expectancy explains the rapidly growing proportion of adults over the age of 60 (Taeuber, & Rosenwaike, 1993; for a review, see DiPrieto, 1996).

As a result of lower infant mortality and greater efficacy of treatment of chronic diseases, the elderly population are projected to increase more in size and proportion relative to the overall United States population (United States Bureau of the Census, 1996). It is projected that 18.5% of the U.S. population will be 65 years old and over by 2025, up from 12.8% in 1996 (Spencer, 1989), with the oldest old (85 years and older) the most rapidly growing segment (U.S. Bureau of the Census, 1996). In consideration of the recent reduction in infant mortality, it is not redundant to point out that life expectancy among older adults has also increased dramatically. In 1990, there were approximately 3 million persons aged 85 years or older living in the U.S., and this number is projected to increase to between 10 and 18 million by 2040 (For a review, see DiPrieto, 1996; DiPrieto, & Seals, 1995). Therefore, if the mortality rates in those born in
the 1940s and 1950s continue to decline, the size of the population of older adults will show a continual, rapid rise (DiPrieto, 1996; for a review, see DiPrieto & Seals, 1995).

Of concern in our aging society is the ever-increasing medical expenses incurred by older adults. This is due not only to the increase in the cost of medical care, but also due to an increase in the prevalence of functional impairment and chronic disease and the total number of people living with chronic disease. Approximately 80% of older adults have at least one chronic health problem, such as CV disease, cancer, diabetes, and auditory and/or visual impairment (Guralnik et al., 1989; U.S. Department of Health and Human Services, 1984). Thus, the use the long-term care services and the associated costs increase with age (Schneider & Guralnik, 1990). Chronic health problems among older Americans over 65 years account for approximately 30% of U.S. health care expenditures. It is estimated that the current annual cost of physical frailty is between 60 and 80 billion dollars, and it is projected that this cost will escalate to over 130 billion dollars by the year 2030 unless disability rates are lowered (Select Committee on Aging, 1992).

Aside from the costs incurred by the individual and to society, functional impairment has also negative consequences for autonomy and quality of life (King, 1991). This is evidenced by research from our own laboratory that has linked physical and cognitive function to quality of life among adults aged 60-93 (Wood, Reyes-Alvarez, Metoyer, & Welsch, 1999). When one considers that approximately 42% of older adults have some functional limitations, and that 10% require long-term care (Katz, 1983, World Health Organization, 1987), it is apparent that advances in the prevention of disablement has the potential to positively affect quality of life for a great many individuals.
The extent to which these are the consequences of aging or the cumulative effects of physical inactivity, nutritional deficits and/or other controllable factors that accompany aging is of concern to biologists, and economists alike. The further elucidation of the role of social, behavioral, and environmental influences on disablement may have profound impact on the health of an "aging" society.

1.2 Disability in Age

Disability has been defined from different contextual viewpoints. For example, disability in older population has been defined as the inability to perform or complete particular tasks or roles that previously could be accomplished without difficulty or the help of another person (Verbrugge, 1994). This definition suggests a deterioration or reduction in ability over time. Disability has also been defined as a difference, or gap, between an older individual’s capability to complete a particular task and the demand imposed by the task (Verbrugge, 1990).

It has been observed that the disability gap can either increase or decrease by changing or altering either capability or demand. For instance, if the demand of a task is held constant, decreasing capability widens the disability gap. A widening gap typically results in increasing difficulty, eventually resulting in loss of independence (Cornoni-Huntley, Foley, & Guralnik, 1991). Loss of independence, in turn, increases the risk for institutionalization or long-term care and death (Wolisly et al., 1993; Guralnik et al., 1991, 1993; Branch, Jette, & Evashiwick, 1981; Manton, 1988; Ostir et al., 1999).

Conversely, individuals might reduce or slow the widening disability gap using medical interventions (e.g., medications) or making adjustments in their lifestyles to accommodate or manage their disabilities better (e.g., relying on formal or informal...
supports or on mechanical devices such as walkers, reducing the demand of the task using ramps or elevators in place of steps, or making living arrangements to decrease the individual’s need to climb stairs). In any case, the main purpose would be to maintain function among older adults, even those mildly to moderately limited, an active, independent life. This is based on evidence indicating that older individuals who maintain an active life tend to be healthier, live longer, experience less disability, and less likely to be institutionalized than those who are inactive (Guralnik, & Simonsick, 1993).

According to schemes for disability in older adults, the progression to disablement begins with disease/pathology affecting one or more organ systems (Nagi, 1991; Verbrugge, & Jette, 1993; World Health Organization, 1980; Rikli, & Jones, 1997). The incidence of such disease or chronic condition may lead to impairment (i.e., loss or abnormality of psychological, physiological, or anatomical structure and function with consequences on physical, mental, or social functioning). Impairment progresses to functional limitation (i.e., restrictions in performing fundamental physical and mental actions used in daily life by one’s age group), and functional limitation leads to disability (i.e., experienced difficulty doing activities in any domain of life due to a health or physical problems).

In addition, Rikli & Jones (1997) suggest that the progression to disability might originate from inactivity as well as specific disease states, denoting that physical inactivity (disuse) and pathology may both have unique and interrelated effects on the disablement process. The independent or direct effect of physical inactivity on physical impairment and functional decline is associated with considerable physical atrophy that
results from disuse (Bloomfield, & Coyle, 1993; Bortz, 1982; Saltin et al., 1968; Shephard, 1994).

Additionally, physical inactivity has an indirect impact on the progression toward disability because of its influence on the disease process. The association between inactivity and the major chronic disease of aging (stroke, coronary artery disease, hypertension, non-insulin-dependent diabetes, osteoporosis, colon cancer, and some mental health problems) has been well documented in scientific reviews (Bouchard, Shephard, & Stephens, 1994), by leading health organizations (Pate et al., 1995), and most recently in the Surgeon General’s report on physical activity and health (Department of Health and Human Service, 1996).

Additional support for the role of physical inactivity relative to disability comes from epidemiological studies on factors affecting physical functioning in older adults (DiPrieto, 1996). Data from these studies (Kaplan, Stranwbridge, Camacho, & Cohen, 1993; Mor et al., 1989; Seeman et al., 1995) indicate that physical inactivity, along with chronic disease, is a determinant of functional decline (DiPrieto, 1996).

Equally relevant is the indication that physical decline, whether due to disease or disuse, is modifiable through proper physical activity intervention. This is supported by the results of number of experimental research studies (Cress et al., 1991; Fiatarone et al., 1990, 1994; Nichols, Hitzelberger, Sherman, & Patterson, 1995; Pyka, Linderberger, Charette, & Marcus, 1994; Rikli, & Edwards, 1991). Moreover, results from the Established Population for Epidemiological Studies (EPESE) (Lacroix et al., 1993) and from the Medical Outcomes Study (Stewart et al., 1994) indicate that the benefits of physical activity on functional capacity occur independent of disease. More, specifically,
findings from these studies show that physical activity participation is associated with higher levels of mobility, even for older adults who already have chronic conditions. Thus, it appears that disablement models should indicate that physical inactivity (disuse) and pathology might have independent as well as interrelated effects on the disablement process (Nagi, 1991; Rikli, & Jones, 1997).

1.3 Research Design Issues in the Elderly

Designing investigations of age-related changes in the structure and function of specific organ systems is a formidable challenge. Many factors covary with age, and are likely to have a substantial impact on or interaction with the "aging" process, thereby confounding our ability to ascertain the true effects of chronological aging. This is certainly true when investigating the structure and function of the cardiovascular system, where the influences of the highly prevalent cardiovascular diseases make the investigation of "age" practically impossible (For reviews, see Brooks, Fahey & White, 1996; DiPrieto, 1996; DiPrieto, & Seals, 1995; Lakatta, 1993; Limacher, 1994; Spirduso, 1996).

The potential influence of disease is one confounding factor in aging research. Others include: (a) the interpretation of data describing differences in cardiovascular function between older and younger individuals without having knowledge, or control for, interactions among age, disease, and life style; (b) the effects of non-symptomatic or undiagnosed pathologies; (c) differential aging of various organ systems, not only between individuals, but within individuals as well; (d) aspects of study design (cross-sectional studies or longitudinal study design) which neither quantify nor controlled for life-long habits of nutrition, exercise, cohort effects, or the development of occult disease or changes in life-style; (e) changes in methodology or of investigators over long periods
of time required to complete longitudinal studies, and substitutions of technology (practical impediments to the longitudinal approach); (f) how the “aging” term has been applied to a spectrum of life, so that changes in some cardiovascular regulatory mechanisms occurring over only parts of this age spectrum may be the same as, or opposite to, those occurring over other parts of the spectrum; (g) how the age range of adult individuals is sampled (erroneous and incomplete conclusions, interpretations, or generalizations regarding an age effect may be reached); (h) generalization of conclusions regarding age affects from studies in which the population has been arbitrarily dichotomized (e.g., humans older and younger than 60 years of age); (i) the fact that, in older individuals, non-cardiovascular factors often limit the amount of physical work that can be achieved (subtle age-associated differences in motivation to continue exercise, orthopedic limitations, alterations in body compositions, a reduction in muscle mass and strength, or a reduced threshold for neuromuscular fatigue interact to limit aerobic capacity in older individuals); (j) whether age-associated differences in noncardiac or cardiac factors that may limit aerobic work capacity are due to aging, per se, or, in part, to the sedentary life-style that accompanies aging; (k) different types of physiological aging among individuals (substantial variability among individuals) might have significant effects on the rate of decline in many bodily functions, and substantial variability among individuals; and (l) selective survival (robust surviving cohort) may influence the variability of adaptations to exercise training both within older populations and between younger and older adults. The individuals most susceptible to putative risk factors, and who might benefit most from training, die at earlier ages. Thus, research results may be biased because they are derived from studies of the effects of exercise
training on the more robust surviving cohort. (For a review, see Brooks, Fahey & White, 1996; DiPrieto, 1996; DiPrieto, & Seals, 1995; Folkow & Svanborg, 1993; Hagberg, 1994; Lakatta, 1993; Limacher, 1994; Seals, 1993; Spirduso, 1996).

1.4 Purpose of the Review

More than half of all deaths in older adults are due to cardiovascular (CV) disease (Surgeon General Report, 1996), and physical inactivity is known to be a major risk factor for the development and progression of CV diseases. Also, physical inactivity has been reported as one of the causes of dysfunction in later years (Wolisly et al., 1993; Guralnik et al., 1991, 1993; Branch, Jette, & Evashiwick, 1981; Manton, 1988; for a review see Ostir et al., 1999). Moreover, despite the awareness that physical activity may attenuate, or even reverse, the decline in function and ability due to inactivity, recent data suggest that only a very small percentage of older adults are active at a level commensurate with the recommendations for maintaining positive health. Furthermore, the positive influence of RE training in older adults has been recently reflect in the ACSM recommendations for exercise training for older adults. These guidelines suggest that older adults should engage in programs that incorporate both CV and resistance activities. However, beyond this general recommendation, there is little scientific evidence to guide clinicians in providing specific exercise programs (i.e. specific combinations of activities) that will maximize health and fitness benefits for older adults beyond this general recommendation. To this end, it would be of great value to examine the potential cardiovascular adaptations to resistance exercise in older adults. Therefore, purpose of this review is to address cardiovascular adaptations or benefits to resistance/strength training in older adults.
Several limitations have been imposed on this discussion: (1) for the purpose of this review, the aging process is operationally defined as changes that occur from early to late adulthood; (2) the discussion focuses primarily on healthy humans in an attempt to understand the direct effects of the aging process, and the complex interactions between aging and overt cardiovascular disease are not exhaustively discussed; and (3) the discussion focuses on dynamic resistance exercise performed with large groups, while information on the effects of aging on cardiovascular adjustments to isometric exercise (Ng et al., 1994a; Taylor et al., 1991) is limited and will not be discussed.
CHAPTER 2. AGING OF THE CARDIOVASCULAR SYSTEM

Cardiovascular (CV) health and function are dependent upon the structure and function of the heart, the aorta and arterial tree, and the components and volume of blood. It is therefore important to discuss structural and functional changes that occur within the CV system with age at the central and peripheral levels. These issues have been previously reviewed by Dempsey & Seal, 1995; Duchenry, 1990; Folkow & Svanborg, 1993; Fleg, 1986, & 1994; Lakatta, 1990, & 1993; Limacher, 1994; Seals, 1993; Seals et al., 1994; Spirduso, 1996; Shephard, 1997; and are briefly summarized here.

2.1 Changes in the Cardiovascular System with the Aging Process at Rest

The primary function of the cardiovascular (CV) system is to transport gases, nutrients, and metabolic waste products; maintain body temperature and acid-base balance; and transport hormones from the endocrine glands to their target organs (Wilmore and Costill, 1994). To be effective and efficient (i.e., to maintain function), the CV system must respond to environmental cues such as increased skeletal muscle activity, postural change, or thermal stress. Thus, CV function is often described in terms of the maximum capacity of the system to adapt to such stresses (e.g., increased skeletal muscle activity, thermal stress).

In order to accomplish these functions, the CV system basically consists of a pump (i.e., heart or central CV system), and the vascular system (i.e., peripheral vascular system). The term cardio refers to the heart, and the term vascular refers to the blood vessels, blood components and blood volume. Accordingly, in the following sub-section, structural and functional changes in both central and peripheral function are summarized.
At rest, the CV system in healthy people has little difficulty supplying oxygen and fuel to the tissues. Therefore, while CV structure and function change with age in the healthy adult, such changes do not typically provide an immediate threat to the need for delivery of blood under resting conditions. However, these changes do significantly impact the older adult's ability to adjust to and sustain elevated physical demands such as during vigorous dynamic exercise (Gerstenblith, Renlund, & Lakatta, 1987; Lakatta, 1990).

2.1.1 Central Function

As stated previously, it is difficult to separate completely the effects of age from those of other factors known to influence CV structure and function in human beings. The presence of heart disease appears to have a particularly weighty confounding influence. Autopsy studies show that as many as 60% of older patients have some evidence of coronary artery disease (CAD), and that clinical manifestations of coronary disease are likely to be present in at least half of them (Ackerman et al., 1950; Elveback & Lie, 1984; White et al., 1950). Nonetheless, age-related structural and functional changes in the heart have been reported, and are believed to occur, to the best of our knowledge, even in the absence of disease. These age-related structural and functional changes in the heart are summarized below.

**Age-related changes in the structure of the heart.** There are not many macro or microscopic changes in the heart due to the aging process alone. There is evidence of degeneration and loss of heart cells, but these changes are not thought to lead to functional abnormalities (For a review, see Fleg, 1986; Lakatta, 1993; and Spirduso, 1996). The main structural changes in the heart due to aging are reported in Table 1.
Table 1. Structural Changes in the Heart due to Aging.

<table>
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<th>Change</th>
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<tr>
<td>Increased size and heart mass</td>
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<tr>
<td>Increased ratio of heart weight to body weight</td>
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<tr>
<td>Increased inter-ventricular septal thickness</td>
</tr>
<tr>
<td>Increased left ventricular free wall thickness</td>
</tr>
<tr>
<td>Increased cardiothoracic ratio</td>
</tr>
<tr>
<td>Increased end-diastolic and end-systolic left ventricular wall thickness and estimated left ventricular mass</td>
</tr>
<tr>
<td>Increased LV cavity size at end-diastolic and end-systolic</td>
</tr>
<tr>
<td>Increased myocyte size, and cardio muscle-to-collagen ratio</td>
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</table>

These changes have been observed in studies using autopsy data from human hearts (Kitzman et al. 1988; Linzbach & Akuanoa-Boaten, 1973), and cross-sectional and longitudinal studies using chest X-rays (Ensor et al., 1983; Potter et al., 1982; Stradell, 1964), echocardiography (Smith et al., 1991; Sutton et al, 1980), or gated blood-pool scans (Fleg et al., 1990b; Rodeheffer et al., 1984; Vantosh et al., 1980). In one of the most impressive autopsy studies conducted, Linzbach & Akuanoa-Boaten (1973) found that heart weight increased an average of 1g/yr in men and 1.5 g/year in women between ages 30 and 90. This study analyzed 7,112 human hearts of normal patients and those with CV diseases (CVD), and demonstrated an increase in the heart to body weight ratio. However, recent autopsy analyses of female hearts without CAD have documented an age-related increase in heart mass (Kitzman et al. 1988). This suggests that some of the age-related increases in cardiac mass may reflect the higher prevalence of disease in the older heart. Closer inspection of the aging heart reveals that the intraventricular septal wall thickness increases more with age than does the left ventricular free wall thickness (For a review, see Fleg, 1986; Lakatta, 1993).
Resting heart size. The increase in the resting heart size with age has been observed in the supine position in some (Granath & Strandell, 1964; Vantosh et al., 1980) but not all (Mann et al., 1986) studies. An increase in heart mass with age is partly attributed to an increase in myocyte size (Unverferth et al., 1983). In some older, apparently healthy individuals in who left ventricular (LV) mass decreases with age (Olivetti et al., 1991), cardiac myocyte enlargement occurs concurrently with an estimated decrease in myocyte number. For example, in a series of patients undergoing endomyocardial biopsy, an observed age-related increase in LV weight was attributed to cellular hypertrophy rather than hyperplasia (Unverferth et al., 1983). Hypertrophy usually occurs in valvular disease and/or hypertension. As wall thickness increases, the abnormally elevated LV stress (systolic blood pressure x radius/wall thickness) returns toward normal. Myocyte hypertrophy enhances the shortening of each cardiac muscle cell and allows normal ejection of blood during systole. Because neither resting LV dimension nor SV increases substantially with age, volume overload would seem an unlikely stimulus of LV hypertrophy. However, it has long been recognized that systolic blood pressure increases with age at both rest and submaximal workloads, even in normotensive subjects. Thus, the modest age-related hypertrophy of the LV may be due to an adaptation to the rise in systolic blood pressure that occurs with age. Further, the progressive dilation of the ascending aorta with age (Gardin et al., 1979) creates a higher inertial force that the heart must overcome before ejection can occur (For a review, see Fleg, 1986; Lakatta, 1993). Thus, the increased afterload imposed on the heart is suspected to promote or exacerbate LV hypertrophy.
Cardiothoracic ratio. Cross-sectional studies using chest X-rays have found that the cardiothoracic ratio (the ratio between heart size and thorax size) increases or does not change with age, yet the mechanisms for this change may vary across individuals. Longitudinal studies consistently report that heart size increases between 60 and 98 years of age (Ensor et al., 1983; Potter et al., 1982). From the Baltimore Longitudinal Study of Aging (BLSA), Ensor et al. (1983) examined 66 healthy men (initial mean age: 48 yrs) and found that the cardiothoracic ratio increased from .405 to .427 over a 17-year follow-up. This increase reflected a small increase in cardiac diameter, as well as a diminution in chest diameter with advancing age. Stradell (1964) demonstrated gender-related influences on the cardiac thoracic ratio. In men, the change was attributed to an increase in the cardiac diameter and in women it was attributed to a reduction in thoracic diameter.

End-diastolic and end-systolic left ventricular (LV) wall thickness and estimated LV mass. Cross-sectional studies with sedentary volunteer subjects without disease, using M-mode echocardiography, indicate that the end-diastolic and end-systolic left ventricular (LV) wall thickness and estimated LV mass increase progressively with age (Gardin et al., 1979; Gerstenblith et al., 1977; Sjogren, 1971; Smith et al., 1991; Sutton et al., 1980). For example, in 62 healthy men aged 25 to 84 with resting blood pressure <140/90 mm Hg, Gerstenblith et al. (1977) found that the increase in LV posterior wall thickness correlated with increasing age. Sjögren (1971), in 100 healthy men and women, demonstrated a progressive increase in left ventricular (LV) posterior wall thickness between the ages of 15 and 65. The increase in LV wall thickness, which is observed in both diastole and systole, approximates 30% between ages 25 and 80 and is similar
whether or not measurements are corrected for body surface area (Gerstenblith et al., 1977; Sjogren, 1971). Other studies using M-mode echocardiography have found a minor increase in LV mass with age in healthy women and no change in healthy men (Dannenberg et al., 1989). The difference in these results may be due to how the M-mode echocardiography is used. M-mode echocardiography extrapolates the global LV thickness taking samples of a single small area of the LV posterior wall or ventricular septum. LV mass is then estimated assuming a constant ventricular geometry at all ages (For a review, see Fleg, 1986). In addition, it should be noted that the determination of dimension changes by echocardiography might be less accurate than other derived parameters because of reliance upon a distance measurement between the interventricular septum and one area of the left ventricular posterior wall. Movements along this line may not be as representative of the entire left ventricle in some age groups as it is in others. Therefore, lateral resolution problems due to an inability to completely focus ultrasonic waves over the entire depth range of the beam may distort left ventricular septal and endocardial echoes (Gerstenblith et al., 1977; Roelandt et al., 1976)

Another age-related structural change is in LV diastolic and systolic cavity size. Some studies examining LV diastolic and systolic cavity dimensions report no age-related changes (Gerstenblith et al., 1977; Sjogren, 1972; Gardin et al., 1979). However, Fleg et al. (1990b), Rodeheffer et al. (1984) and Vantosh et al. (1980) found that the LV cavity size at end-diastole and end-systole increase moderately with age in healthy, normotensive, sedentary men but do not vary with age in women. LV cavity dimensions were measured either in the semisupine position by two-dimensional echocardiography or in the sitting position by gated cardiac blood pool scans of technetium-labeled red
blood cells. The reduction in LV cavity volume observed in a small number of older
individuals, particularly women (Karan et al., 1989; Topol et al., 1985), is an exception to
the rule and is likely associated with clinical arterial hypertension (Joint National

M-mode echocardiography has revealed additional cardiac anatomic changes with
age. For example, (1) Gerstenblith et al. (1977) found that the age-related increase in
aortic root diastolic diameter (i.e., cross-sectional measurement of aortic diastolic
dimension from echocardiogram) averaged 6% between the fourth and eighth decades in
men from the BLSA; (2) Gardin et al. (1979) found a 20% increase in left atrial
dimension between ages 18 and 93 (similar increases in left atrial size have been found in
other conditions accompanied by LV hypertrophy, suggesting decreased LV compliance
as a causative factor); and (3) Early diastolic closure rate of the mitral valve
is observed
to decrease approximately 50% between ages 25 and 80 in BLSA, when indexed by the
E-F slope (i.e., segment of the reflected wave representation of the anterior mitral valve
leaflet movements and that corresponds to the LV reduced filling phase) (Berne and
Levy, 1998; Gardin et al., 1979; Gerstenblith et al., 1977). Such a decrease in mitral
valve closure rate may reflect impaired early diastolic filling with advanced age. This
decline could reflect a limitation in the rate of opening valve motion imposed by age-
associated changes in either LV compliance (Harrison et al., 1964) or primary stiffness of
the mitral valve itself (Winters, Hafer, & Soloff, 1969. The increase in the isovolumetric
relaxation time by 40% between the third and ninth decade (Harrison et al., 1964), and
the demonstration of a slowing of the peak velocity of rapid mitral inflow with age
(Miyatake et al., 1984) support that diminished LV compliance might account for
decreased closure rate of mitral valve. Alternatively, the reported findings that closure rate of diseased valves is related to the presence of valve thickening, and pathological studies describing age-associated change in the leaflets themselves may also account for the decreased in E-F slope (Harrinson et al., 1964; Sell & Scully, 1965; McMillan, & Lev, 1964). In addition, Miyatake et al. (1985) found an age-related increase of mitral inflow velocity during atrial contraction. Thus, the relative influence of early and late diastolic filling phases might be reversed with advancing age. (For a review, see Fleg, 1986).

Other reported age-related structural changes in the heart with advanced age are: (1) an increase in the amount of and a change in physical properties of collagen within the myocardium (for review, see Gerstenblith et al., 1976). However, the cardiac muscle-to-collagen ratio remains constant or increases in the older heart (Olivetti et al., 1991; for a review, see Lakatta, 1993). (2) An increase in circumference of valves of the heart, particularly the aortic and pulmonary valves (For a review, see Spirduso, 1996).

Myocardial lipofuscin increases with age (Strehler et al, 1959), but the consequences of this morphological change are unknown. Additionally, some forms of amyloid protein can be found in the heart of about one-half of individuals over 70 years of age (Lakatta et al., 1987). Whether the cardiac form of amyloid can strictly be considered a feature of “normal” aging is debatable, since it is not an invariable finding, even in centenarians. The type of amyloid accumulation in primary cardiac amyloidosis (i.e., that associated with atrophy of the myofibers and a firm, large, waxy heart) is not a feature of cardiac amyloid deposition in healthy older individuals (Lakatta et al., 1987).
In summary, aging between 20 and 80 years appears to be associated with a modest increase in LV wall thickness, due mainly to an increase in the size of cardiac myocytes. The increase in heart mass with age is exaggerated by coexisting disease (coronary artery and/or hypertension).

Cardiac function and age: The heart is actually two separate pumps: a right heart that pumps the blood to the lungs and a left heart that pumps the blood to the systemic circulation and the organs it serves (Berne, & Levy, 1998). Each side of the heart provides a pulsatile two-chamber pump comprised of an atrium and a ventricle. The atrium functions principally as a primer pump for the ventricle, helping to move the blood into the ventricle and also serves to help siphon venous blood back to the heart. The ventricle in turn supplies the main force that propels the blood through either the pulmonary or the systemic circulation (Guyton & Hall, 1996). Thus, the purpose of cardiac muscle contraction is to generate pressure within the blood-filled atrial and ventricular chambers of the heart and propel blood down a pressure gradient.

Cardiac function/performance is generally described in terms of cardiac output (CO), and determined by four primary factors: heart rate, diastolic filling (preload), inotropic state (contractility), and aortic pressure (afterload) (see figure 1). Heart rate is the speed at which the heart is beating or the number of times that the heart beats over one minute (e.g., 90 beats/minute). Preload is the extent to which the ventricles are stretched when they fill with blood. With increasing preload (i.e., greater stretch) the ventricles increase the force of contraction, with maximum force generated somewhere around 115%-120% or normal resting length. When stretched beyond this point, myocardial force production begins to decline. Inotropic state, or contractility, is the strength of the myocardial
contraction and afterload is the resistance the heart meets as it attempts to pump the blood into the circulation. Increased systemic blood pressure increases afterload and, consequently, decreases CO (Brooks, Fahey, & White, 1995).

![Figure 1. Determinant Factors of Cardiac Output](image)

**Cardiac output (CO):** Cardiac output, the product of heart rate (HR) and stroke volume (SV), is defined as the amount of blood ejected per minute by the heart, specifically by the LV. Cardiac output, called cardiac index (CI) when normalized for body surface size, is probably the most important overall measure of cardiac performance. It represents the ability of the heart to meet the oxygen requirements of the entire body. During times of increased demand, CO increases as a result of a rise in HR and/or SV. Human CO can be measured through invasive (catherization, Fick method or indicator-dilution method) and non-invasive (echocardiography, radionuclide imaging, and impedance cardiography) procedures.
There is some debate as to the influence of the aging process on resting CO, as several cross-sectional studies report an age-related reduction in resting CO (Brandfonbrener et al., 1955; Conway et al., 1971; Cournand et al., 1945; Granath et al., 1964; Hunt et al., 1997; Julius et al., 1967; Lewis, 1938; Messerli et al., 1981; Seals, 1993; Strandell, 1964a; & 1964b), while others suggest that resting CO remains unchanged (Dinenno et al., 1999; Fleg et al., 1990b; Higginbotham et al., 1984; Rodeheffer et al., 1986; & 1984). In a group of 67 hospitalized men without obvious cardiovascular disease, resting CO (determined by indicator dilution) fell from 6.5 liters/min. in men in their 20s to 3.9 liters/min. in men in their 80s; an average decline of 1% per year (Brandfonbrener et al., 1955). This change tended to be more pronounced in the supine than in the upright position, and remained highly significant, even after correction for a small age-related diminution of body surface area. This age-related difference could not be accounted for by an age-related decline in metabolic rate. In other words, resting CO was also lower when expressed per unit oxygen consumption (Granath et al., 1964). However, it is probable that a significant portion of elderly subjects with latent CAD were included in this and other studies showing similar results (Dempsey & Seals, 1995; Fleg, 1986; Lakatta, 1993; Seals, 1993; Spirduso, 1996). Therefore, disease related changes in cardiac performance couldn’t be ruled out as a potential source of the decline in CO that has been observed in older subjects. Other studies have also reported a lower resting CO with age in healthy women (Hunt et al., 1997) (For a review; Dempsey & Seals, 1995; Fleg, 1986; Lakatta, 1993; Spirduso, 1996).

Among those who have found that resting CO does not change with age are Rodeheffer et al. In contrast to the studies mentioned above, the subjects in this
investigation, aged 30 to 80, did not display symptoms of CVD, and their electrocardiograms at rest and during maximal exercise were normal. Moreover, all subjects aged 40 and older had undergone exercise thallium scintigraphy and showed no myocardial perfusion defects. Later, Fleg et al. (1990b), in a population of 95-males without CAD, found that the resting CI does not change with age, and indicated that an increase in stroke volume index (SVI), due to an increase in the end diastolic volume index (EDVI), compensates for age-associated reduction in HR. In the same study, however, recumbent CI was found to decrease slightly with age in women, in part due to the absence of cardiac dilation at end diastole and its accompanying increase in SVI.

It should be noted that studies of CO are difficult to interpret for several reasons: (1) the invasive methodology used in these studies may not be used to measure cardiac performance under true resting conditions, since age-related differences elicited under such conditions may represent differences in response to stress; (2) a lack of standardization with respect to screening for the presence of CAD; (3) physical conditioning level differs widely among participants; (4) the meaningfulness of CO or SV as a measure of intrinsic cardiac performance is questionable because differences in preload or afterload may modify these measurements; and (5) the effect of age on CO appears to be highly variable across different populations. As to this latter point, studies of resting hemodynamic variables by Brandfonbrener et al. (1955) and Rodeheffer et al (1984) reveal slight age-related decreases in HR and increases in systolic blood pressure (SBP), but only Brandfonbrener et al found a decrease in SV and an increase in systemic vascular resistance. (For a review, see Fleg, 1986).
Additionally, the variability of the results among CO studies may be attributed to the following factors: body composition, age range studied, gender differences, number of individuals selected, and the body position during study. For example, CO is influenced by the basal metabolic rate and body composition, both of which vary substantially across age groups. However, age-associated declines in basal metabolic rate are abolished when O2 consumption (VO2) is normalized to an index of lean muscle mass (Tzankoff & Norris, 1977). In regard to the age range studied, it has been observed that age influences the measurement of cardiac function at rest. However, one study reporting a marked age-associated decline in resting CO and in ejection fraction (Kuikka & Lansimies, 1982) demonstrated that most of the age effect occurred between the ages of 6 and 20 years of age. Lastly, there appears to be some gender influence with age on the outcome of cardiac function studies. For example, Fleg et al. (1990b) found that resting CI does not change with age in men; however, in the same study, the resting, sitting CI was found to decrease slightly with age in women (For a review; Lakatta, 1993).

In summary, resting CO in healthy aging individuals free of cardiac disease is relatively unchanged with age, at least in men (Dinneno et al., 1999; Fleg et al., 1990b & 1995; Rodeheffer et al., 1984). Cardiac output in these healthy people is unchanged because SV and EDV (preload) are maintained or even slightly increased with advancing age (Fleg et al., 1990b; Rodeheffer et al., 1984). Thus, older adult men can increase or maintain resting CO (Dinneno et al., 1999; Fleg et al, 1990b; Geokas et al., 1990; Lund-Johansen, 1988) by a smaller increase in HR but a greater reliance on the Frank-Starling reflex mechanism (Mann et al., 1986). In contrast, neither EDV nor SVI appear to increase with age in women (Fleg et al, 1990b; Hunt et al., 1997), possibly as a result of
the lack of estrogen after menopause (Giraud et al., 1993; Pines et al., 1991; Scheuer et al., 1987); thus, resting CO decreases slightly with age in women. Furthermore, persons with hypertension do experience an age-related decline in CO (Lund-Johansen, 1989) (For a review, see Dempsey & Seals, 1995; Fleg, 1986; Lakatta, 1993; Spirduso, 1996)

Heart rate and rhythm. In comparing younger and older individuals, most cross-sectional studies indicate that the supine basal HR is not different (Fleg et al., 1990ba; & 1990b; Schwartz et al., 1991; Simpson & Wicks, 1988). However, studies in the sitting and upright position indicate that resting HR decreases with age in both men and women (Fleg et al., 1990b; Schwartz et al., 1991; Simpson & Wicks, 1988). Furthermore, among participants of a longitudinal study over a period of 30 years, resting HR decreased from 81 to 60 beats/min (Arora et al., 1987). (For a review, see Dempsey & Seals, 1995; Docherty, 1990; Granath et al., 1964; Lakatta, 1993; Seals, 1993; Seals et al., 1994; Spirdusso, 1996). An increased prevalence of arrhythmias is also observed with aging, but this is not necessarily clinically significant or an indication of impaired function (Lakatta, 1993).

There are three potential explanations for the age-related decline in resting HR. The interaction of age and posture on HR, as noted above, suggests that age-associated changes might be present in mechanisms that regulate HR. For example, the balance of sympathetic and parasympathetic tone (i.e., autonomic tone) partly modulates heart rate, with the parasympathetic predominating at rest. This is supported by the observation that variation of the HR, which is also determined largely by autonomic tone, is diminished with advancing age (Davies, 1975; Kawamoto et al., 1989; Kostis et al., 1982; Pfeifer et al., 1983; Korkushko et al., 1991; Schwartz et al., 1991; Simpson et al., 1988). The lower
variation in HR with aging has been thought to result from a reduction in both parasympathetic and sympathetic modulation. (For a review, see Lakatta, 1993).

The age-related decline in resting HR may also be related to impaired responsiveness beta-adrenergic stimulation; i.e., changes in adrenergic receptor sensitivity (Conway et al., 1971; Seal et al., 1994c; Yin et al., 1976). Plasma levels of norepinephrine and epinephrine at rest appear to increase with age in many (Esler et al., 1981; Featherstone et al., 1984; Kawamoto et al., 1989; Rowe et al., 1980) but not all studies (Fleg et al., 1985). This has been interpreted as an indication of spillover secondary to decreased beta-receptor sensitivity (i.e., a decreased sensibility to catecholamine with age) or possibly beta-receptor number. However, the influence of such a change at rest is questionable since sympathetic control of the heart is not particularly important. In fact, some studies have found that blocking only the sympathetic system at rest with propranolol does not produce a differential effect with age on HR or LV hemodynamics in healthy men at rest (Yin et al., 1978). Rather, a third, and perhaps more promising explanation, is the possibility of changes in the intrinsic sinus node rate (Lakatta, 1993). Dual blocked studies reveal that the intrinsic rate is significantly diminished with age. At age 20, the average intrinsic HR is 104 beats/min as compared with 92 beats/min in a 45-55-year age group (Jose, 1966). More data are needed to describe intrinsic HR of older individuals (For a review, see Lakatta, 1993).

This third explanation (change in the intrinsic rate) for the age-related decline in resting HR focuses on the possibility that some age-related structural changes on the heart might be related to rate changes in the intrinsic sinus node of the heart. These structural changes might be: (1) an increase in elastic and collagenous tissue in all parts
of the conduction system and fat accumulation around the sinoatrial (SA) node; (2) a pronounced decrease beginning by age 60 years in the number of pacemaker cells in the SA node, and <10% of the cell number found in the young adult remains by age 75 years (Fleg, Gersteblith, & Lakatta, 1988); and (3) a variable degree of calcification of the left side of the cardiac skeleton, which includes the aortic and mitral annuli, the central fibrous body, and the summit of the interventricular septum. Because of their proximity to these structures, the atrioventricular (AV) node, AV bundle, bifurcation, and proximal left and right bundle branches may be affected by this process, resulting in so-called primary or idiopathic heart block. A modest prolongation of the P-R interval within the normal range (<20 ms) occurs with aging in healthy individuals and is localized to the proximal P-R segment, probably reflecting a delay within the AV junction (Fleg et al., 1990b).

*Stroke volume (SV).* Stroke volume, which is determined by the difference between ventricular filling (EDV) and ventricular emptying (ESV), is the amount the blood (~70 milliliters) that is ejected from the ventricle during a systole. When normalizes according to body surface area, it is called stroke volume index (SVI).

A few studies have demonstrated a decrease in resting SV with advancing age (Brandfonbrener et al., 1955; Nichols et al., 1985; and see Gerstenblith et al., 1976 for review). For example, Brandfonbrener et al. (1955), in a group of 67 hospitalized men without obvious cardiovascular disease, demonstrated a decline in resting SV of ~5% per decade. In this investigation, SV was determined by indicator dilution method and declined from 85 ml (~20 yrs) to 60 ml (~80 yrs). These age-related differences, even after correction for a small age-related diminution of body surface area, remained highly
significant. It might be possible that a significant portion of elderly subjects with latent CAD were included in this and other studies showing similar results (For a review, see Dempsey & Seals, 1995; Fleg, 1986). It has been documented that older adults with CVD have lower SV in comparison to healthy, sedentary- or active-counterparts (Blackmon et al., 1967; Rowell, 1993).

In contrast, some studies have found that resting SVI in the sitting position does not decrease with advancing age in normotensive subjects (Fleg et al., 1990b; Higginbotham et al., 1986a; Proper & Wall, 1972; Rodeheffer et al., 1984; Wei et al., 1989; Younis et al., 1990). Rodeheffer et al. (1984), using gated blood pool scan and gated radionuclide angiography, found an age-related increase in SVI. This increase in SVI was sufficient to prevent a decline in CO that otherwise would have been expected as a result of the decline in resting HR. This subset of BLSA participants consisted of active volunteers in whom angina and symptoms of CV disease were absent and in whom electrocardiograms at rest and during maximal exercise were normal. Therefore, all subjects (≥ 40 yrs) showed no myocardial perfusion defects. It should be noted that even in these carefully screened subjects, daily activity energy expenditure declined with age, indicating that the elderly participants did not represent an atypical, highly conditioned subgroup (For a review, see Fleg, 1986; Lakatta, 1993; Spirduso, 1996). Other investigators have found that resting SVI increases slightly with age in men but does not in women (Fleg et al., 1990b; Featherstone et al., 1987). The increase in SVI in men is attributed to the increase in EDVI, whereas among healthy women neither the resting EDVI nor SVI increases with age, and the end systolic volume index (ESVI) tends to decrease slightly (Fleg et al.,
The abnormal changes in men appear after middle age (Higginbotham et al., 1986a; Lakatta, 1993).

The gender differences in SVI, and other cardiac volumes (EDVI, and ESVI), among healthy sedentary individuals may be partly attributed to differences in fitness levels between men and women as gender added no further predictive value to step-wise multiple regression of various influences on cardiac volumes once age and fitness levels were stepped in (Fleg et al., 1990b). This is also supported by a prior matching of younger and older subjects with similar exercise capacities (Lakatta et al., 1991). Furthermore, it is unclear if any observed decline in SV with age is linked to a smaller total blood volume (Davy & Seals, 1994; Strandell, 1964c) (For a review, see Dempsey & Seals, 1995).

The electrical and mechanical changes that occur in the heart during and following a single heartbeat is called cardiac cycle. This cycle include the contraction (ventricular systole) and relaxation (ventricular diastole) of the myocardium, as well as pressure and volume changes. To describe and study the electrical and mechanical changes on the heart for each cardiac cycle, the ventricular systole is subdivided in two phases (isovolumetric contraction and ejection phases) and the ventricular diastole in four (isovolumetric relaxation, rapid or early filling, slow filling or diastasis and atrial systole phases). The next subdivision is referred to LV filling, which includes three out of four ventricular diastole phases (rapid or early filling, slow filling or diastasis and atrial systole).

**Left ventricle filling.** The majority (@75%) of LV filling occurs immediately on opening of the AV valves. At this point the blood that had returned to the atria during the
previous ventricular systole is abruptly released into the relaxing ventricles. This period of LV filling is called "the rapid or early diastolic filling phase." This rapid flow of blood from atria to relaxing ventricles is a result of a decrease in atrial and ventricle pressures and a sharp increase in ventricular volume. After this rapid filling phase, blood returning from the periphery flows into the right ventricle, and blood from the lungs flows into the LV. This small, slow addition (5-10%) to ventricular filling, called "slow or late diastolic filling phase (diastasis)," is characterized by a gradual rise in atrial, ventricular, and venous pressures and in ventricular volume. Lastly, the transfer of blood from atrium to ventricle completes the period of ventricle filling. This last phase, "the atrial systole," is responsible for the remainder increase (15-20%) in ventricular volume, as well as a further increase in atrial, ventricular, and venous pressures (Berne and Levy, 1998).

Several studies suggest the existence of age-related changes in LV filling pattern. Investigations employing Doppler echocardiography reveal that early ventricular flow is reduced and that the contribution of atrial flow is increased with advancing age (Arora et al., 1987; Bonow et al., 1988; Chou et al., 1990; Gardin et al., 1987; Gerstenblith et al., 1977; Kitzman et al., 1991; Kuo et al., 1987; Miyatake et al., 1984; Sartori et al., 1987; Spirito, & Maron, 1988). Similar finding have been reported with Doppler color-flow imaging (Bryg et al., 1987) as well as with radionuclide angiography imaging (Miller et al., 1986), suggesting the possibility of an age-related alteration in left ventricular diastolic function. For example, Kitzman et al. (1991), in a small study sample in which neither systolic arterial pressure nor LV wall thickness increased with age, found that the pulmonary capillary wedge pressure (an index of the LV end-diastolic filling) was still observed to increase with age, and a marked reduction of early filling rate occurred in the
older individuals. This might also suggest also that the early LV filling deficit that accompanies aging may not be directly related to an increase in LV wall thickness.

It should be mentioned that in hypertensives, the decrease in the early ventricular filling rate varies directly with the isovolumetric relaxation time (Smith et al., 1987). Therefore, after older hypertensives have been treated with Ca\(^{2+}\) channel blocker treatment, increases in LV peak filling rate were accompanied by reductions in LV mass (Schulman et al., 1990). (For a review, see Spirdusso, 1996).

The suggested reasons for a reduced early diastolic filling include a diminished LV compliance, and/or a primary stiffening of the mitral valve. In the case of LV compliance, it should be noted that LV filling begins as ventricular pressure decreases below that in the atrium and continues during cardiac diastole with a further increase of AV pressure gradient. However, one main determinant of the ventricular pressure and thus of the AV pressure gradient is the compliance/stiffness of the ventricular myocardium. The initial ventricular pressure reduction is due to relaxation of myocardial fibers from the prior systole, that begins before ventricular filling (i.e., during the isovolumetric relaxation period), and it continues to occur following opening of the mitral valve (during the early LV filling period). While ventricular stiffness (more often referred to as compliance) is often thought to reflect the structural properties of the myocardium during the early filling period, the ventricular stiffness is due, in large part, to the extent of the declining Ca\(^{2+}\) -dependent myofilament interaction. Thus active (i.e., Ca\(^{2+}\) -dependent) and structural mechanisms likely regulate the rates of ventricular pressure decay and early filling. In summary, it has been suggested that a diminished AV pressure gradient (due to a diminished LV compliance and a reduced ventricular...
relaxation) causes a decrease in the early diastolic filling. Alternatively, as reported above (see structural changes in the heart), an impaired early diastolic filling has been suggested as a result of the decreased early diastolic closure rate of the mitral valve (Gerstenblith et al., 1977).

It should be mentioned that Doppler diastolic indexes might be difficult to interpret because it has been shown that they can be altered for different factors or conditions. For example, early systemic and pulmonary hypertension, as well coronary artery, myocardial and valvular heart disease (Inouye et al., 1984; Spiritu, & Maron, 1988b; Louie et al., 1986), all of which are common in the elderly and may be undetected (Wei, 1988; Cobb et al., 1986; Kitzman et al., 1991; Shenoy, et al., 1986). Furthermore, other studies have demonstrated that Doppler and other noninvasive indexes of LV diastolic filling are influenced by HR (Gardin et al., 1987; Kuo et al., 1987; Spiritu, & Maron, 1988b; Vandenberg et al., 1988; Harrison et al., 1991; Johannessen et al., 1991), preload (Spiritu, & Maron, 1988b; Choog et al., 1987; Triulzi et al., Stoddard et al., 1989; Choong et al., 1988; Nishimura et al., 1989), afterload (Choong et al., 1988; Nishimura et al., 1989; Sheikh et al., 1989), contractility (Vandenberg et al., 1988, Sheikh et al., 1989) and LV mass (Schulman et al., 1990), all of which have been variably reported to be altered in elderly subjects (Spiritu, & Maron, 1988b; Wei, 1988; Sheikh et al., 1989; Schulman et al., 1990; Higginbothman, et al., 1986; Kitzmam et al., 1990; Fagard et al., 1991; Downes et al., 1989; Davidson, & Free, 1990; Granath et al., 1964; Nixon et al., 1985; Gerstenblith et al., 1977; Henry et al., 1980; Port et al., 1980; Templeton et al., 1979)). In addition, it has been reported that life-style variables (e.g., physical activity and ethanol) affect the LV filling rate measured via Doppler technique in younger
individuals (Voutilainen et al., 1991). However, the peak LV early filling rate measured by radionuclide imaging or by echo-Doppler techniques does not differ between older endurance-trained athletes and age-matched sedentary controls (Forman et al., 1992; Schulman et al., 1994).

In addition to the change in the early ventricular flow, an increased atrial contribution (30-40%) in the ventricular filling (Arora et al., 1987; Chou et al., 1990; Kuo et al., 1987; Miyatake et al., 1984; Pearson, Gudipati, & Labovitz, 1991; Spirito et al., 1988; Swinne et al., 1992) has also been observed. This percentage of atrial contribution is two-fold larger than the 15-20% in healthy younger and middle age persons. For example, Pearson, Gudipati, & Labovitz (1991), in one group of healthy, physically active subjects over 65 years of age, showed that the atrial contribution to ventricular filling was almost twice (37%) that of 25-year-old men (19%). This additional increment in the LV filling through the atrial systole compensates for the reduced early diastolic filling rate, and maintains the LV filling volume at a normal level. Furthermore, the enhanced atrial contribution to ventricular filling with advancing age has been associated with a left atrial enlargement (Gardin et al., 1979; Gerstenblith et al., 1977), and is the basis of an audible fourth heart sound in most healthy older individuals (Fleg et al., 1988). Gardin et al (1979) showed a 20% increase in left atrial dimension between ages 18 and 93, which have been found in other conditions accompanied by LV hypertrophy. It might suggest a decreased LV compliance as a causative factor (For a review, see Spirdusso, 1996).

In summary, it has been documented that in younger and middle-aged persons, the early diastolic filling represents the greatest contribution to the EDV. However, LV
filling, during early diastole filling, is markedly (50%) reduced between the ages of 20 and 80 years in healthy men and women. This finding has been shown by studies using echocardiography, echo-Doppler, or radionuclide technique (Arora et al., 1987; Bonow et al., 1988; Chou et al., 1990; Gerstenblith et al., 1977; Kitzman et al., 1991; Kostis et al., 1982; Kuecherer et al., 1988; Messerli et al., 1984; Smith et al., 1985; Spiritu, & Maron, 1988; Swinne et al., 1992; Voutilanen et al., 1991. Furthermore, an increment in the LV filling through the atrial systole, which has been associated with a left atrial enlargement has been observed (Gardin et al., 1979; Gerstenblith et al., 1977).

**Preload or End-diastolic volume:** EDV is the amount of blood (about 110 to 120 milliliter) in the ventricles at the end of diastole, just prior to the next contraction. EDV, which is referred to as “preload,” influences cardiac performance through the Frank-Starling Law of the heart. That is, the increase in EDV results in a lengthening of cardiac fibers, which improves the force of contraction. Thus, a rise in cardiac contraction results in an increase in the amount of blood pumped per beat (see figure 1). The principal variable that influences EDV is the rate of venous return to the heart, so that an increase in venous return results in a rise in EDV and thus an increase in cardiac performance.

Despite the reduction in the LV early diastolic filling rate with age (Miyatake et al., 1984; Bryg et al., 1987; Sartori et al., 1987; Gardin et al., 1987; Kuo et al., 1987; Spirito, & Maron, 1988), and while blood volume in healthy men does not appear to change with age (Strandell, 1964), the LV EDV at rest is not reduced in older healthy individuals (Fleg et al., 1990b; Lakatta et al., 1991; Rodeheffer et al., 1986; Rodeheffer et al., 1984). In fact, LV EDV moderately increases in healthy sedentary older- versus younger-men in the sitting position (Fleg et al., 1990b; for a review, see Fleg, 1986; Lakatta, 1993). The
reduction in early filling rate does not result in a reduced EDV because greater filling occurs in the atrial contraction (Arora et al., 1987; Chou et al., 1990; Kuo et al., 1987; Miyatake et al., 1984; Spirito et al., 1988; Swinne et al., 1992).

**End-systolic volume:** End-systolic volume is the remaining amount the blood (~40 to 50 milliliters) in each ventricle after contraction, just before it begins to refill. When normalized by surface area, it is called end-systolic volume index (ESVI). Resting ESV does not significantly change with age in healthy men but it tends to decrease slightly in healthy women (Fleg et al., 1990b).

![Diagram of Myocardial Inotropic Contractile Properties](image)

**Figure 2. Myocardial Inotropic Contractile Properties**
[Adapted from Physiology Medical Handout, 1996]

*Myocardial contractile property or inotropic state* is defined as strength of myocardial contraction or as a shift of the Frank-Starling curve either upward to the left (increased contractility or positive inotropic effect) or downward to the right (decreased contractility or negative inotropic effect) (see figure 1). A change in inotropic state means a greater or lesser force of contraction at a given end-diastolic volume (preload), resulting in a change in SV (more or less blood squeezed out). For example, using norepinephrine,
an increase on it affects the heart by increasing the force of contraction without any change in end-diastolic volume, exemplifying why catecholamines are said to have a positive inotropic effect (Foss & Keteyian, 1998).

Myocardial contractility is affected by intrinsic (the basic contractile properties inherent in the cardiac muscle itself), and extrinsic (response to conditions imposed from the outside) regulation of the myocardium. **Intrinsic regulation** involves: (1) myocardial response to stretching prior to contraction (i.e., to added preload stress such as increased diastolic filling, know as the Starling effect), (2) the myocardial response to increased load imposed after contraction has begun (i.e., to afterload stress, such as a rise in aortic diastolic pressure, and (3) myocardial response to heart rate alterations. **Extrinsic factors** that affect the contractility or inotropic state of the heart may be of three general types: (1) neurohumoral effects, due to influences of the sympathetic or parasympathetic systems or of the catecholamines; (2) chemical and pharmacological effects, for example, contractile changes due to alterations in blood K⁺, Ca²⁺, pH, or drugs such as digitalis and sympathetic “blockers”; and (3) pathological effects, for example, those due to ischemia incident to coronary occlusion or toxic effects resulting from bacteria or chemicals (Smith and Kampine, 1990).

Given the preceding situation, the intrinsic contractile behaviors of myocardial fibers might not be determined in situ, due to the complex interaction among multiple factors that regulate cardiac performance (For a review, see Fleg, 1986; Guyton & Hall, 1996; Lakatta, 1993). For example, any index of myocardial contractility, led to assess contractile properties of cardiac muscle among younger and/or older adults, requires specifying the degree of myocardial muscle tension when it begins to contract (preload),
and the load against which the myocardial muscle exerts its contractile force (afterload). Any index might be affected by these two factors that are not related to contractility.

Contractility may be (1) increased by an increased input pressure to the left ventricle (preload), and/or an increased pressure in the aorta (afterload), or (2) the effect of an intrinsic decline in contractility (without any change in ejection fraction) could be masked by either an increase in preload (i.e., the Frank-Starling mechanism), or a decline in afterload. However, it has been documented that the similarity in end-diastolic dimension and volume between younger and older individuals, and an increase rather than a decrease in afterload, may negate the possibilities of a difference in preload between these two groups, or a decreased afterload with advancing age. Additionally, the complexity of the control of contractility makes it difficult to interpret because these factors may differ from one person to another.

Although no satisfactory index of contractility totally independent of preload and afterload exists in man, a variety of methods have been designed to estimate myocardial contractility. Some of the more common measures of cardiac contractility are: ejection indices; ventricular dimensions and their rates of change; isovolumetric indices; and systolic time intervals. **Ejection indices**, which are based on the effectiveness of LV ejection, include aortic flow velocity, acceleration of blood in the aorta (dV/dt), and ejection fraction (the ratio of SV to EDV). **Ventricular dimensions and their rate of change** are estimated by ventricular angiography, radionuclide ventriculography, and echocardiography. **Isovolumetric indices** are based on the pressure rise during the isovolumetric contraction period, when the ventricle is a closed chamber and is undergoing isometric contraction (one of the more commonly used methods of these
indices is left ventricular dP/dt max). **Systolic time intervals** are based on the relative duration of the contraction and relaxation phases of the cardiac cycle and on the generalization that at a fixed HR the more competent ventricle will, if all other factors are held constant, eject a given SV in less time (i.e., have a shorter ejection period). These and other available indices can yield valuable information if they are used in carefully defined circumstances, providing that the techniques and their limitations are understood (Smith and Kampine, 1990).

Other derived indexes that have also been employed to examine myocardial contractility include the ratio of end-systolic pressure to ESV, the ratio of time to peak force and relaxation time, LV ejection time, ejection fraction, and velocity of fiber shortening. Regardless of the index employed, there is general agreement among the findings that there does not appear to be any indication of age-related changes in resting myocardial contractility. For example: in non-invasive studies in human, a crude index of the trajectory of ESV versus mean arterial pressure (i.e., the ratio of end-systolic arterial pressure to ESV) is not reduced at rest with age in either healthy men or women (Fleg et al., 1990b). This index of contractility, although under some conditions it is preload dependent, shares the limelight as being superior to others. It is referred to as E max, and derived from a series of pressure-volume loops measured over a range of cardiac volumes (Sagawa, 1978; 1981; for a review, see Lakatta, 1993). Studies in isolated cardiac muscle preparations indicated that the ability to generate force and the extent of sarcomere shortening during a contraction are not related to age, but a prolongation of contraction duration accompanies senescence in a wide variety of species, including man. The degree of prolongation averages 15 to 20% and is caused by an increase in both time to peak
force and relaxation time. However, long-term exercise eliminated this prolongation of contraction in senescent rats, demonstrating that this apparent age-related abnormality can be modified (Spurgeon et al., 1983). Studies in man have shown age-related increase in LV ejection time (Willems et al., 1970) as well as in the pre-ejection period (Shaw et al., 1973). Prolongation of contraction may reflect an age difference in the time course of contractile activation or in the excitation-contraction process, or aging changes in passive viscoelastic properties (For a review, see Fleg, 1986). Echocardiography and radionuclide ventriculography studies (relatively noninvasive, yet accurate, assessment of LV performance) report that ejection fraction (the most widely used index of pump function) is not age-related at rest, either in BLSA study volunteers (Rodeheffer et al., 1984) or in less rigorously screened, apparently healthy subjects (Port et al., 1980). This confirms prior M-mode echocardiographic investigations in BLSA study subjects and in other apparently healthy populations in whom shortening fraction (a 1-dimensional representation of ejection fraction) was independent of age (Gerstenblith et al., 1977; Gardin et al., 1979). The resting velocity of fiber shortening was unaffected by age in the BLSA study subjects (Gerstenblith et al., 1977). For example, velocity of fiber shortening (i.e., defined echocardiographically as the shortening fraction divided by ejection time), a “purer” index of cardiac muscle function than the ejection fraction, is thought to be relatively independent of preload, but it varies directly with HR and inversely with afterload. However, the maintenance of velocity of fiber shortening with advancing age cannot be explained by alterations in these variables, because HR at rest does not increase and afterload does not decrease with age in man. In summary, despite the difficulty of measuring intrinsic myocardial muscle performance or contractility in man,
there is no evidence for an age-related change in resting cardiac function in healthy people (For a review, see Fleg, 1986).

_Ejection fraction_. Overall systolic function of the heart, as a pump, is best judged from the measurement of the ejection fraction. Ejection fraction is the fraction of the EDV that is ejected with each systole (i.e., EDV-ESV/EDV). At rest, only about 55% to 60% of EDV is normally ejected during each ventricular systole. There is considerable agreement that this widely used index of pump function (ejection fraction) is not altered in healthy men or women at rest with age (Fleg et al., 1990b; Rodeheffer et al., 1984; for a review, see Lakatta, 1993). For example, ejection fraction was not age-related at rest, either in Baltimore study volunteers (Rodeheffer et al., 1984)) or in less rigorously screened, apparently healthy subjects (Port et al., 1980). This might suggest that prior M-mode echocardiographic investigations in BLSA subjects and in other apparently healthy populations in whom shortening fraction (a 1-dimentional representation of ejection fraction) was independent of age (Gardin et al., 1979; Gerstenblith et al., 1977).

_Afterload_ represents the resistance the heart meets as it attempts to pump the blood through the arterial tree into the circulation, and thus to the peripheral tissues. Any change in these blood vessels with advancing age might limit tissue perfusion and influence cardiac performance as well. For example, increased afterload (e.g., increased systemic blood pressure) has a negative influence on cardiac performance (e.g., decrease cardiac output) because it creates an increased workload for the heart. Thus, arterial blood vessels are the major determinant of impedance to LV emptying. Accordingly, the following section describes peripheral vascular changes associated with the aging process, with a particular emphasis on afterload.
2.1.2 Peripheral function / Afterload.

The peripheral vascular system has different functional parts, which include arteries, arterioles, capillaries, venules, and veins. The function of the arteries is to transport blood under high pressure to the tissues. For this reason, the arteries have strong vascular walls, and blood flows rapidly in the arteries. The arterioles are the last small branches of the arterial system, and they act as control valves through which blood is released into the capillaries. The arteriole has a strong muscular wall that is capable of closing the arteriole completely or allowing it to be dilated several-fold, thus having the capability of altering blood flow to the capillaries in response to the needs of the tissues. The function of the capillaries is to exchange fluid, nutrients, electrolytes, hormones, and other substances between the blood and the interstitial fluid. For this reason, the capillary walls are very thin and permeable to small molecular substances. The venules collect blood from the capillaries, and gradually become into progressively larger veins. The veins function as conduits for transport of blood from the tissues back to the heart, but equally important, they serve as a major reservoir of blood. Because the pressure in the venous system is very low, the venous walls are thin. However, they are muscular, and this allows them to contract or expand and thereby act as a controllable reservoir for extra blood, either a small or a large amount, depending on the body needs (Guyton & Hall, 1996).

The other main component of CV system involved in peripheral function is blood, a liquid tissue that has three general functions: transportation, regulation, and protection. The blood volume of an average-sized male is 5 to 6 liters; and an average-sized female has 4 to 5 liters, or 8% of total body weight. Blood is composed of two portions: (1) hematocrit or portion of the blood composed of blood cells and formed elements (red
cells, white cells, and platelets), that is 40% to 45% in males and 35% to 40% in females, and (2) plasma or liquid portion containing dissolved substances, is 55% of total blood volume (Foss & Keteyian, 1998; Smith & Kampine, 1990; Tortora, 1997).

**Age-related changes in the vascular system:** Age-related structural and functional changes have been reported in the peripheral vascular system (Bader, 1983; Fleg, 1986; Folkow & Svanborg, 1993; Gerstenblith et al., 1976; Lakatta, 1993; Seals, 1993; Seals et al., 1994b; Vaitkevicius et al., 1993). (See Table 2).

Studies in vitro and in vivo indicate an increase in the stiffness of large arteries with age (Amery et al., 1969; Aschoff, 1924; Avolio et al., 1983; Bader, 1967a, 1967b, 1983; Bramwell & Hill, 1922; Donatas et al., 1961; Fleg, 1986; Folkow & Svanborg, 1993; Freis et al., 1966; Gerstenblith et al., 1986; Gozna et al., 1974; Haynes et al., 1936; Lagrue et al., 1990; Lakatta, 1993; Nakashima & Tanikawa; 1971; Reneman et al., 1985; Reneman et al., 1989; Roach & Burton, 1959; Seal et al., 1999; Smulyan et al., 1983; Yakovlev, 1971). This age-related change means that the aorta and the arterial tree become thicker and less compliant with age. Accordingly, Bader (1967a) found that vascular stiffness, quantified in terms of its elastic modulus in autopsy samples, increases from 10 kg/cm\(^2\) at age 20 years to 42.5 kg/cm\(^2\) at age 85 years. This in-vitro work revealed that while the static circumferential modulus of the human aorta increased from 7.4 x 10\(^6\) dyn/cm\(^2\) in younger persons (<35 years of age) this value was 16.6 x 10\(^6\) dyn/cm\(^2\) in older adults (36-52 years of age) (Bader, 1967b).

Using non-invasive procedures (e.g., multigated pulse Dopplles system), pressure-strain moduli of both the aorta and pulmonary artery (measured during carotid artery
distensibility coefficient and cross-sectional compliance) is suggested to decrease with age between 20 and 80 years (Rich et al., 1984; Reneman et al., 1989; Lakatta, 1993).

**Table 2. Arterial changes with aging**

<table>
<thead>
<tr>
<th>Changes in arterial geometry and structure with aging</th>
</tr>
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<tbody>
<tr>
<td>Dilatation of the aorta and large arteries</td>
</tr>
<tr>
<td>Increase in arterial wall thickness</td>
</tr>
<tr>
<td>Increase in number of collagen fibers in the arterial wall</td>
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<tr>
<td>Decreased glycoprotein content and increased mineralization (Ca, PO$_4$) of elastin</td>
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<tr>
<td>Functional changes in arteries</td>
</tr>
<tr>
<td>Increased arterial stiffness, manifest as an increased elastic modulus of the arteries and increased arterial pulse-wave velocity</td>
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<tr>
<td>Increased arterial wall tension</td>
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<tr>
<td>Increased peripheral resistance</td>
</tr>
<tr>
<td>Alterations in arterial pressure and impedance</td>
</tr>
<tr>
<td>Increase in systolic pulse pressure</td>
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<tr>
<td>Increased mean arterial pressure</td>
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<tr>
<td>Increased in absolute amplitude of wave reflections</td>
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<tr>
<td>Decreased amplification of the pressure pulse between the ascending aorta and the peripheral arteries, partly due to reflected pulse waves</td>
</tr>
<tr>
<td>Alterations in aortic input impedance spectra</td>
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<tr>
<td>Increase in characteristic impedance</td>
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<tr>
<td>Increase in maximum-minimum impedance moduli</td>
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<tr>
<td>Shift of impedance modulus minimum and phase crossover to higher frequencies</td>
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<tr>
<td>Mismatch between aortic input impedance and energy of LV ejection wave</td>
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<td>Increased systolic pressure time index.</td>
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[Adapted from Lakatta, 1993]
Because arterial stiffness cannot be explained only on the basis of highly prevalent atherosclerosis in older adults (Bader, 1983; Dontas et al., 1961; Fleg, 1986; Folkow & Svanborg; 1993; Freis et al., 1966; Haynes et al., 1936; Lakatta, 1993; Lansing, 1959; Nakashima & Tanikawa; 1971), it has been suggested that age-related increases in arterial stiffness are the result of: (1) intrinsic or biochemical alterations in the wall of the arterial vessel (Harding et al., 1992; King, 1946; Kaplan & Meyer, 1960; Lansing, 1959; Roach & Burton, 1959; Robert et al., 1970; Seligman et al., 1975; Wolinsky, 1972), and (2) extrinsic or neurohumoral factors. The latter includes changes in catecholamine levels, represented by plasma norepinephrine (NE) (Esler et al., 1981; Goldstein et al., 1983a; Hoeldtke & Cilmi, 1985; Morrow et al., 1987; Rowe & Troen, 1980; Seals et al., 1994b; Ziegler et al., 1976) and epinephrine (E) concentrations (Barnes et al., 1982; Prinz et al., 1979; Vargas et al., 1986), and sympathetic nervous system (SNS) activity (Folkow et al., 1983; Goldstein et al., 1983b; van den Meiracker et al., 1989) (For a review, see Dempsey and Seal, 1995; Docherty, 1990; Lakatta, 1993; Seal, 1993).

Age-related intrinsic or biochemical alterations are referred to as changes in glycoprotein and amino acid components of the arterial vessel wall with age (Harding et al., 1992; King, 1946; Kaplan & Meyer, 1960; Lansing, 1959; Roach & Burton, 1959; Robert et al., 1970; Seligman et al., 1975; Wolinsky, 1972). For example: (1) chondroitin sulfate and heparin sulfate increase, hyaluronate and chondroitin content decrease, and total mucopolysaccharide content is unaltered (Kaplan & Meyer, 1960); (2) the relative loss of elastin and an increase of collagen (Roach & Burton, 1959), which have been interpreted to be consistent with alterations in stress-strain curve of aged vessel in vitro. Chemical analysis has indicated that this is the case (Faber & Moller-Hou,
1952); (3) the change in the distribution of unstretched collagen that has been proposed to be involved in a decrease in the recoiling and twisting of molecular chains, and a reduction in effective chain length (Harding et al., 1992; King, 1946); and (4) the glycoprotein component of elastin fibrils decreases and eventually disappears (Robert et al., 1970). This becomes Elastin (in rats) frayed (Wolinsky, 1972), and its Ca$^{2+}$ content increases (Lansing, 1959). Thus, the age-related increase in mineralization (Ca$^{2+}$, phosphorus) of elastin has been associated with an increase in the content of amino acids (Seligman et al., 1975).

The other suggested determining-factors of age-related arterial stiffness are the changes in plasma NE and E (catecholamine) concentrations, and sympathetic nervous system (SNS) activity. In humans, plasma NE concentration, widely used as an estimate of SNS activity (Folkow et al., 1983; Goldstein et al., 1983b; van den Meiracker et al., 1989) has been reported to be elevated at rest, even in healthy older adults free of overt CVD (Esler et al., 1981; Goldstein et al., 1983a; Hoeldtke & Cilmi, 1985; MacGilchrist et al., 1989; Morrow et al., 1987; Rowe & Troen, 1980; Seals et al., 1994b; Ziegler et al., 1976). In contrast, plasma levels of E have not been consistently shown to increase with age in humans. Reports have indicated either no change (Barnes et al., 1982; Prinz et al., 1979; Vargas et al., 1986) or increased plasma concentrations (Fleg et al., 1986). In the rat, plasma levels of NE have been also reported to increase with age (Hoffman et al., 1985).

Plasma NE differences between young and older adults are difficult to interpret due to the combined factors (NE spillover into and clearance from the vascular compartment, and regional sympathetic outflow) that influence these concentrations. Increased plasma
level of NE can be due to an increased rate of NE spillover into the vascular compartment (rate of appearance) or to a decreased NE clearance from the vascular compartment (rate of clearance), or both. The rate of NE spillover into the plasma has been reported to increase with age in humans (Hoeldtke & Cilmi, 1985; MacGilchrist et al., 1989; Morrow et al., 1987; Rubin et al., 1982; Veith et al., 1986), and age and percentage of body fat have been reported to be independent determinants of NE spillover (Schwartz et al., 1987). The rate of NE clearance from the plasma has been variously reported to be unchanged (Young et al., 1980; Rubin et al., 1982) or decreased (Esler et al., 1981; Hoeldtke & Cilmi, 1985; Morrow et al., 1987) by age in humans.

Although these data on plasma NE spillover may be influenced by age-related differences in prejucional modulation of NE release (Bucholz & Piper, 1990) and/or synaptic reuptake, it is suggested that the weight of the available information appears to be consistent with the idea of elevated systemic SNS activity with advancing age in humans (Dempsey and Seal, 1995; Docherty, 1990; Lakatta, 1993; Seal, 1993). However, it should be noted also that later studies, in which non-age-related factors were rigorously controlled, have failed to demonstrate similar age-related differences in plasma NE concentration (Fleg et al., 1985; Taylor et al., 1991, 1992). Thus, these latter findings suggest that plasma NE concentration is not age-related. Inasmuch as an altered plasma clearance is expected to affect plasma NE and E equally, it seems that the important age-related alteration might be an increased rate of NE spillover into the plasma, due either to increased neurotransmission or to a decreased function of the NE reuptake process. An increased neurotransmission with age seems unlikely because, at least in the rat,
catecholamine content of tissues is generally reported to decrease with age (Docherty, 1990).

Lastly, it should be noted that sympathetic outflow might not be necessarily uniform to all regions or tissues of the body (Folkow et al., 1983; Goldstein et al., 1983b; Hjemdahl et al., 1984). Thus, the exact source of the elevated activity remains to be determined. Evidence from studies using direct neural recording indicates that skeletal muscle blood vessels are one target of heightened sympathetic discharge in healthy older adults (Sundölf & Walling, 1978; Morlin et al., 1983; Ng et al., 1993).

In addition to structural properties, arterial stiffness in vivo is determined by vascular smooth muscle (VSM) contractile tonus, which is controlled, in part, by neurohumoral factors, e.g., catecholamines and angiotensin (Peterson et al., 1960; Remington, 1963; Safar et al., 1987; Simon et al., 1984; Ting et al., 1986). For example, the increased arterial stiffness in older patients with cardiomyopathy can be reduced by the vasodilator nitroprusside. This effect has been found to be greater in older than in younger individuals (Carroll et al., 1991). Thus, it is suggested that a component of the increased in-vivo arterial stiffness with age might be due to augmented VSM.

Lastly, it has been suggested that arterial wall smooth muscle might become less sensitive to β-adrenergic stimulation (due to a decreased in the β-adrenergic sensitivity or number of β-receptors), and that alpha-adrenergic responsiveness of the vasculature appears to remain intact (Lakatta, 1986). As a result, the balance between alpha and beta adrenoceptor function changes, the peripheral vasculature leans toward vasoconstriction and, consequently, there is an increased peripheral vascular resistance (Vanhoutte, 1988). The smaller radius of the constricted arterioles provides a smaller tube through
which blood must be pumped and consequently increases resistance to the pumping heart. It is estimated that total peripheral resistance increases about 1% a year with advancing age, even in the absence of coronary artery disease. It has been suggested that this is due partly to an increased rigidity of arterial vessels and partly to decreased vasodilation. In summary, arterial stiffness is a complex condition that is determined by biochemical alterations of the arterial vessel wall, and changes in the plasma NE concentration and sympathetic nerve activity. Regardless of the relative contributions of these possible causative factors, age is associated with increases in the peripheral vascular resistance and, therefore, blood pressure.

Age-related changes in the arterial stiffness are accompanied by some other vascular system changes, including: (1) an increase in arterial diameter and wall thickness in both humans and rats (Cliff, 1970; Ensor et al., 1983; Nichols et al., 1985; Wolinski, 1972). Nichols et al (1985) found that the mean systolic internal radius of the ascending aorta in humans increases 9% per decade over the age-range of 20-60 years. Ensor et al. (1983), using chest X-rays in the same previously cited study from BLSA, found that the aortic knob diameter increased from 3.4 to 3.8 cm. This last finding agrees with the age-related increase in aortic dimension noted in echocardiographic cross-sectional studies. (2) In rats, a decrease in the average number of nuclei in the aortic wall (Cliff, 1970), which is similar to that in the myocardium in humans; (3) No age-related change in the aortic buffering capacity, up to the age of 60 years. It is suggested that this buffering capacity is not markedly diminished by increased aortic wall stiffness because of a compensatory increase in aortic volume. Thus, any given of volume injected into it is accommodated with less change in radius (Smulyan et al., 1983). It has been estimated that the thoracic
aorta provides about one-half of the total blood volume buffering capacity of the arterial system (Bader, 1967); (4) No age-related change in volume elasticity (change pressure for a given volume change). The volume elasticity measured in vitro shows no age-associated changes up to the age of ~60 years. It is suggested that it is due to the large increases in aortic volume up to that age (Bader, 1967); (5) A decreased aortic contribution to forward flow. As the aorta stiffens with age, less diastolic aortic recoil occurs and results in a decreased aortic contribution to forward flow; (6) Age-related changes in diameter and thickness in more peripheral vessels (Busby & Burton, 1965; Carter, 1964; Landowne, 1957; Roach & Burton, 1959). These changes have been characterized in that the increase in diameter of these vessels (peripheral vessel) is less, but the increase in wall thickness is greater than that of the aorta (Roach & Burton 1959). For example, in individuals who have no symptoms of peripheral vascular disease, the pressure-strain elastic modulus of the femoral artery, measured noninvasively via an ultrasonic tracking system and auscultatory pressure, increases greater than two-fold throughout the lifespan (Mithoefer & Karetzky, 1968). It should be noted, however, that considerable variation has been reported within and among age groups (For a review, see Fleg, 1986; Lakatta, 1993). The difference in these results might be attributed to gender, physical fitness level or health status differences among the individuals participating in those studies.

It has been documented that the age-related increase in arterial stiffness is also reflected in an increase in the pulse-wave velocity (Astrad et al., 1973; Bramwell & Hill, 1922; Fleg et al., 1990ba; Freis et al., 1966; Haynes et al., 1936; Vaitkevicius et al., 1991; Yakovlev, 1971). It has been suggested that the age-related increase in aortic pulse-wave...
velocity causes waves to be reflected from peripheral sites to the ascending aorta earlier in older than in younger subjects. Reflected pulse waves merge (or sum) with the incident (or forward) waves generated by LV ejection and influence the contour of measured pressure and flow waves (Milnor, 1982; Murgo et al., 1980 & 1981; Nichols et al., 1985 & 1987; O’Rourke, 1982a).

Changes of the pressure pulse contour with age have been widely described and associated with age-related changes in the vascular system (Dontas, Taylor & Keys, 1961; Freis et al., 1966; Kelly et al., 1989; Murgo et al., 1980 & 1981; Nichols et al., 1985; O’Rourke, 1982b; Vaitkevicius et al., 1991). For example, age-related changes of the pressure pulse contour include a large secondary systolic wave (dicrotic wave) in older individuals and the appearance of the diastolic pressure wave (O’Rourke, 1982b). As measured from a piezogram of the carotid pulse, it has been demonstrated that the duration of the dicrotic wave is related to arterial stiffness, correlated with the pulse-wave velocity, and decreases with advancing age (Lagrué et al., 1990). These altered features of the central arterial pulse pressure have been attributed to early-reflected pulse waves (O’Rourke, 1982a). Thus, it is suggested that the early reflected-pulse waves are the primary determinant for the long recognized increase in systolic and pulse pressures with aging (Kannel et al., 1981; Merillon et al., 1982; Murgo et al., 1980 & 1981; Nichols et al., 1985; O’Rourke, 1982a). Therefore, it has been also suggested that changes in the pressure pulse contour of central arteries are related not only to the stiffness properties of the large vessels, but also to the properties of the small resistance vessels that determine the total peripheral resistance (TPR). For example, amplification of the late systolic peak in the pressure pulse contour is produced by agents that increase
TPR (e.g., angiotensin), and reduced by vasodilator drugs (e.g., amyl nitrate), presumable by dilation of smaller arteries (Fitchett et al., 1988; Kelly et al., 1990; Lakatta, 1993).

It should be noted that, in addition to causing a late increase in systolic blood pressure (e.g., due to earlier reflected pulse waves), the stiffening of the large arteries (with its concomitant increase in pulse-wave velocity) ought to lead to a reduction in diastolic pressure. This is because the reflected pulse wave usually returns after aortic valve closure (O’Rourke et al., 1982b). However, diastolic blood pressure (DBP) has been observed to increase rather than decrease in older adults. This observation has been reconciled by the observation that TPR increases with age. This is particularly evident in individuals with clinical hypertension, (Kawamoto et al., 1989; Lund-Johansen, 1991; Messerli et al., 1981 & 1984) but less so among older normotensive subjects, where a great deal of heterogeneity in TPR has been observed (Brandfonbrener et al., 1955; Fleg et al., 1990b; Julius et al., 1967). In healthy sedentary men, TPR, estimated in the sitting position at rest, does not increase with age (Fleg et al., 1990b).

Mechanisms mediating the extent to which the TPR is increased in older individuals are not presently well defined but include wide variations of basal CO and heterogeneities among older individuals in the age-related decreases in skeletal muscle mass, basal metabolic rate, and capillary density. For example, although renal blood flow per gram decreases progressively after the fourth decade (cortical flow decreases to a greater extent than medullary flow; see Lindeman & Goldman, 1986), the decline is due to renal vasoconstriction and not attributable to a reduced CO (Danzinger et al., 1990). Thus, while the nature of this increase in renal vascular resistance with aging is incompletely understood, there is presently no conclusive evidence that renal ischemia is

244
a cause of the age-associated changes in renal structure or of the increase in TPR in some elderly normotensives (For a review, see Lakatta, 1993).

Finally, it should be noted that, even though the plentiful documented increase in arterial stiffness and pressure with age, these age-related changes appear to be modified by life-style and diet. For example, (1) the NaCl dependence of arterial pressure increases with age, manifested by an increase in the pulse-wave velocity, differs in two Chinese populations (Avolio et al., 1985), in which exercise and dietary habits are different. These two populations differ in dietary habits, specifically in reference to the amount of NaCl ingested. (2) The expected age-associated increases in aortic, arm, and leg pulse velocities, in a study population advised to ingest low quantities of NaCl (44 mmol/24hr) for an average period of 2 years, did not occur. (Avolio et al., 1986). (3) The age-associated increases in pulse-wave velocity (i.e., carotid pressure pulse late augmentation) are blunted in exercise-trained older athletes (Haber et al., 1984; Vaitkevicius et al., 1991). (4) The age-related elevation in 24-h SBP and pulse-pressure (PP) found in sedentary adult females did not occur in women who regularly perform endurance exercise (Seal et al., 1999). (5) The arterial stiffness, in a healthy sedentary population of a broad age range, varied inversely with aerobic capacity (Vaitkevicius et al., 1991). This inverse relationship between arterial stiffness and aerobic capacity occurred over and above the effects of age to increase the former and decrease the latter (Vaitkevicius et al., 1991). (6) It has been demonstrated that chronic exercise can decrease the TPR in elderly individuals (Matin et al., 1990); thus the increase in TPR might not be directly related to changes with age (For a review, see Lakatta, 1993; Spirduso, 1996).
Cardiovascular-acting hormones: This issue has been previously reviewed (Docherty, 1990) and a detailed explanation is not within the scope of this review. However, the most relevant reported findings in the cardiovascular-acting hormone are summarized herein. In general, no consistent overall pattern has emerged with regard to possible age-related effects in the circulating levels of cardiovascular-acting hormones (epinephrine, renin-angiotensin-aldosterone system, Vasopressin, and atrial natriuretic peptide). For example: (1) Plasma epinephrine concentration. Generally, there are no significant differences in plasma E concentration in young and older humans at rest (Esler et al., 1989; Morrow et al., 1987; Ziegler et al., 1976), with reports of no change (Barnes et al., 1982; Prinz et al., 1979; Vargas et al., 1986) or increased levels (Fleg et al., 1985). Therefore, it is suggested that any particular concentration of this hormone in the plasma appears to have less of a target tissue effect due to impaired beta-adrenergic responsiveness (Docherty, 1990; Fleg, 1986; Gregerman, 1986; Nielsen & Vestal, 1989).

(2) Renin-angiotensin-aldosterone system. Angiotensin is involved both in the short-term control of blood pressure (BP) and, together with aldosterone, in the long-term control of blood volume. In the short-term control of BP, angiotensin acts both as a direct vasoconstrictor and to facilitate vascular contractions particularly involving α2-adrenoceptors (Palluk et al., 1985) and to facilitate adrenergic vasoconstriction by a prejunctional action (Gothert and Kollecker, 1986). Although the vasoconstrictor response to angiotensin II is reported to be unchanged by age in the human vasculature, the activity of the renin-angiotensin-aldosterone system is reported to be reduced in older adults in terms of morning plasma levels of renin and aldosterone activity (Crane, & Harris, 1976; Tuck et al., 1873; Korkushko et al., 1984; James et al., 1986) and in terms
of 24-h tonic and phasic levels (Cugini et al., 1987). That is, plasma levels of renin, angiotensin II, and aldosterone appear to decline progressively from birth, especially after the age of 40 (Crane & Harris, 1976; Haller et al., 1987; Noth et al., 1977; Skøtt & Giese, 1983; Tsunodo et al., 1986). As a result, it has been suggested that the ability of angiotensin-converting enzyme inhibitors to decrease BP is diminished in older adult hypertensive due to lower plasma renin levels (Buhler et al., 1980). However, other studies have found angiotensin-converting enzyme inhibitors equally effective in older adult hypertensives (Baker, 1988; Cummings et al., 1989; Jenkins et al., 1985) perhaps because elderly hypertensives have increased plasma renin levels making them comparable to young hypertensives (Chebotarev et al., 1985; Cummings et al., 1989). More information about differences in this renin-angiotensin-aldosterone system by tissue, and the co-action of angiotensin II along with other vasoconstrictor are out the scope of this review. For more specific information about it the reader is referred to more specific review such as Docherty, 1990.

(3) **Vasopressin.** Although vasopressin (arginine vasopressin) has vasoconstrictor actions, its primary role is BP homeostasis by control of blood volume, and hence it is also known as antidiuretic hormone. The secretory capacity of vasopressin is reported to be unchanged or even increased during normal aging in humans (Helderman et al., 1978; Kirkland et al., 1984) and experimental animal (Fliers & Swaab, 1983; Froikis et al., 1982; Miller, 1987; Davies et a., 1980), and basal circulatory levels of vasopressin are increased in the elderly (Kirkland et al., 1984; Rondeau et al., 1982; Vargas et al., 1986; Davis, & Davis, 1987). However, increased vasopressin in older adults is offset by a
decreased response of collecting tubules to vasopressin both in humans (Davis & Davis, 1987) and the rat (Miller, 1987; Phelps et al., 1989).

(4) Atrial natriuretic peptide (ANP). Even though, atrial natriuretic peptides also produce vasodilation, the major role of the ANPs (atriopeptins) from the mammalian atria is in the hormonal control of blood volume by causing sodium and water excretion. Infusion of physiological concentration of ANP causes increased sodium excretion without affecting BP in humans (Anderson et al., 1987), but higher pharmacological concentrations decrease BP (Ferrari et al., 1990). It is thought that ANP is mainly released in response to atrial stretch, and hence levels are reported to be elevated in chronic heart failure (Katoh et al., 1986; Schiffrin, 1988). In humans, plasma levels of ANP are reported to increase with age (Duggan et al., 1988; Haller et al., 1987; Montorsi et al., 1987; Portaluppi et al., 1991; Singer et al., 1987). Despite increased resting plasma levels of ANP, older adults have reduced ability to concentrate urine and excrete a saline load (Davis & Davis, 1987), at least partly due to the 50% decrease in renal blood flow and glomerular filtration rate which occurs during aging (Schmuker, 1985). However, although older adults respond to a saline infusion with a larger absolute increase in plasma ANP, the increase is smaller than that occurring in a younger group when expressed as a percentage increase over basal levels (Haller et al., 1987). The higher plasma level of ANP found in older adults might be due to decreased renal responsiveness to ANP or to decreased plasma clearance or to the increased atrial pressure reported in older adults (Ehram et al., 1983), but there is no information concerning the effects of aging on this interaction, given that the activity of the renin-angiotensin-aldosterone system declines with age. In conclusion, the influence, in most
cases, of these age-related cardiovascular-acting hormones changes on target tissue or CV function in humans remains to be determined (Docherty, 1990).

**Peripheral circulation or regional hemodynamic.** With respect to regional hemodynamic, it has been demonstrated that blood flow (BF) is reduced and TPR increased in several regional circulations (Folkow & Svanborg, 1993; Seals, 1993). However, other studies (e.g., myocardial BF studies) have reported that BF increases with age (Czernin et al., 1993). The increases in vascular resistance, systemically and in select regions, are likely mediated by a combination of structural and neurohumoral mechanisms (Bader, 1983; Fleg, 1986; Folkow & Svanborg, 1993; Gerstenblith et al., 1976; Lakatta, 1993; Seals, 1993; Seals et al., 1994b; Vaitkevicius et al., 1993). As reported previously, this combination of structural and neurohumoral mechanisms might be a result of age-related structural changes in the walls of arterial vessels (Bader, 1983; Fleg, 1986; Folkow & Svanborg, 1993; Gerstenblith et al., 1976; Lakatta, 1993), changes in plasma NE concentrations (Esler et al., 1981; Goldstein et al., 1983a; Hoeldtke & Cilmi, 1985; Morrow et al., 1987; Seals et al., 1994b; Ziegler et al., 1976), and changes sympathetic nerve activity to skeletal muscle (Morlin et al., 1983; Ng et al., 1993; Seals et al., 1994b; Sundlof & Wallin, 1978). However, the respective contributions of these and other neurohumoral changes, and structural changes in the age-related increases in vascular resistance and arterial pressure are not completely known. Therefore, the possible role of the age-related well-documented impairments in arterial and cardiopulmonary baroreceptor function has not been determined (Cleroux et al., 1989; Gribbin et al., 1971; Lindbald, 1977; for a review, see Dempsey and Seals, 1995).
Even though the information concerning the effect of advanced age on regional BF at rest, in some cases, is not completely conclusive, decreased BF and increased levels of vascular resistance with advancing age have been reported in some specific regional circulation. For example, decreased BF and increased level of vascular resistance have been documented in the hepatic (Leithe et al., 1984; Skauric et al., 1970), renal (Davies & Shock, 1950; Landowne & Shock, 1951; Leithe et al., 1984; Lewis & Alving, 1958), and cerebral (Fazekas et al., 1952, 1953; Martin et al., 1991; Scheinberg & Stead, 1949) circulations. Therefore, while BF data in skeletal muscle or skin are not available per se, whole forearm BF has been reported to be higher (Hellon & Clarke, 1959), not different (Hellow et al., 1956; Lind et al., 1970), and lower (Leither et al., 1984; Sagawa et al., 1988) in older compared to young humans. No age-related differences have been observed for the hand (Duff, 1956), foot (Allwood, 1958) or whole calf BF (Allwood, 1958; Taylor et al., 1991). In all these studies, except one (Taylor et al., 1991), limb vascular resistance was higher in the older subjects. Thus, it should be noted that BF was maintained at the same level as young subjects despite a higher arterial (perfusion) pressure created by an elevated resistance (For a review, see Dempsey and Seals, 1995; Seals, 1993).

Studies using venous occlusion plethysmography have variously reported a trend for a reduction (Leithe et al., 1984; Seals, 1994), no difference (Allowood, 1958), or an increase (Hellon & Clarke, 1959) in some regional circulations (forearm or calf BF) with age in adult humans. However, the subjects studied in some of these earlier investigations might have included older adults with CVD, which may explain the difference in these results. Therefore, questions as to whether this technique can be used to measure absolute levels of limb blood flow have been raised.
Other studies, for example those using Doppler ultrasound procedure to test the effects of age on absolute levels of resting limb flow in healthy humans, have found that whole-limb BF was 26% lower under resting conditions in healthy adult men (mean age=63 years) as compared with young (in their 20s) adult men (Dinenno et al. (1999). In this study, the lower whole-limb BF was not associated with a reduction in systemic arterial flow (cardiac output), but it was to a lower (32%) vascular conductance and a higher (45%) vascular resistance. Both lower vascular conductance and higher vascular resistance were associated with tonically elevated (75%) sympathetic vasoconstrictor nerve activity. Therefore, the lower limb BF was directly related to a lower (15%) estimated oxygen demand, which was independent of tissue mass (Dinenno et al., 1999).

In summary, it appears to be difficult to establish a conclusive age-related limb and regional BF pattern so far, because of the multiple factors implied in BF control and regulation, differences in limb and regional circulation, and differences in the procedures and protocol used. Therefore, it has been suggested that the influence of other determining factors (e.g., physical fitness level, health status, gender differences, body weight and composition, and/or body position during the test) requires more study.

**Blood volume and blood constituents.** Blood volume and blood constituent studies, in some cases, have reported that age-related changes in hematocrit and hemoglobin are very small and gradual. For example, Bowdler and Foster (1987) found that aging is associated with a slight but significant, gradual decline in hematocrit, beginning in the relatively early decades; Timiras and Browntstein (1987) found that mean hemoglobin levels were maintained in healthy aging individuals until about 85 yrs of age, at which time the level decreased slightly; and anemia has been reported to be prevalent among
older individuals, especially those over age 70. However, whether the decline in hematocrit and hemoglobin values is a function of aging or of other, more indirect factors remains to be determined. In a review of 1,024 geriatric screening charts, 17.7% of males and 8.4% of females were classified as anemic (Timiras & Brownstein, 1987). The major causes for anemia found in older adults are: (1) change in diet (due to a decrease in total calories per day or changes in types of food consumed), (2) the loss of appetite, impaired mobility to obtain groceries, reduced income to purchase food, and/or lowered capacity to cook, especially those living alone, and (3) age-related diseases, that might contribute to anemia condition (unrecognized internal bleeding or deterioration of gastrointestinal absorption of iron or vitamin B₁₂). However, some evidence suggests that mean hemoglobin level are not different in the elderly, except in males over 85, and should not be expected to change with advancing age (For a review, see Spirduso, 1996).

**Systolic and diastolic blood pressure.** It has been documented in human that arterial BP increases with advancing age in the overall population (Harlan et al., 1984; Harris et al., 1985; Lakatta, 1990; Schoenberger, 1986; Sowers, 1987). However, BP change might not be a purely age-related phenomenon because: (1) it does not increase with advanced age in some, usually small, isolated, communities (Docherty, 1990; Kotchen et al., 1982; Marmot, 1984; Ikeme, 1989); (2) later laboratory studies of resting hemodynamics in healthy older adults have reported little or no age-related differences (Rodeheffer, 1984; Seals et al., 1985; Taylor et al., 1991); (3) in studies comparing sedentary and endurance-trained older adults, the increase in resting BP does not occur in the trained subjects (Seal et al., 1999); and (4) in healthy, active, non-obese, older individuals elevations in mean
arterial pressure are either not observed or are relatively minor (Ng et al., 1993; Rodeheffer et al., 1984; Seals et al., 1985; Taylor et al., 1991).

All above documented reasons might suggest that the aging process per-se is not responsible for the increase of arterial pressure observed in the overall population. (Fleg, 1986; Folkow & Svanborg, 1993; Gerstenblith et al., 1976; Lakatta, 1993; Seals, 1993). Thus, if there is a direct effect of the aging process, it is probably confined primarily to a rise in systolic blood pressure (SBP), most likely because of structural changes in the arterial blood vessels (described previously) (Bader, 1983; Fleg, 1986). Therefore, the available data on cardiac output indicate that any rise in arterial pressure with advancing age is mediated by an increase in TPR (reduction in total vascular conductance) (Fleg, 1986; Folkow & Svanborg, 1993; Gerstenblith et al., 1976; Lakatta, 1993; Seals, 1993; Dempsey and Seals, 1995; Spirdusso, 1996).

In addition, studies on gender-related differences have documented that the primary effect of aging (a mild increase in SBP) has been especially found in women (Ng et al., 1993; Seals, 1993), and that the rate of rise of mean arterial BP with advancing age is especially accelerated after menopause (Folkow & Svanborg, 1993; Harlan et al., 1984; Imai et al., 1993; Schoenberger, 1986). However, in a study comparing healthy-normotensive premenopausal and postmenopausal sedentary, and premenopausal and postmenopausal endurance-exercise trained women, it was found that 24-h SBP and PP were higher (~10mmHg) in postmenopausal women than in premenopausal women in the sedentary group, but in the endurance-trained group there were not different with age. Thus, it is suggested that the elevation in 24-h SBP and PP with age in sedentary adult females might not occur in women who regularly perform endurance exercise. Thus, the
age-difference in BP in women might not be related to the aging process per-se, suggesting for more studies to bring out the direct reasons of this difference.

Resting BP is influenced by neurohumoral and reflex control, and by structural and pathological changes in the cardiovascular system. Some of these factors such as the sympathetic nervous system and the baroreflex are involved mainly in short-term control of BP, whereas other factors such as aldosterone and vasopressin are involved mainly in long-term control of BP regulating blood volume. Levels of circulating hormones can affect BP in the short-term (by direct vascular actions and by indirectly influencing responses to other circulating agents) and in the long term (by altering blood volume). Thus, it is worthwhile to consider how aging affects blood levels of hormones (see cardiovascular-acting hormone, page 43). For example, plasma levels of NE are increased by age, but it is known that these increased levels do not have any significance on cardiovascular system performance. Other hormones, such as angiotensin and arginine vasopressin (i.e., vasoconstrictor agent), and the vasodilator agent ANP, are involved in the long-term control of BP. Because the majority of control system, both long and short term, act to increase BP, it would appear that these systems have evolved mainly to maintain flow to vital organs in hypotensive situations and this may explain the tendency for increasing BP with age.

*Autonomic Reflex-Decline in Baroreceptor Reflex Function.* Homeostatic reflexes have evolved particularly to maintain blood flow to vital organs in hypotensive situations. The most important short-term autonomic CV reflex is the baroreflex mediated by the baroreceptors in the aortic arch and carotid sinus. The baroreceptors are mechanoreceptors that respond to changes in BP. They project to the nucleus tractus
solitarius in the dorsal medulla, which has synaptic connections with interneurons that inhibit the vasomotor center and stimulate the vagal cardioinhibitory center. Baroreflex function can be altered pathologically at a variety of levels: altered sensitivity of baroreceptors to changes in BP, altered central integration, altered efferent nerve activity in both sympathetic and parasympathetic nerves, and alterations in cardiovascular receptors. The baroreflex responds rapidly to maintain BP near normal but quickly adapts so that the reflex begins to fade after a few hours. Long-term control of BP is mainly under renal control, involving various systems including the renin-angiotensin-aldosterone system and vasopressin.

The baroreflex can be assessed in terms of the reflex bradycardia to a pressor agent or the reflex tachycardia to a depressor agent. The vagal component of the baroreflex bradycardia to the \( \alpha \)-adrenoceptor agonist phenylephrine is reported to increase (Lo et al., 1988; Su et al., 1986) or even to decrease (Tanabe & Bunag, 1989) during maturation and to decrease with age (Rothbaum et al., 1974) in rat. In humans the vagal reflex of the Valsalva maneuver is decreased by aging (Oimomi et al., 1986), as is the bradycardia to phenylephrine (Gribbin et al., 1971; Yin et al., 1978a; Elliot et al., 1982).

It has been widely reported that the baroreflex tachycardia to depressor agents or to postural change decreased with aging in human (Elliot et al., 1982; McGarry et al., 1983; Collins et al., 1980; Dambrink & Wieling, 1987; Simpson & Wicks, 1988) and in rats (Docherty et al., 1986), although not in the anaesthetized dog (Cox et al., 1981). Furthermore, the reduction of baroreflex function with age in nonhypertensive subjects was independent of BP level, so that age is related to altered baroreflex sensitivity independent of BP (Shimada et al., 1986). Similarly, the sympathetic response to cold
and heat stress is reduced in aged rats (McCarty, 1985) and humans (Collins et al., 1980), although this may be partly due to a reduced response of microvessels because capillary blood flow is reduced in the nailfold circulation of the elderly (Richardson & Schwartz, 1985). In individual studies, heart rate variability decreases with age in humans (Maclennan et al., 1980; Mancia et al., 1983; Cinelli et al., 1987; Simpson & Wicks, 1988), and the difference from young was more marked in the standing position (Simpson & Wicks, 1988). Even when comparing studies, the variation between studies in HR for the aged is less than that for the young, both in humans and conscious, but not anaesthetized, rats. Likewise, respiratory sinus arrhythmia decreases with aging (Hellman & Stacy, 1976). The older adults respond to postural stress with diminished changes of HR and DBP even in the absence of postural hypotension (Collins et al., 1980; MacLennan et al., 1980; Williams et al., 1985; Vargas et al., 1986; Smith et al., 1987; Dambrink & Wieling, 1987).

**Autonomic reflex-Postural hypotension:** Postural hypotension is reported to be common in older adults, with an overall incidence of 22% (MacLennan et al., 1980) or 24% (Caird et al., 1973) in younger adults; and an incidence of 24% (MacLennan et al., 1980), 30% (Caird et al., 1973), or 30-50% (Robbins & Rubenstein, 1984) in those >75 years of age, but other studies report a prevalence as low as 10.7% (Mader et al., 1987). Prevalence of postural hypotension was found to be as low as 6.4% in a group in which known risk factors for postural hypotension were excluded (Mader et al., 1987). However, various factors can explain differences between studies in incidence of postural hypotension, including day to day (Lipsitz, 1989), time of day, and time of measurement after standing variability (MacRae & Bulpitt, 1989). In older adult subjects with postural
hypotension a failure to increase peripheral resistance sufficiently (Williams et al., 1985) or arterial rigidity (MacLennan et al., 1980) have been implicated. Postprandial hypotension is also reported to be a problem in older adults both in a normal population (Lipsitz et al., 1983) and in a population with orthostatic hypotension (Robertson et al., 1981). Older adult subjects respond to the postprandial decrease in SBP with insufficient tachycardia and therefore failed to compensate adequately for the pooling of blood in the splanchnic circulation during digestion (Docherty, 1990).

Age-related changes in neural control mechanisms of the arterial tree are also seen in postural hypotension, which is a loss in responsiveness of homeostatic reflexes. Postural hypotension is fairly common in the elderly, ranging from 22% to 30% in the young elderly and 30% to 50% in those over 75 years of age (Docherty, 1990). However, Bennett and Gardiner (1988) suggests that if the most rigorous standards are applied to the available literature on the subjects, the substantive evidence is weak for “well-known” age-related changes such as baroreflexes, sympathetic hyperactivation, adrenoreceptor dysfunction, and neurohumoral abnormalities (For a review, see Spirduso, 1996).

It is not know whether any of the age-related autonomic-circulatory changes described above are associated with altered baroreceptor function. Arterial and cardiopulmonary baroreflex control of HR and TPR may be impaired in older humans (Cleroux et al., 1989; Gribbin et al., 1971; Lindblad, 1977). However, arterial baroreflexes are thought to be primarily responsible for regulating moment-to-moment fluctuations in arterial BP (Mancia & Mark, 1983); thus, their role in determining chronic levels of arterial pressure via their modulating effects on the above mentioned
neurohumoral mechanisms is not clear. Moreover, the contribution of altered cardiopulmonary baroreflexes (which monitor cardiac filling pressure and thus, indirectly, central blood volume [Mark & Mancia, 1983]), to age-related changes in sympathetic outflow, TPR, and arterial pressure has yet to be determined in the human (For a review, see Seals, 1993).

In summary, resting HR, LV EDV and contractility are similarly unaffected by aging in man. Resting CO does not appear to decline with advancing age in normotensive, active community dwellers that are free of CAD. Under resting conditions, aging is associated with a rise in arterial blood pressure due to an increase in TPR. The specific regions that contribute to the elevation in TPR have not been ascertained, but may include the renal, hepatic, and cerebral circulations, and perhaps skeletal muscle and/or skin. Although afterload increases modestly with age, it appears to be that the compensatory LV hypertrophy might allow maintenance of normal wall stress and normal systolic function. A significant age-related retardation of LV filling rate and prolongation of isometric relaxation do not impair resting cardiac performance. There is no compelling evidence that LV contractility declines with age (e.g., ejection fraction is not altered). However, there are several other age-related left ventricular changes, including increases in afterload, wall thickness, and overall mass, and a reduction in peak diastolic filling rate. Arteries increase in diameter and wall thickness with age, and these changes are associated with an increase in arterial wall stiffness and a reduction in volume elasticity. The elevations in regional TPR may be mediated in part by elevated sympathetic vasoconstriction outflow and release of norepinephrine, although age-related structural alterations in the arterial system probably also contribute. However, the TPR
does not markedly increase with aging in healthy normotensive individuals. No obvious role has been identified for age-associated changes in circulating vasoactive hormones in the elevations in vascular resistance and arterial pressure. The extent to which some of these changes occur with age appears to be modified by diet (reduced NaCl intake) and physical conditioning. Therefore, at least some of these circulatory changes are independent of the direct effects of aging because they are not consistently observed in healthy, physically active, non-obese, older individuals (Dempsey & Seals, 1995; Docherty, 1990; Fleg, 1986; Lakatta, 1993; Spirduso, 1996).

2.2 Cardiovascular Performance during Aerobic and Anaerobic Work

When moved from rest to a higher energy requirement (i.e., any physical task, change in body position change, and change in temperature), the human body responds through a series of integrated adaptations in function. These integrated adaptations may involve most, if not all, of the physiologic systems (e.g., musculoskeletal, cardiovascular, respiratory, endocrine, and immune systems). For example, movement requires activation and control of the musculoskeletal system, as well of the cardiovascular and respiratory systems to provide the ability to sustain this movement over extended periods. A more specific example, it might be the normal human CV system. It rapidly adjusts to the demands of exercise by calling on a number of interrelated responses that, in concert, serve to augment CO at adequate levels to meet the requirements of the exercising skeletal muscles. Along with increasing oxygen consumption, there is a linear increase in CO that is accomplished by an increase in HR and the rapid response of the left ventricle to augment SV. The augmentation of SV is accomplished both by use of the Frank-Starling mechanism and by use of LV contractile reserve (Bonow, 1994).
In this regard, this section primarily provides an overview of how the CV system responds (e.g., circulatory and/or left ventricular response) to an episode of physical exercise without any superimposed loads from the environment or others stresses in healthy-younger adults. Subsequently, an overview of the CV responses to exercise in older adults is also provided. Detailed reviews on CV responses to aerobic or dynamic exercises (Åstrand & Rodahl, 1986; Bevegård & Sherpherd, 1967; Bonow, 1994; Chapman, 1967; Clausen, 1977, 1976; Dempsey & Seal, 1995; MacDougall, 1994; McMurray, 1986; Rowel, 1993, 1974; Saltin, 1964; Seals, 1993; Wade & Bishop, 1962), and anaerobic exercises (Kraemer & Daniel, 1986; MacDougall, 1994, 1985; McCartney, 1999; Mitchell, 1990, 1985, 1983; Mitchell & Wildenthal; 1974) are plentiful, and are therefore, briefly summarized herein.

At the onset (including immediately prior to) and during the performance of physical activity, the CV system adapts to meet the demands imposed. (Åstrand & Rodahl, 1986; Bonow, 1994; Haslam et al., 1988; Lakatta, 1993; Lentini et al., 1993; MacDougall, 1994; MacDougall et al, 1992, 1985; McMurray, 1986; Miles et al., 1987; Sale et al., 1993; Seals, 1993; Spirduso, 1996; Surgeon General Report, 1996; Weicek, McCartney, & McKelvie, 1990). However, not all forms of activity elicit the same cardiac response. For example, aerobic or dynamic exercise induces increases in CO, HR, SV, and BP (MacDougall, 1994; Rowell, 1993) that differs markedly from the changes observed during isometric resistance exercise. Similarly, dynamic upper body exercise, which involves a smaller muscle mass than dynamic exercise with the larger muscles of the legs, also results in a slightly different cardiac response (Robergs, & Roberts, 1996).
In general, CV adjustments during activity are dependent upon: the metabolic demands of the activity (e.g., primarily oxidative vs. primarily non-oxidative) (Åstrand & Rodahl, 1986; Bergh, Kanstrup, & Ekblom, 1976; McMurray, 1986; Rowell, 1993; Taylor, Buskirk, & Henschel, 1955), the mode of muscle contraction (e.g., static versus dynamic contractions) (MacDougall, 1994; Mitchell, 1990, 1985; Michell, Kaufman, & Iwamoto, 1983; Mitchell, & Wildenthal, 1974), and strength and frequency of the muscle contraction (Perez-González, 1981).

Additionally, CV adjustments are mediated by many other factors that are specific to the physical activity being executed (e.g., dynamic, isometric, or resistance exercise) and particular to the individual performing the task. For example, acute CV responses to dynamic exercise are mediated by factors such as exercise intensity and duration (Åstrand & Rodahl, 1986; Åstrand & Saltin, 1961a, 1961b; Kraemer & Daniel, 1986; McMurray, 1986), mode of exercise (e.g., walking, running, or cycling) (Åstrand & Rodahl, 1986; McArdle, Katch, & Katch, 1991), body position in which the exercise is performed (Bonow, 1994, Poliner et al., 1980; Rowell, 1993), muscle mass involved in the task (Åstrand & Rodahl, 1986; Bergh et al., 1976; Clausen, 1977; Hermanse, 1973; Rowell, 1974), subject’s physical fitness level (Hagberg et al., 1985, Rowell, 1993) and body composition (McArdle, Katch, & Katch, 1991, Rowell, 1993), gender (Adams et al., 1987; Bonow, 1994; Fleg, 1995; Gibbons, Lee, Cobb, & Jones, 1981; Hagberg et al., 1985; Hankey et al., 1989; Higginbotham et al., 1984; Ogawa et al., 1992; Rodeheffer et al., 1984), cardiovascular health (Fleg, Rothfeld, & Gottlieb, 1991; Rerych et al., 1978), and age (Fleg, 1995, 1986; Gerstenblith, Lakatta, & Weisfeldt, 1976; Lakatta, 1993, 1980, 1979; Spirdudo, 1996; Strandell, 1976). For example, high intensity dynamic
exercises requiring large muscle masses (e.g., large muscle groups performing rhythmical
c kontractions) cause larger CV responses (i.e., larger increases in CO, HR and SBP, and
little change in DBP) than those CV responses caused by light intensity dynamic
exercises requiring the same muscle mass (Bergh et al., 1976; Rowell, 1993).

On the other hand, CV responses to static and resistance exercise depend on other
specific factors. For example, CV responses to static exercise depend on factors such as:
the relative tension exerted by the muscles (i.e. percentage of maximal voluntary
contraction -% MVC-) (Freyenschuss, 1970; Lind, McNicol, & Donald, 1966; Lind and
McNicol, 1967; Mitchell, 1990), duration of muscle contraction (Lind, McNicol, &
Donald, 1966; Mitchell, 1990; Shepherd et al., 1981), joint angle (MacDougall et al.,
1992), and mass of skeletal muscle that is recruited (Humphreys & Lind, 1963;
Blomqvist et al., 1981; Edwards & Wiles, 1981; McCloskey & Streatfeild, 1975;
(1990; 1986), MacDougall (1994, 1992), and McCartney (1999) have reported other
specific factors mediating CV responses. These factors include: mode of exercise (leg
press versus leg extension), absolute or relative load, number of repetitions and sets, joint
angle, muscle mass, and valsalva maneuver (MacDougall, 1994; McCartney, 1999).

Central Command and Afferent Autonomic Control. It should be noted that the
response of the CV response to aerobic exercise and/or non-robic exercise requires
appropriate input from the autonomic nervous system. That is, when the intensity of the
physical activity is increased, there is an overall increase in activity of the sympathetic
nervous system and a decrease in activity of the parasympathetic nervous system. The
increased sympathetic neural activity plays a role in controlling the rate and contractility
of the heart, and the caliber of the resistance and capacitance vessels in the systemic circulation. The decreased parasympathetic neural activity evokes an increase in HR (Mitchell, 1990, 1985, 1983; Rowell, 1993, 1974), but is not known to alter cardiac contractility or distribution of systemic BF.

The proposed mechanisms that are responsible for the regulation of sympathetic and parasympathetic efferent nerve activity to the heart and blood vessels during an increase in physical activity have been previously studied. In 1895, Johansson postulated that there were two separate and distinct mechanisms of neural control of circulatory adjustments to exercise. Since that time, two mechanisms of neural control of the cardiovascular system during exercise have been documented and generally accepted (Mitchell, 1990, 1985, 1983; Shepherd et al., 1981). In one mechanism, changes in autonomic nerve activity are thought to be caused by signals arising in a central area of the brain that activate the CV control areas in the medulla. This central neural mechanism, which was first termed “cortical irradiation” (Krogh & Lindhard, 1913-1914) and later “central command” (Goodwin, McCloskey, & Mitchell, 1972; McCloskey, 1981; Mitchell, 1985), serves as a feedforward control (Asmussen et al., 1965; Eldrigge, Millhorn, Waldrop, 1981; Freund et al., 1981; Freyschuss, 1970; Goodwin, McCloskey, & Mitchell, 1972; Hobbs, 1982; Iwamoto et al., 1984; Leonard et al., 1985; Ochwardt et al., 1959; Schibye et al., 1981). In the other mechanism, the changes in autonomic nerve activity are thought to be due to signals arising in the contracting skeletal muscles themselves, which reflexly activate the same CV control areas in the medulla (Alam & Smirk, 1937; 1938; Kao & Ray, 1954; Patterson, 1928). This reflex neural mechanism termed the “exercise pressor reflex” (Mitchell, Kaufman,
& Iwamoto, 1983) serves as a feedback control (Adams et al., 1984; Alam & Smirk, 1937; 1938; Asmussen, Nielsen, & Wieth-Pedersen, 1943; Hultman & Sjöholm, 1982; Kao & Ray, 1954; Mitchell, Kaufman, & Iwamoto, 1983; Mitchell, Reardon, & McCloskey, 1977; Patterson, 1928).

Studies suggesting that a central neural mechanism might be responsible for the CV changes that occur during exercise have been performed in anesthetized cats (Eldrigde et al., 1985, 1981; Orlovskii, 1969; Waldrop et al., 1986a, 1986b; Waller, 1940), and in human subjects using a different experimental model (e.g., neuromuscular blockade or epidural anesthesia) during static exercise (Freyschuss, 1970; Frienman et al., 1990; Goodwin, McCloskey, & Mitchell, 1972; Leonard et al., 1985; McCloskey, & Mitchell, 1972; Mitchell, 1989a, 1989b) and dynamic exercise (Asmussen et al., 1965; Fernandes et al., 1990; Freund et al., 1979; Galbo, Kjaer, & Secher, 1987; Ochwadt et al., 1959). For example, anesthetized cat studies have documented that electrical or chemical stimulation of the subthalamic locomotor region causes a CV response even when the cat is paralyzed or heavily anesthetized and does not walk (Eldrigde et al., 1985, 1981; Waldrop et al., 1986a, 1986b). With the onset of the stimulation, there was an increase above control values in heart rate, arterial pressure, left ventricular systolic pressure development (dP/dt), with no change in LVED pressure. Therefore, adding radioactive microspheres procedures, an increase in blood flow to the heart, diaphram, and limb skeletal muscles and a decrease in blood flow to the kidneys were observed. When the stimulation was stopped, the CV responses returned to control values (For a review, see Mitchell, 1990).
Studies with human subjects designed to determine the relative role of the central neural mechanism on CV responses during exercise have used partial neuromuscular blockade during static (Freyschuss, 1970; Iwamoto et al., 1987; Leonard et al., 1985; Mitchell, 1989b) and dynamic (Asmussen et al., 1965; Galbo, Kjaer, & Secher, 1987; Ochwardt et al., 1959) exercises, as well epidural anesthesia during static (Mitchell et al., 1989a) and dynamic (Fernandes et al., 1990; Freund et al., 1979) exercises. Partial neuromuscular blockade has been used to reduce muscle strength, thereby increasing the central command in relation to the actual force developed. Epidural anesthesia has been used to block sensory feedback from the contracting muscles, increasing thus the central command in relation to the actual force developed, with a simultaneous decrease in the exercise pressor reflex.

The main findings from studies (emphasizing the role of central neural mechanism on CV response to exercise) with human subjects are: (1) Leonard et al. (1985), using partial neuromuscular blockade with tubocurarine during static exercise, found that HR and arterial BP during partial neuromuscular blockade were greater than the control study at the same absolute force (10% of the control MVC). However, HR and BP responses during neuromuscular blockade were the same as during the control study, at the same relative force. (2) Asmussen et al. (1965) and Ochawardt et al. (1959), using partial curarization to reduce the strength of exercise muscle, found that HR and BP responses during dynamic exercise were greater than during dynamic exercise without curarization, even though the workload were the same for both conditions. (3) Goodwin et al. (1972), activating primary muscle afferents either in the contracting muscle or in its antagonist (to vary the central command required to achieved a given tension in man), found that
when the same muscle tension was achieved with less central command during static exercise, the increase in BP and HR was less, and when the same tension was achieved with more central command, the cardiovascular response was greater. (4) Freund et al. (1979), using epidural anesthesia to block the sensory input from working muscle and reflex neural mechanism, found that the relation of cardiac output to oxygen consumption during dynamic exercise, and the increase in arterial BP to the tension developed by the active muscle during static exercise were not altered. There was an increase in mean arterial BP appropriate to the amount of force developed by the contractile muscle, while the sensory input from working muscle remained blocked and motor strength progressively recovered after spinal anesthesia. In summary, all these previous results appear to suggest that an appropriate CV response to both static exercise and dynamic exercise occurs when the reflex neural mechanism is blocked or reduced, and only the central neural mechanism is operative, which appears to suggest that central neural mechanism mediates the CV response to exercise (For a review, see Mitchell, 1990, 1985, 1983).

On the other hand, there are some other studies emphasizing that the changes in autonomic nerve activity are due to signals arising in the contracting skeletal muscles themselves, which reflexly activate the same cardiovascular control areas in the medulla (Adams et al., 1984; Alam & Smirk, 1937; 1938; Coote, Hilton, & Pérez-González, 1971; Kao & Ray, 1954; McCloskey, Mitchell, 1972; Mitchell, Kaufman, & Iwamoto, 1983; Mitchell, Reardond, & McCloskey, 1977; Patterson, 1928). These studies have been also performed in anesthetized cats (Coote, Hilton, & Pérez-González, 1971; McCloskey, Mitchell, 1972; Mitchel et al. 1977; Mitchell, Reardond, & McCloskey, 1977) and in
human subjects (Alam & Smirk, 1937; 1938; Hultman, & Sjöholm, 1988; Mitchell et al., 1989a). The latter studies, those with human subjects, have been also performed using the same previously mentioned experimental model (neuromuscular blockade and epidural anesthesia) during static exercise (Alam & Smirk, 1937; 1938; Hultman, & Sjöholm, 1988; Mitchell et al., 1989a) and dynamic exercise (Adams et al., 1984; Asmussen, Nielsen, & Wieth-Pedersen, 1943; Fernandes et al., 1990; Galbo, Kjaer, & Secher, 1987).

The main findings from studies suggesting the role of the reflex neural mechanism or exercise pressor reflex are: (1) Coote, Hilton, & Pérez-González (1971), McCloskey, & Mitchell (1972), and Mitchell, Reardond, & McCloskey (1977) using anesthetized cat studies found that electrically stimulating the peripheral ends of sectioned spinal ventral roots, which induces contractions in the muscles of the handling, causes an increase in arterial BP and HR. (2) Mitchell et al. (1977), using induced static contraction, reported an increase in HR, arterial pressure, LV systolic pressure, and the maximal rate of LV pressure development (dP/dt), with no change in LVED pressure. At the cessation of induced static exercise, these CV parameters returned to their control values. (3) Adam et al. (1984) and Asmussen, Nielsen, & Wieth-Pedersen (1943) comparing the CV response to voluntary leg extension with those produced by direct electrical stimulation of the leg muscles found evidence of a reflex neural mechanism during dynamic exercise. (4) Hultman & Sjöholm (1982) comparing quadriceps voluntary static contraction to quadriceps electrical stimulated contraction found that HR and BP responses were exactly the same whether the static contractions were electrically elicited (no central command) or voluntarily produced. (5) Mitchell (1989a) using epidural anesthesia during static contractions with or without inflation of a cuff placed around the upper thigh found
that: (a) the mean arterial pressure and HR responses to static exercise during epidural anesthesia were the same as during the control study at the same absolute force (10% of the control MVC); (b) the mean arterial pressure and HR responses were less during epidural anesthesia than during the control study at the same relative force (30% of MVC before and after epidural anesthesia); (c) after static contractions were completed without cuff occlusion with both absolute and relative force, mean arterial pressure decreased rapidly and during cuff occlusion the pressure remained elevated until the cuff was released; and (d) the mean arterial pressure tended to be higher with the cuff inflated during the control study than with the cuff inflated during epidural anesthesia. These results appear to suggest that the reflex neural mechanism might be important in determining the CV response to static exercise. In summary, these findings appear to suggest that appropriate CV responses to static and dynamic exercises might be due to a reflex neural mechanism (exercise pressor reflex) arising in the muscle and that central command was not necessary for the response (Mitchell, 1990, 1985, 1983).

Alternatively, based on the findings from both central neural mechanism and reflex neural mechanism studies, it has been suggested that both mechanisms might be present and that either or both might bring about the CV changes that occur during muscular exercise (Mitchell, 1990; Mitchell & Schmidt, 1983; Sherpherd et al., 1981). For example, Mitchell (1990) and Sherpherd et al. (1981) suggest that the neural activity responsible for the recruitment of motor units (central mechanism) initiates the CV response and determines the immediate changes in the level of the afferent activity of the sympathetic and parasympathetic autonomic nervous system to the heart and blood vessels. Subsequently, mechanical and metabolic activity in the contracting muscle
(exercise pressor reflex) determines the level of autonomic activity to the circulatory system. They suggest that receptors (i.e., ergoreceptors) activated by exercise respond to metabolic and mechanical alterations and their afferent impulses are conducted by groups III and IV fibers to the spinal cord where they ascend to the CV areas. As a result of these two inputs to the CV areas, the parasympathetic activity to the heart decreases and the sympathetic activity to the heart, blood vessels and adrenal medulla increase (For a review, see Mitchell, 1990, 1985; Sherpherd et al., 1981).

### 2.2.1 Acute Cardiovascular Response to Aerobic Work

In this review, aerobic activity/exercise is defined as any physical activity or task in which the required energy to perform the task is supplied or generated through the aerobic metabolic processes (aerobic oxidation pathway). This subset of exercise, called dynamic or isotonic exercises because skeletal muscle contraction causes a change in length with little change in tension, represents exercises such as walking, jogging, running, cycling, swimming, or dancing. Throughout the performance of these exercises, muscle contractions are rhythmic, so that muscles are active during a portion of the movement cycle and relaxed during the remainder. As the speed of the movement increases, the actual duration of each phase decreases, but the proportion of time spent in contraction or relaxation remains relatively constant. (For a review, see MacDougall, 1994; Mitchel & Wildenthal, 1974; Surgeon General Report, 1996).

**Circulatory and left-ventricular response to aerobic exercise in younger adults:**

Cardiovascular responses to aerobic or dynamic exercise in healthy-younger adults have been previously reviewed in detail (Åstrand & Rodahl, 1986; Bevegärd & Sherpherd, 1967; Bonow, 1994; Chapman, 1967; Clausen, 1977, 1976; Dempsey & Seal, 1995;
MacDougall, 1994; McMurray, 1986; Rowel, 1993, 1974; Saltin, 1964; Seals, 1993; Wade & Bishop, 1962), and are only briefly summarized herein. Furthermore, this section is organized according to the Fick principle, which is expressed as:

\[ \text{VO}_2 = \text{CO} \times (A-V) \text{O}_2 \text{ diff} \]

\[ \text{VO}_2 = (HR \times SV) \times (A-V) \text{O}_2 \text{ diff} \]

In these equations, \( \text{VO}_2 \) is oxygen uptake; \( \text{CO} \) is cardiac output, that is \( \text{CO} = HR \times SV \); \( HR \) is heart rate; \( SV \) is stroke volume; and \( (A-V) \text{O}_2 \text{ diff} \) is arterial-mixed venous oxygen difference (arteriovenous oxygen difference).

**Oxygen uptake**: With the onset (or even before) and during the performance of aerobic or dynamic exercise, there is a rapid increase in energy requirements (Åstrand & Rodahl, 1986; McArdle, Katch, & Katch, 1991; Smith, & Kampine, 1990), which is matched with equally rapid circulatory adjustments. These adjustments basically are led to meet the increased need for \( O_2 \) and nutrients by the exercising muscle, to remove the end products of metabolism such as \( CO_2 \) and lactic acid, and to dissipate the excess heat produced by the reactions.

Accordingly, there is an increase in the rate of oxygen uptake (\( \text{VO}_2 \)) that is directly proportional to the work performed until a plateau of oxygen uptake is eventually reached [i.e., up to maximal work capacity (\( \text{VO}_{2\text{max}} \))]. Generally, the plateau phase is reached before the effort becomes intolerable (Åstrand & Rodahl, 1986; Brooks, Fahey, & White, 1996; Hill, & Lupton, 1923; McArdle, Katch, & Katch, 1991; Rowell, 1993; Seals, 1993). Maximal oxygen uptake (\( \text{VO}_{2\text{max}} \)) is described as the greatest amount of \( O_2 \) that can be utilized at the cellular level for the entire body (Åstrand & Rodahl, 1986). It is determined by the rate at which \( O_2 \) is transported to the tissues, the oxygen-carrying
capacity of blood, and the amount of O₂ extracted from the blood. (Williams, 1994; Brooks, Fahey, & White, 1996).

While resting oxygen uptake (VO₂) value is estimated at 3.5 ml O₂ per kg of body weight per min (value defined as 1 metabolic equivalent, or MET), maximal oxygen uptake values may range from 40 to 80 ml · Kg⁻¹ · min⁻¹, or 11.4 to 22.9 METs, in normal, healthy individuals (Williams, 1994; Rowell, 1993). Basically, VO₂max is determined by CO, HR, SV and arteriovenous oxygen difference (Rowell, 1993), and is proportional to the subject’s physical fitness level (Guyton, & Hall, 1996; McArdle, Katch, & Katch, 1991; Rowell, 1993). The increase in oxygen demand is met basically through a central mechanism—an increase in cardiac output (CO)—and a peripheral mechanism—an increase in O₂ extraction from the blood [(a – v)O₂ difference] (Seals, 1993). Thus, the main cardiovascular responses to aerobic exercise that have been documented are:

- **Cardiac output, heart rate, and stroke volume:** Studies in men performing graded upright exercise show that CO, HR, SV, (a – v) O₂ difference, ejection fraction, and blood pressure increase with increasing workload (Åstrand & Rodahl, 1986; Bonow, 1994; Plotnick et al., 1986, Rowell, 1993). The increase in CO is directly proportional (nearly linearly) to oxygen uptake. However, the increase in CO and LV function differs at various levels of exercise intensity. For example, during early stages of exercise, the initial increase in CO is accomplished by increases in HR and SV (Higginbotham et al., 1986a, 1984; Plotnick et al., 1986; Weiss et al., 1979), while at higher and maximal exercise intensities, the increase in CO is accomplished mainly by an increase in HR, due to the early level-off of the SV (Åstrand & Rodahl, 1986; Bonow, 1994; Gerstenblith,
In addition, it has been observed that CO adjustments during aerobic and dynamic exercises are different according to the body position assumed in exercise (Bonow, 1994; Burge, Carey, & Payne 1993; McCartney et al., 1993), the subject’s physical fitness level (Clausen, 1969; Ekblom, & Hermansen, 1968; Hanson, Tabakin, Levy, & Neddle, 1968; Tabakin, Hanson, & Levy, 1965), subject’s health status (Blackmon et al., 1967), gender (Åstrand et al., 1964; Freedson et al., 1979; Kilbom, & Åstrand, 1971), and age (Gerstenblith, Renlund, & Lakatta, 1987; Hagberg, 1987).

With respect to body position, during exercise performed in the upright position, the increment in CO is mediated by increases in both HR and SV, whereas during supine exercise the increase in CO is achieved mainly through an increase in HR (Bonow, 1994; Rowell, 1993; Seals, 1993). During supine exercise, SV is greater (than in the upright position).
position) and with increasing exercise it is maintained at nearly constant levels. Thus, maximal SV is achieved at the onset of exercise (Burke, Carey, & Payne 1993; McCartney et al., 1993).

With respect to physical fitness level, while resting CO between trained and untrained subjects (with average values ranging between 5 and 6 L \( \cdot \) min\(^{-1} \)) show little difference in values, maximal CO in trained male subjects can reach values in excess of 30 L \( \cdot \) min\(^{-1} \) (five- to six-fold increase over resting values). In fact, values of CO as high as 42 L \( \cdot \) min\(^{-1} \) have been observed in elite endurance athletes (Ekblom, & Hermansen, 1968). Conversely, untrained male subjects, who have lower aerobic capacities, have lower maximal cardiac outputs (about 20 to 25 L \( \cdot \) min\(^{-1} \)). The same lower CO values have been also observed in persons with CV diseases (e.g., patients with mitral stenosis), who had approximately a maximal CO of 8.2 L \( \cdot \) min\(^{-1} \) (Blackmon et al., 1967). Therefore, exercising at similar submaximal work rates, the CO of untrained subjects may be slightly higher than (Clausen, 1969; Ekblom et al., 1968; Hanson, Tabakin, Levy, & Neddle, 1968; Tabakin, Hanson, & Levy, 1965) or the same as trained subjects (Hartley et al., 1969; Saltin et al., 1968).

Additionally, gender differences in the CO response to exercise have been determined (Åstrand et al., 1964; Freedson et al., 1979; Kilbom, & Åstrand, 1971). Adjustments in CO for men and women during exercise are similar (as described above); however, in comparison with men, women tend to have a slightly higher CO when exercising at the same VO\(_2\). This slight difference amounts to between 1.5 and 1.75 L \( \cdot \) min\(^{-1} \), which has been attributed to the lower hemoglobin concentrations typically seen in women. Further,
maximal CO of trained and untrained women are generally lower than that of their male counterparts (For a review, see Foss, & Keteyian, 1998).

Lastly, age influences the CO response to aerobic or dynamic exercise. This will be discussed in greater detail in subsequent chapters. However, in general, maximal CO tends to decrease in a linear fashion in both men and women after 30 years of age (Gerstenblith, Renlund, & Lakatta, 1987; Hagberg, 1987). This has been attributed to an age-related decrease in HR_{max} with age (Gerstenblith, Renlund, & Lakatta, 1987).

**Heart rate:** HR increases with exercise intensity up to a rate of approximately 220-age (Åstrand & Rodahl, 1986; Bonow, 1994) (see figure 2). Traditionally, the nature of this relationship is thought to be linear; however, data from ramp protocols suggest the possibility that it is curvilinear, with only a small portion of the curve resembling a linear function (Robergs, & Roberts, 1997). Regardless, this intensity-dependent increase in HR is an important factor for increasing CO during exercise (Rowell, 1993).

During dynamic exercise, the pattern of the HR response has been described as: (1) HR increases with exercise intensity and oxygen consumption (Åstrand & Rodahl, 1986; Bonow, 1994; Rowell, 1993; Seals, 1993), and levels off at VO_{2max} (Rowell, 1993). (2) During a constant level of submaximal exercise, HR increases and then levels off as the oxygen requirements of the activity have been satisfied; however, as exercise intensity increases, it takes longer for HR to level off. At higher intensities, HR might not level off. (3) During prolonged exercise, HR increases steadily at the same work rate. This phenomenon, called cardiovascular drift, results from a decreased SV with prolonged exercise. Heart rate must increase in order to maintain CO and blood pressure at the same level. Cardiovascular drift is caused by a diminished capacity of the circulation to return
blood to the heart (i.e., decreased venous return). Decreased venous return may be due, in turn, to decreased plasma volume caused by filtration of fluid from the blood or by sweating. It may also be due to decreased sympathetic tone.

**Figure 4. Heart Rate and Exercise Intensity Relationship**

[Taken from Surgeon General Report, 1996]

At the same power output, HR is higher during upper-body exercise than lower-body exercise. Upper-body exercise also results in higher VO$_{2\text{max}}$, mean arterial pressure, and total peripheral resistance. The higher circulatory load in upper-body exercise results from the use of a smaller muscle mass, increased intra-thoracic pressure, and less effective muscle pump. Near maximal contraction using smaller muscle mass restricts blood flow. All three factors decrease venous return of blood to the heart.

During submaximal exercise, women tend to have a higher HR at any given work rate than their male counterparts. This is, as mentioned previously, because women tend to have a smaller SV for the same VO$_2$.

The increase in HR at low levels of exercise is due to vagal release, and the increase at higher levels of exercise is due to combined vagal release and sympathetic activation.
The maximal heart rate (HR$_{max}$), which is usually achieved at VO$_{2max}$, is considered an important limiting factor in cardiovascular performance.

**Figure 5. Stroke Volume and Exercise Intensity Relationship**

[Taken from Surgeon General Report, 1996]

*Stroke volume*: During upright exercise, it has been documented that stroke volume (SV) increases during the progression from rest to moderate work (Bevegard, Freyschuss, & Strandell, 1966; Chapman, Fisher, & Sproule, 1960; Keteyian et al., 1994; Stenberg et al., 1967). Thereafter, SV may level off (Bevegard, Freyschuss, & Strandell, 1966; Stenberg et al., 1967) or continue to increase more gradually (Gledhill, Cox, & Jamnik, 1994). Early studies (e.g., Bevegard, Freyschuss, & Strandell, 1966; Stenberg et al., 1967) indicate that SV plateaus at approximately 50% of VO$_{2max}$. However, later studies (e.g., Gledhill, Cox, & Jamnik, 1994; Scruggs et al. 1991) show that SV in trained individuals continues to increase to VO$_{2max}$, suggesting that this training adaptations may explain some of the training induced increases in maximal CO and VO$_{2max}$ (For a review, see Robergs, and Roberts, 1997; Surgeon General Report, 1996).
Other sources of variation in the SV response during dynamic exercise have been attributed to body position, physical fitness level, and gender. For example, the previously described increase in SV is typical for exercise performed in the vertical or upright position. However, studies conducted on individuals exercising in a recumbent or supine position have shown that maximal SV is attained at the onset of exercise (Burge, Carey, & Payne, 1993; McCartney et al., 1993), presumably resulting from the lack of hydrostatic pressures that resist venous return to the heart (Robergs, & Roberts, 1997).

During maximal exercise, it has been found that SV in trained males may approach 150 to 170 mL \( \text{beat}^{-1} \). In fact, among elite, highly trained endurance athletes, maximal SV might reach or even exceed 200 mL \( \text{beat}^{-1} \). (Ekblom et al., 1968). Conversely, SV in untrained males appear to approach between 100 to 120 mL \( \text{beat}^{-1} \). Thus, the primary contributing factor for the much larger CO observed in endurance-trained athletes appears to be greater maximal SV. Maximum HRs are generally similar in both trained and untrained individuals, whereas the maximum SV of an athlete may approach twice that of the nonathlete (Rowell, 1993).

Additionally, it has been documented that SV for women during exercise is generally lower than those for men under all conditions. Therefore, maximal SVs for untrained and trained women (between 80 and 100 mL \( \text{beat}^{-1} \) and 100 and 120 mL \( \text{beat}^{-1} \), respectively) are lower than those for untrained and trained men. At submaximal workload requiring the same oxygen consumption, SV is lower in women than in the men due to the smaller heart volume of the women (For a review, see Foss, & Keteyian, 1998).
The mechanisms responsible for the increase in SV during exercise include increased preload (Frank-Starling mechanism) and heightened inotropic state (For a review, see Foss, & Keteyian, 1998). As a rough approximation, SV increases by 30%-50% during upright exercise (Higginbotham et al., 1986b, 1984; Plotnick et al., 1986 Poliner et al., 1980) with the greatest increase occurring during the initial minutes. The increase in SV during early stages of exercise is accomplished predominantly by an increase in left ventricular end-diastolic volume (preload). This also places the left ventricle under a greater stretch resulting in a heightened contractility according to Frank-Starling. Together, the increase in EDV and the concomitant increase in contractility are responsible for the rapid and significant increase in SV that occurs with the onset and at lower levels of exercise.

After the initial increase in end-diastolic volume (EDV) and SV that occur with the onset of exercise at low levels, EDV plateaus at higher intermediate levels of exercise (Higginbotham et al., 1986a, 1984; Plotnick et al., 1986). However, the small increases in SV that continue to occur during submaximal exercise are elicited by a sympathetic-mediated enhanced LV inotropic state, and consequently, a decrease in LV end-systolic volume (i.e. greater ejection fraction).

At higher exercise intensities, EDV begins to decline; however, SV is maintained due to heightened sympathetic drive, and therefore, greater LV contractility. Thus, as SV plateaus at higher workloads, the continual increase in CO that occurs during such incremental work is primarily the result of the progressive increase in HR. (Åstrand & Rodahl, 1986; Bonow, 1994; Fox, Bowers, & Foss, 1993; Gerstenblith, Renlund, &

In summary, the increase in CO is accomplished through increases in both HR and SV. The increase in HR at low levels of exercise is due to vagal release, but at high levels it is due to combined vagal release and sympathetic activation. SV is augmented during early exercise primarily through the use of preload reserve and Frank-Starling relationships, whereas it is maintained at these levels during maximal exercise predominantly through the sympathetic-mediated recruitment of LV contractile reserve.

It should be noted that differences in the CV responses to dynamic exercise have been attributed to body position (Bonow, 1994) and muscle mass involved during exercise (Rowell, 1993). For example, during exercise performed in the upright position, the increment in CO is mediated by increases in both HR and SV, whereas during supine exercise, because of increased filling pressure, SV is greater (than in the upright position) and with increasing exercise it is maintained nearly constant, so that the CO increase in the supine position is achieved mainly through increase in HR. In the sitting or standing position, SV is about one-third less than in the supine position. Thus, in upright position exercise, SV is initially smaller but increases progressively at increasing work levels until about 40% of $VO_{2\text{max}}$, at which point it reaches its maximal value (for upright position) and plateaus. However, the SV at $VO_{2\text{max}}$ is still lower in upright than in supine exercise (Bonow, 1994; Rowell, 1993; Seals, 1993).

With respect to muscle mass, it has been demonstrated that $VO_{2\text{max}}$ is dependent on the mass and type of muscle involved. For example, it has been reported that the $VO_{2\text{max}}$ in arm ergometry is only 60% to 80% of that of leg ergometry or of combined arm and
leg ergometry (Bonow, 1994). The reason for the VO$_{2\text{max}}$ limitation when smaller muscle groups (e.g. the arm) are involved has been attributed to a lesser mechanical efficiency (i.e., a smaller ratio of work output to caloric expenditure) or a lesser degree of conditioning (which usually prevails in arm vs. leg muscles). But regardless of the cause, there is a greater HR and pressor response to arm-work compared to a similar level of legwork. Aside from the lesser mechanical efficiency of small muscle groups, there is also a lesser “myocardial efficiency.” For an equivalent degree of work, arm exercise induces a greater systolic pressure-HR increment than legwork. Since the pressure-rate product is a close correlate of myocardial oxygen consumption, arm work is more likely to produce myocardial ischemia in angina patients.

*Arterial-mixed venous oxygen difference (A-VO$_2$diff)*: The arterial-mixed venous oxygen difference or arteriovenous oxygen difference (amount of O$_2$ that is taken up from 100 ml of blood by the tissues during one trip around the systemic circuit) increases during exercise. That is, venous O$_2$ saturation decreases progressively at increasing exercise levels, and since the arterial O$_2$ content is essentially unchanged, the a – v O$_2$ difference may increase 2- to 3-fold (Rowell, 1993; Smith, & Kampine, 1990).

At rest (a – v) O$_2$ difference is normally 4.5 ml 100 ml$^{-1}$ (approximately 23 percent extraction), while at VO$_{2\text{max}}$ this difference is commonly close to 16 ml 100 ml$^{-1}$. Usually 80 to 85 percent of the available oxygen is extracted from the total blood volume at VO$_{2\text{max}}$. Some oxygenated blood returns to the heart, because blood flowing through metabolically less active tissues does not fully extract the oxygen from the blood. Consequently, the difference for oxygen concentration in arterial and the mixed venous or pulmonary arterial blood increases with increasing oxygen consumption.
This increased O₂ extraction (amount of O₂ taken up and used for the oxidative production of ATP by skeletal muscle) along with CO is a determining factor increasing O₂ uptake in aerobic and dynamic exercise. However, there are no differences in (a – v) O₂ difference among trained, untrained and CV disease patients at VO₂max. Arteriovenous oxygen difference at VO₂max reaches ~17 ml 100 ml in the mitral stenosis patients as it often does in well-conditioned individuals (Rowell, 1993).

*Arterial blood pressure:* During aerobic or dynamic exercise blood flow must be maintained to critical areas such as the heart and brain, while, at the same time, the requirements of working muscles and skin need to be met. Furthermore, CV responses during exercise (increases in CO and decrease in peripheral resistance) are directed toward maintaining arterial blood pressure. Thus, CV regulation balances the need for more blood to active tissue with the need to maintain blood pressure. Blood pressure (BP) is a function of CO and peripheral resistance (PR), which is expressed as:

\[
\text{Blood pressure (mm Hg)} = \text{CO} \times \text{TPR}
\]

Where CO is cardiac output, and TPR = total peripheral resistance.

The typical BP response to progressive dynamic exercise is presented in Figure 2. During this mode of exercise, there is an almost linear, and pronounced rise in SBP, reaching levels of 200 mm Hg or more at VO₂max. Although during this time CO may have increased four-to six-fold, the rise in SBP is to a large extent buffered by the concomitant decrease in TPR caused by the progressive vasodilation that occurs in the vessels of the exercising muscles. Because of this decrease in TPR, DBP shows little or no increase, and in normotensive persons generally decreases slightly at higher
workloads. Mean BP during this type of exercise seldomly exceeds 120 mm Hg (For a review, see MacDougall, 1994).

![Blood Pressure Response to Dynamic Exercise](image)

**Figure 6. Typical Blood Pressure Response to Dynamic Exercise**

[Adapted from MacDougall, 1994].

TPR decreases during exercise because of local autoregulatory mechanisms that allow an increase in BF to working skeletal muscles. This "autoregulation" overrides a general sympathetic mediated vasoconstrictive response that serves to restrict flow to less active tissue. The end result is generally a decrease in TPR, provided the mass of the active tissue is sufficient to mediate such an effect. Regardless of the balance between active and inactive skeletal muscle mass, the increase in CO will super-cede the decrease in TPR. As a result, mean arterial pressure will increase during dynamic exercise.

There is debate about the maximum safe SBP during exercise. Some experts are comfortable with SBP even higher than 250 mmHg; others suggest terminating exercise tests when the SBP exceeds 220 mmHg. In this debate, the characteristics of the subject are important and must be considered.
Blood flow: As alluded to above, the heart and circulation respond to the requirements of metabolism during exercise by increasing BF to active areas and decreasing it to less critical areas. A rise in BF to the exercising muscles is brought about through both an increase and a redistribution of CO. At rest, only about 15% to 20% of CO is distributed to skeletal muscle, in contrast to 85% to 90% during maximal exercise. Considering that CO may increase as much as 5-fold during maximal exercise, the redistribution of blood flow to the skeletal muscles represents a 15- to 20-fold increase from rest to maximal work. Similarly, and worthy of mention, there is a 3- to 5-fold increase in blood flow to the myocardium from rest to maximal work.

The redistribution of flow, which is highly important in the shunting of oxygenated blood to active muscle, results not only from a decreased vascular resistance in skeletal muscle but also from strong sympathetic vasoconstriction in other tissues such as kidney, splanchnic area, and nonexercising muscle. Total BF to the vasculature of the brain is unchanged, while myocardial BF increases consistent with the change in CO (Bevegärd & Shepherd, 1966). Thus, as might be expected during leg exercise, blood flow to the non-exercising forearm muscles is decreased. Although the wide vasodilatation of skeletal muscle arterioles, the veins of both exercising and nonexercising tissues are constricted, particularly those of the splanchnic venous bed. This is a sympathetic effect that assists in shunting blood into the central circulation, thereby aiding CO.

In skeletal muscle, the increased flow is initiated by anticipatory sympathetic cholinergic vasodilation and is reinforced by the metabolic hyperemia. As exercise continues, the general tendency to sympathetic vasoconstrictions in skeletal muscle is overwhelmed by metabolic vasodilations (autoregulation), thereby resulting in a massive
increase in BF to this tissue. The vasodilation in the capillary bed of working muscle is probably due to increases in PCO$_2$, K$^+$ and H$^+$.

*Plasma catecholamines:* Plasma catecholamines increase markedly, depending on the intensity of the exercise and the active muscle mass involved. This reflects the sympathetic hyperactivity that is characteristic of the exercise response. Plasma levels of epinephrine may increase 3- to 4-fold and norepinephrine levels 5- to 7-fold in intensive exercise. The plasma catecholamines increase only slowly during submaximal exercise, but when approaching VO$_{2\text{max}}$, rise steeply. The plasma levels of renin activity, vasopressin, and prolactin also increase with exercise.

In summary, with the onset and during aerobic exercise demands on the system increase considerably. The amount of oxygen delivered to the working muscles increases with the intensity of the exercise. CO increases due to a higher HR and SV, and reductions in peripheral vascular resistance. HR and CO tend to plateau at a near VO$_{2\text{max}}$, but SV levels off at about 50% of VO$_{2\text{max}}$. SBP tends to increase, but DBP does not increase, thus the increases in mean arterial pressure are modest. Blood flow is maintained or increased to the heart and brain. At the same time, blood is directed away from less active tissues, such as the viscera and inactive muscles, and goes to the skin to help dissipate heat produced by the increased metabolism of the exercise. The changes in LV EDV relative to ESV throughout the spectrum of exercise result in a progressive increase in ejection fraction at each level of exercise. The limitation to maximal dynamic exercise performance lies in the transport of O$_2$ to the tissue and removal of CO$_2$ and waste products. The specific limitation is either in the limited CO or in the enzymatic
capabilities of the muscle tissue to extract the oxygen (Brooks, Fahey & White, 1996; McCartney, & McKelvie, 1996; McCartney, 1999; Mitchell, & Wildenthal, 1974).

*Aging and circulatory and left-ventricular response to aerobic exercise:* Resting CV function is largely unaffected by age. However, impairment in CV function is likely to be manifest as a reduction in functional reserve, and therefore requires the examination of function when the system is stressed. As a result, the detection of age-related changes in functional reserve would require excluding the possibility the disease might be limiting CV performance. The same consideration must be made with respect to excluding poor physical fitness, tobacco or alcohol abuse, and obesity, all of which have a larger effect on CV performance during exercise than that observed at rest. Another difficulty in identifying effects of age on CV function relates to selective attrition. That is, if one survives to a very-old age, it is likely that he/she was relatively "healthy" at younger ages. Thus potentially confounds our ability to detect effects of age from a cross-sectional design (For a review, see Fleg, 1986).

Nonetheless, there has been a recent interest in describing the age-related changes in CV responses to aerobic exercise. Much of this work has been reviewed in detail by Dempsey & Seal (1995), Dochertry (1990), Folkow & Svanborg (1993), Fleg (1994, 1986), Lakatta (1993, 1990), Limacher (1994), Seals (1993), Seals et al. (1994), Shephard (1997), and Spirduso (1996). This section attempts to integrate the observations and conclusions from such prior sources. The content is organized according to the Fick principle:

\[
\text{VO}_2 = \text{CO} \times (A - V) \text{O}_2 \text{Diff}
\]
During aerobic exercise involving a large mass of skeletal muscle, there is great demand for oxygen to supply the increased metabolic needs of the contracting muscles (McCartney, & McKelvie, 1996; McCartney, 1999; Mitchell, & Wildenthal, 1974). Therefore, CV responses to aerobic exercise have been described as a ‘volume load’ to the heart (Mitchell, & Wildenthal, 1974). That is, a large increase in CO mediated by a rise in both SV and HR together with reductions in TPR. Systolic blood pressure tends to increase, but DBP does not, thus increases in mean arterial pressure are modest. All these CV adjustments to aerobic exercise appear to be similar in older adults; however, some age-related differences have been observed as described below.

**Oxygen uptake:** Whether expressed as the maximal attainable work rate (power output), maximal oxygen consumption (VO$_{2\text{max}}$), or otherwise, the maximal capacity to perform dynamic exercise with large muscle groups declines with advancing age in humans (Buskirk, & Hodgson, 1987; Dehn, & Bruce, 1972; Fleg, 1995; Raven, & Mitchell, 1980; Robinson, 1938). However, it should be noted that when VO$_{2\text{max}}$ is used as an index of maximal aerobic exercise capacity, the age-related decline appears to be highly variable (Buskirk, & Hodgson, 1987).

Accordingly, it has been suggested that the differences among investigations might be due to methodological factors such as use of cross-sectional versus longitudinal study designs, subject selection, and the ability to measure a “true” VO$_{2\text{max}}$, especially in older subjects. Conversely, much of the variability within populations might be due to factors (e.g., mode of exercise, fitness level, body composition, cardiovascular health) that modulate the rate of decline in VO$_{2\text{max}}$ independently of the aging process (Dempsey, & Seals, 1995).
Given the decline in VO$_{2\text{max}}$ with age, the same absolute work rate represents a higher percentage of an older subject’s maximal (peak) work capacity in any particular mode of large-muscle exercise (i.e., a higher relative work load). Therefore, to identify age-related changes in CV responses to aerobic exercise, it might be necessary to examine the control of a particular autonomic or circulatory adjustment in term of equal or similar primary stimulus (e.g., same relative level of exercise stress) (Clausen, 1976; Rowell, 1993, 1974). For example, some authors (e.g., Granath et al., 1964; Strandell, 1964) have reported that VO$_2$ increases similarly in younger and older adults performing the same absolute submaximal work rates during cycling exercise, whereas other authors (e.g., Montoye, 1982) have reported that older adults as compared to younger adults attain greater VO$_2$ during treadmill walking at the same speed and grade. Even though, the differences in VO$_2$ in Montoye’s study might be construed as meaning a decreased efficiency in walking among older adults (Montoye, 1982), the workloads used in both
studies represent a higher relative effort for the older adult group, given the age-related decline in VO$_{2\text{max}}$. For a review, see Fleg, 1986). Thus, from a standpoint of identifying age-related changes in the CV response to aerobic exercise, it has been suggested to use workloads that represent the same relative effort (Clausen, 1976; Rowell, 1993, 1974).

The degree of decline in VO$_{2\text{max}}$ with age is also affected by factors such as physical conditioning (Buskirk, & Hodgson, 1987; Hagberg et al., 1985; Heath et al., 1981; Ogawa et al., 1992), gender (Fleg, 1995; Ogawa et al., 1992), and body composition (Montoye et al., 1965; Poehlman, & Horton, 1990). For example, the age-related decline in VO$_{2\text{max}}$ in non-physically trained subjects average approximately 10% per decade between 25 and 75 years of age (Buskirk, & Hodgson, 1987; Dehn, & Bruce, 1972; Fleg, 1995; Ogawa et al., 1992). Further, the age-related decline in this population appears to be curvilinear rather than linear in nature, with an accelerated reduction after age 60 (Buskirk, & Hodgson, 1987). However, the degree of this decline in retired athletes (i.e., physical conditioning effect) who continue to train is approximately half (i.e. 5% per decade) of what has been observed in sedentary persons (Hagberg et al., 1985; Heath et al., 1981; Ogawa et al., 1992). This has also been confirmed in longitudinal studies that suggest that the rate of decrease in VO$_{2\text{max}}$ with age, at least in men, is up to 50% less in individuals who continue to perform vigorous aerobic-type exercise on a regular basis (Buskirk, & Hodgson, 1987; Fleg, 1995; Hagberg et al., 1985; Heath et al., 1981; Ogawa et al., 1992).

The scope of longitudinal work in this area indicates that: (1) VO$_{2\text{max}}$ can be fairly well maintained over phases of middle age (30-50 yr of age) lasting 10-20 years in men who continue to train vigorously (Hagberg, 1987, Kash et al., 1990; Marti, & Howald,
1990; Pollock et al., 1987; Rogers et al., 1990); (2) the greatest rates of decline in VO$_{2\text{max}}$ appear to occur in highly endurance-trained subjects who subsequently stop or markedly reduce their activity (Burskirk, & Hodgson, 1987; Kasch et al., 1990; Marti, & Howard, 1990; Powell et al., 1987); and (3) the average decline in VO$_{2\text{max}}$ with age in endurance athletes is essentially the same whether evaluated cross-sectionally or longitudinally (Saltin, 1986). In summary, the available data indicate a strong, inverse relationship between the intensity and/or volume of an individual’s habitual physical activity and the corresponding decline in VO$_{2\text{max}}$ (Dempsey, & Seals, 1995).

Gender also appears to influence the magnitude of decline in VO$_{2\text{max}}$ with age. While VO$_{2\text{max}}$ decreases in both men and women (Fleg, 1995, 1986; Hagberg et al., 1985; Ogawa et al., 1992; Sullivan, Cobb, & Higginbotham, 1991; Younis et al., 1990), the rate of decline in VO$_{2\text{max}}$, (expressed in ml$\cdot$kg$^{-1}$.$\cdot$min$^{-1}$ per year) appears smaller in women than in men (Burskirk, & Hodgson, 1987).

There is considerable controversy regarding the mechanisms responsible for the reduction in VO$_{2\text{max}}$ with age. Most of the available information has been obtained from cross-sectional investigations of either non-physically trained subjects of different ages or of highly trained masters athletes compared to young athletes. The latter have been used in an attempt to eliminate the influence of factors such as physical activity and body composition changes that occur with age in the general population. However, this model likely only minimizes such effects because older athletes cannot train at the same absolute intensities as their younger elite counterparts, nor do they all necessarily avoid changes in body composition. In some cases, the overall training volume of the masters athletes studied was only ~50-60% as great as that of the young athletic controls.
(Meredith et al., 1987; Rivera et al., 1989). Thus, some investigators have compared elite middle-aged and older athletes to non-elite young runners with similar levels of body fat, training volumes, and/or competitive performance (Fuchi et al., 1989; Hagberg et al., 1985). The limitation of this latter approach is that the non-elite young runners likely have inferior predisposition for endurance performance compared to the elite master runners.

With regard to the main determinants of VO$_{2\text{max}}$: maximal CO (i.e., maximal HR and SV) and maximal (a – v )O$_2$ diff, the consistent finding is that an age-related decrease in HR$_{\text{max}}$ reduces the maximal achievable CO, and therefore, VO$_{2\text{max}}$ (Hagberg et al., 1985; Ogawa et al., 1992; Rivera et al., 1989). Some data in endurance-trained women and/or men indicate that the decrease in VO$_{2\text{max}}$ is due to solely to the decline in HR$_{\text{max}}$ (Hagberg et al., 1985), whereas other findings suggests a contribution of only 25-40% from this mechanism (Ogawa et al., 1992). Such discrepancies are likely the result of different approaches to estimating maximal SV and/or of differences in the ages of the masters athletes studied (Ogawa et al., 1992). In studies concluding that the decline in HR$_{\text{max}}$ explains only a portion of the age-associated reduction in VO$_{2\text{max}}$, decreases in maximal SV were reported to explain up to 50% of the remaining difference (Ogawa et al., 1992).

Additionally, other studies have documented that total blood volume is lower in healthy older compared to young untrained men, and that these reductions are strongly related to the age-related decline in VO$_{2\text{max}}$ (Davy, & Seals, 1994). Therefore, VO$_{2\text{max}}$ and total blood volume are directly correlated in postmenopausal women who differ in their levels of physical activity (Stevenson et al., 1994). Because total blood volume
exerts an important influence on maximal SV and VO$_{2\text{max}}$ in young adults (Covertino, 1991; Coyle et al., 1986), it has been suggested that the age-associated decreases in blood volume might contribute to the lower maximal SV observed in older subjects. In any event, depending on the gender and chronic physical activity levels of the subjects, the reduction in maximal CO appears to explain between 50% and 100% of the total reduction in VO$_{2\text{max}}$ with age, with the remainder due to a lower maximal (a – v) O$_2$ diff (Fuchi et al., 1989; Hagberg et al., 1985; Ogawa et al., 1992; Saltin, 1986).

The influence of physical training on the age-related changes in these determinants of VO$_{2\text{max}}$ is not entirely clear. Some findings indicate that the apparent lesser rate of decline in VO$_{2\text{max}}$ with age in athletes who remain active is associated with smaller reductions in HR$_{\text{max}}$ and (a – v) O$_2$ diff compared with untrained subjects (Ogawa et al., 1992; Rogers et al., 1990). On the other hand, age-related decreases in maximal HRs have been reported to be similar in master athletes whose activity levels decreased over time compared to those who maintained their training levels (Pollock et al., 1987; Saltin, 1986). It is interesting to speculate that the apparent lesser decline in maximal (a – v) O$_2$ diff reported in highly trained older endurance athletes (Ogawa et al., 1992) is due to the maintenance of high levels of skeletal muscle oxidative capacity (Coggan et al., 1992; Rogers, & Evans, 1993; Saltin, 1986).

Finally, changes in body composition appear to play an important role in the age-related decline in VO$_{2\text{max}}$ in at least two ways. First, independent of any change in the absolute level of whole-body VO$_{2\text{max}}$ (i.e., L/min), an increase in body weight with age will result in a reduction in VO$_{2\text{max}}$ as traditionally expressed in mL kg min$^{-1}$. Second, at any given level of body weight, aging typically is associated with a decrease in fat-free
mass in general and skeletal muscle mass in particular, as well as with an increase in body fat content (Montoye et al., 1995; Poehlman, & Horton, 1990). Because the absolute VO\textsubscript{2max} is directly related to the size of the active skeletal muscle mass, these age-associated shifts in body composition will, independent of changes in cardiac pumping or peripheral oxidative capacities, cause VO\textsubscript{2max} to decline with age (Fleg, & Lakatta, 1988). Although the exact magnitude of the contribution differs among studies (Booth, 1989; Fleg, & Lakatta, 1988; Ogawa et al., 1992), it is clear that at least some of the age-related decrease in VO\textsubscript{2max} is due to changes in body composition, such as an increase in body fat content (Ogawa et al., 1992).

**Cardiac Output (Heart Rate, and Stroke Volume):** The absolute increase in heart rate in response to the same relative exercise stress (e.g., a particular % VO\textsubscript{2max}) becomes smaller with age (Kohrt et al., 1993; Sachs et al., 1985; Seal, 1993; Taylor et al., 1992). This smaller HR elevation in older adults appears to be appropriate for the absolute level of VO\textsubscript{2}, which is lower in the average older versus younger subjects at the same relative intensity of exercise. Further, during vigorous treadmill or cycle exercise in the upright position, it has been found a lower HR\textsubscript{max} in older adults as compared to younger individuals (Fleg et al., 1995, 1990b; Pollock, Wilmore, & Fox, 1978; for a review, see Gerstenblith, Lakatta, & Weisfeldt, 1976). Thus, HR\textsubscript{max} decreases progressively with advancing age (Fleg et al., 1995; Pollock, Wilmore, & Fox, 1978; Gerstenblith, Lakatta, & Weisfeldt, 1976), and the rate of decline is greater in men than in women (Hossack, & Bruce, 1982; for a review, see Dempsey, & Seals, 1995).

The lower HR\textsubscript{max} achieved during exercise in older individuals is not attributed to disease or a sedentary life-style, since a deficit of similar magnitude occurs both in
healthy sedentary men and women and in older athletes (Ehsani et al., 1991; Fleg et al., 1988; Heath et al., 1981; Lakatta, 1993). However, it has been suggested that an impaired responsiveness to beta-adrenergic stimulation (Conway et al., 1971; Seal et al., 1994b; Yin et al., 1976) and a decline in the intrinsic HR (Lakatta, 1993) both likely contribute. The decline in the intrinsic HR, as mentioned previously, may be due to morphological and electrophysiological changes in the sinoatrial node and other portions of the conduction system that result in reduced conduction velocity (Saltin, 1986).

In some studies, a reduced SV during submaximal and peak upright cycling exercise has been reported in older versus younger individuals (Granath et al., 1964; Julius et al., 1967; Kuikka, & Lansimies, 1982; Strandell, 1964). While in other studies, SV during vigorous upright cycling exercise has been equivalent in older and younger individuals (Fleg et al., 1995, 1990b; Higginbotham et al., 1986a, 1986b; Lakatta, 1993; Younis et al., 1990); in some others, SV has been greater in older individuals than in younger counterparts (Rodeheffer et al., 1984). Additionally, in studies performing progressive treadmill exercise (which might elicit a true VO\textsubscript{max}), significant reductions in SV with age in both nonphysically trained subjects and highly trained endurance athletes have been reported (e.g., Ogawa et al., 1992).

Different potential mechanisms have been suggested to explain age-related changes in SV. Those potential mechanisms include: (1) a reduction in LV filling; however, findings of age-associated increases in LV EDV (Fleg et al., 1995; Rodeheffer et al., 1984) and in filling pressures (Ehrsam, 1983) during cycling exercise specifically in active healthy men do not support this concept. It should be noted that increases in LV EDV do not appear to occur with aging in women (Fleg et al., 1995); (2) a decline in the
peak LV diastolic filling rate during both submaximal and maximal supine cycling exercise with age (Levy et al., 1993; Schulman et al., 1992); however, the lower exercise HRs of older adults may counteract this reduction in peak filling rate by increasing diastolic filling time; (3) an increase in LV wall stress and afterload (which impedes emptying), which may contribute to the age-related decline in SV (Ehsani, 1987), especially in older subjects who demonstrate elevated levels of SBP during exercise due to reduced arterial compliance; (4) impairment of LV contractile reserve after age 60, at least in men, as indicated by an attenuated increase in ejection fraction from rest to peak cycling exercise (Port et al., 1980); (5) an age-related reduction in the response to beta-adrenergic stimulation likely contributes to the impairment in systolic contractile performance (Schulman et al., 1992; Seals et al., 1994b; Stratton et al., 1992); and (6) an increase in LV stiffness and wall motion abnormalities, as well as a prolongation of myocardial contraction and relation time, could play a role in these age-related changes in the LV response to exercise (Ehsani, 1987; Lakatta, 1993, Dempsey, & Seals, 1995).

This heterogeneity of the SV observations contributes to variable CO results observed during upright cycle exercise among older individuals (Fleg et al., 1995, 1990b; Granath, Jonnson, & Strandell, 1964; Granath, & Strandell, 1964; Higginbotham et al., 1986a; Julius et al., 1967; Kuikka, & Lansimies, 1982; Ogawa et al., 1992; Rodeheffer et al., 1984). For example, Fleg et al (1995) report that the maximum CO during cycle-ergometry declines with age, which was explained by smaller HR increments in older individuals, whereas SVI did not decline with age in males or females; Rodeheffer et al (1984) found that CO was not related to age, and the age-related decrease in HR was compensated for an age-related increase in SV; and Ehram (1983), Granath et al (1964),
Julius et al (1967), Ogawa (1992), Strandell (1964) report that maximal CO is reduced with advancing age because of reduction in both $HR_{\text{max}}$ and $SV_{\text{max}}$. Therefore, as in young subjects, there is some evidence reporting that maximal CO is lower in older women than in older men, because of a lower stroke volume (Ogawa et al., 1992).

There is not a conclusive agreement on the effects of aging on SV and CO during exercise. It is due, in part, to that researchers have not specified, for example, whether CO was measured under submaximal or maximal exercise conditions, and whether the subjects measured were trained or untrained. In healthy aging individuals free of cardiac disease, the major age-related cardiovascular change is a decrease in maximum HR. Thus, older adults increase CO by a smaller increase in HR but a greater reliance on the Frank-Starling reflex mechanism (Mann, Denenberg, Gash, Makler, and Bove, 1986). Because the Frank-Starling reflex can be enhanced and used as a compensatory mechanism, the CO of healthy older individuals during exercise can be maintained (Fleg, 1986; Geokas et al., 1990; Lund-Johansen, 1988). However, persons with hypertension do experience an age-related decline in CO (Lund-Johansen, 1989; for a review, see Spirdusso, 1996).

Other potential sources of variation in SV and CO during exercise in older adults are: heart size, body composition, mode of exercise (e.g., cycling or walking), body position during exercise (e.g., upright, sitting, or supine position), technique used to measure CO (e.g., direct-Fick or indicator-dilution methodology, ventricular imaging methods, acetylene re-breathing procedure), and heart disease. For example, some studies report an age-related decline in the CO during maximum cycling exercise in the sitting position (Granath, Jonnson, & Strandell, 1964; Julius et al., 1967). However, the decline in CO,
which is more marked than that in Fleg et al (1990b) study, was due to a greater reduction in work performed by older individuals as compared to Fleg et al study. Furthermore, it should be mentioned that the SVI achieved during exercise in the healthy older, sedentary normotensive men and women in Fleg et al is determined largely by their respective heart volumes at rest (Fleg et al., 1990b). That is, a larger healthy heart, in older sedentary men delivers a larger SV both at rest and during exercise than the heart does in women. Lastly, cross-sectional and longitudinal studies in hypertensive men report a age-related decline in SVI during exercise. An effect that may, in part, be related to age-associated deficits in EDVI at rest in these individuals (Lakatta, 1993).

**End-diastolic and end-systolic volumes:** In studies reporting a reduced SVI during dynamic exercise in older individuals (e.g., Julius et al., 1967; Kuikka, & Lansimies, 1982), the EDVI and ESVI have not been measured and SVI has been calculated from the measured VO₂, HR, and CI. Thus, from these studies it is not known whether the failure of SVI to increase in older individuals to the extent that it does in younger ones is attributable to a relative reduction in venous return and EDVI, perhaps due in part to a markedly diminished myocardial compliance, or from failure of the ESVI to decrease to the same extent in elderly versus younger individuals. However, in studies in which cardiac volumes were measured (e.g., Fleg et al., 1995) in healthy sedentary individuals of a broad age range (22-86 yr of age) during upright cycle exercise, EDV at peak effort increased with age in men, but neither was related to age in women. Furthermore, when hemodynamics of the cardiac volumes were expressed as the change from rest to peak effort (an index of cardiovascular reserve function), both sexes demonstrated age-
associated increases in EDV and ESV. However, the exercise-induced reduction in ESV from rest was greater in men than in women.

In this same study (Fleg et al., 1985), in order to examine gender differences in the hemodynamic response at relative work rates from rest to peak effort in age-matched subsets, younger (<40 yr) and older (>60 yr), Fleg et al. found that the rate of increase in peak diastolic filling is similar in both older and younger individuals, and no deficit in EDVI during upright cycle exercise is observed in these individuals. In these healthy sedentary older men (>60 yr), the EDVI is higher at rest and during exhaustive upright cycle exercise as compared to younger (<40 yr) counterpart (Fleg et al., 1995). However, an age-associated increase in EDVI during exercise is not present in women (Fleg et al., 1995). Thus, it should be noted that the age-gender interaction in EDVI might confound the interpretations of cardiac volume responses to exercise in men and women across a broad age range (Higginbotham et al., 1984; Sullivan, Cobb, & Higginbotham, 1991; for a review, see Lakatta, 1993).

A greater end-diastolic dilation has also been observed during supine or semi-supine exercise in older versus younger individuals (Granath, Jonsson, & Strandell, 1964; Mann et al., 1986; Lakatta, 1993). However, studies in which the age range of subjects was truncated (20-50 yr) have failed to detect this age-associated increase in EDVI (Higginbotham et al., 1986a, & 1986b).

An enhanced EDVI during exercise in some older men, even in the absence of ventricular compliance changes, may be accompanied by an enhanced filling pressure. This may explain the observation that during exercise, older individuals whose filling pressure increased the most, also showed the greatest increase in SV (Granath, Jonsson,
& Strandell, 1964). However, it should be mentioned that the generation of a given ventricular pressure requires a greater ventricular wall stress (force/unit cross-sectional area). Thus, if the ventricular radius is increased, a greater level of myocardial contractility and energy consumption per stroke is required by the dilated aged heart. The age-associated increase in ventricular wall thickness, as noted above, reduces the magnitude of the increased LV stress due to ventricular dilation (Lakatta, 1993).

While EDVI and SVI are preserved or enhanced during exercise in healthy sedentary older individuals relative to younger ones, the reduction in ESVI during exercise (Fleg et al., 1995, 1990b) is diminished in both healthy older men and women (Fleg et al., 1995; Mann et al., 1986; Rodeheffer et al., 1984). This may be attributed, in part, to an apparent coupling that has been observed between the change in EDV and ESV in the transition between rests to exercise in individuals of any age (Rendlund et al., 1990). Alternatively, the increase in ESVI during exercise in older individuals may exceed that expected on the basis of the increase in EDVI and may reflect a relative reduction on myocardial contractile reserve, or a relatively greater increase in impedance to LV ejection with aging during exercise. Indeed, the ratio of ESVI and systolic arterial pressure, a crude index of myocardial contractility, although not reduced in older individuals at rest, does decrease as a function of age during exercise (Fleg et al., 1995).

The failure for SVI at peak exercise (Fleg et al., 1995, 1990b) to remain higher in older than younger men, as it is at rest, appears to be related to the failure for ESVI to decrease in older men to the extent that it does in younger men. Because the augmentation of the ejection fraction during exercise influences the extent to which the ESV becomes reduced (Renlund et al., 1990), it is not surprising that ejection fraction
increases less during exercise in older than in younger men and women (Fleg et al., 1995, 1990b; Rodeheffer et al., 1984). Because both the maximum myocardial contractile reserve and regulation of vascular impedance during exercise depend, in part, on the response to β-adrenergic stimulation, a deficit in the responsiveness to beta-adrenergic stimulation with aging could account for the failure of ESVI to decrease during exercise in older individuals to the extent that it does in younger ones.

As noted above, interactions of age, disease, and life-style (e.g., fitness) confound the interpretation of measures of CV performance, particularly during stress. In older individuals with occult coronary artery disease, the age-associated trends for LV end-diastolic dilation, and increased ESV, and a reduced ejection fraction during exercise are exaggerated (Fleg et al., 1990a; 1990c; Port et al., 1980; Lakatta, 1993).

Oxygen extraction. In older adults, as in younger individuals, the arteriovenous oxygen difference [(a - v)O₂ diff] increases with increasing rates of work and results from increased oxygen extraction from arterial blood as it passes through exercising muscle. At rest (a - v)O₂ diff is normally 4.5 ml of O₂ for every 100 ml of blood (ml/100 ml); and as that rate of work approaches maximal levels, the (a - v)O₂ diff reaches 15- to 16-ml/100 ml of blood (Rowell, 1993). However, during exercise, the (a - v)O₂ diff decreases with age (Dempsey, & Seals, 1995; Ogawa et al., 1992; Rogers et al., 1990; Lakatta, 1993), which contributes to the diminished aerobic capacity. The decrease is from approximately 16 volumes % in a 20 year old to 12-13 volumes % in a 65 year old (Brooks, Fahey, & White, 1995). The mechanisms suggested for a decreased (a - v)O₂ diff during exercise in older adults are: a reduction in the fiber/capillary ratio, total hemoglobin, the respiratory capacity of muscle, and the failure to shunt a larger portion
of the cardiac output away from areas with limited oxygen extraction, such as the skin and viscera. It is also suggested a decrease of muscle mitochondrial mass, along with decrease in several oxidative enzymes (Brooks, Fahey, & White, 1995; Dempsey, & Seals, 1995; Ogawa et al., 1992; Rogers et al., 1990; Lakatta, 1993).

**Arterial Blood pressure.** The arterial blood pressure (BP) response to submaximal and maximal exercise appears to be either unchanged (Fleg et al., 1995), or greater with advancing age (Granath et al., 1964; Julius et al., 1967; Martin et al., 1991; Montoye, 1984; Rodeheffer et al., 1984; Seals, 1993; Standell, 1964). Absolute levels of arterial BP, especially SBP, are higher during exercise in older adults as a result of their elevated resting levels (Granath et al., 1964; Julius et al., 1967; Montoye, 1984; Rodeheffer et al., 1984; Standell, 1964). Therefore, absolute SBP during maximal dynamic exercise has been found to be greater in older subjects, both men and women (Martin et al., 1991), or only in men (Fleg et al., 1995), but the magnitude of this age-related increase appears to be greater in women than in men (Fleg et al., 1995; Hossack, & Bruce, 1982). It has been suggested that because maximal CO is reduced with age, this elevated arterial pressure during maximal exercise must be mediated by a greater systemic vascular resistance (Gerstenblith et al., 1976; Hagberg et al., 1985; Julius et al., 1967; Ogawa et al., 1992; Strandell, 1964; for a review, see Dempsey, & Seals, 1995).

Studies on autonomic-cardiovascular regulation at rest and in response to exercise in young and older adults (Ng et al., 1993, 1994a; Davy et al., 1995; Taylor et al., 1992, 1992) have found that the adjustments in arterial BPs of the older adults to both brief (Taylor et al., 1992) and prolonged (Davy et al., 1995) dynamic exercise are similar to those observed in the young controls (Dempsey, & Seals, 1995). However, because
exercise intensity appears to be the primary determinant of the arterial BP response to exercise (Bezucha et al., 1982; Lewis et al., 1983; Reyes, 2001), studies on age-related changes in arterial BP regulation during exercise must consider the appropriate stimulus or levels of exercise (% maximum) at which to compare older and young subjects. Accordingly, studies that have reported greater increase in the absolute level of arterial BP with age, as compared older subjects and young controls at the same absolute submaximal exercise intensity or level of oxygen consumption (Granath et al., 1964; Julius et al., 1967; Montoye, 1984; Standell, 1964), the exercise stimulus represented a higher relative intensity in the older subjects. Thus, a greater BP response in older adults might suggest that arterial BPs were regulated appropriately (Dempsey, & Seals, 1995).

It is well known that with exercise primarily involving the upper body muscles, both SBP and DBP are higher than when the same absolute work is performed with the legs. Several explanations have been suggested for the apparently higher TPR during arm exercise compared with leg exercise requiring the same CO. One possibility is that because of the relatively smaller active muscle mass involved in arm exercise, the total size of the vascular bed is such that even when maximally dilated it effects a smaller drop in TPR than that with comparable exercise involving the leg muscles. A second possibility is that the exercise pressor response is most tightly coupled to the relative exercise intensity, and thus would be greater at a given absolute power output with arm exercise since this would represent a higher relative intensity than when performing the same exercise with the leg. A third possibility may relate to a greater relative involvement of the Valsalva or partial Valsalva maneuver with upper-body exercise (For a review, see MacDougall, 1994).
Simultaneous measurements of brachial arterial pressure and cardiac volumes during exercise have been made with respect to age only during upright cycle ergometry. During cycle exercise, unlike treadmill exercise, DBP increases as a result of involuntary isometric handgrip employed by subject during this procedure. The age-associated increases in brachial SBP at rest persist at a similar magnitude during cycle exercise (Fleg et al., 1990b); in some instances brachial SBP at maximum exercise increases more in older than in younger individuals than it does at rest (Julius et al., 1967). With respect to the arterial load on the LV, it is important to recall that, as noted above, the relationship between brachial and aortic arterial SBP in young individuals differs from that in older ones, due to the early return of reflected pulse waves in the latter. Specifically, when brachial pressures are of equal magnitude in younger and older individuals, the aortic pressure is greater in older than in younger individuals. The extent to which TPR is reduced and arterial BP and impedance increase during exercise depends on the maximum work capacity, which in part, depends on the physical fitness and on the interaction of the noncardiac, neuroendocrine, and metabolic factors involved. In some studies the reduction in TPR that occurs during exercise was noted to be less in older than in younger individuals, whereas in others (Fleg et al., 1990b) this age effect is minimal in men but substantial in women. During exercise in young humans, the aortic impedance does not appear to increase, probably due to an increase in the aortic diameter. In older individuals, a larger and further increase in aortic impedance during exercise than noted at rest, it may explain, in part at least, the observed age-associated differences in the pattern of ventricular ejection during exercise; however, the effect of age on vascular impedance during exercise has not been studied in humans (Lakatta, 1993).
**Blood flow.** There is not consistent evidence indicating an age-related impairment in the ability to augment BF to active skeletal muscle, at least during submaximal exercise. One study reports a lower absolute level of leg BF at the same absolute submaximal loads of cycling in middle-aged versus young male athletes (Wahren et al., 1974), but this observation is not supported by other findings (Carlson, & Pernow, 1961; Jasperse et al., 1994). A lower peak level of BF to active muscles would be expected with age because of the declines in the maximal achievable exercise intensity, maximal CO, and VO$_{2\text{max}}$ (For a review, see Dempsey, & Seals, 1995).

Of additional importance might be the exercise-evoked adjustments in the regional circulations that undergo vasoconstriction during exercise with age (Dempsey, & Seals, 1995). Exercising at the same relative exercise intensity (i.e., the same % of VO$_{2\text{max}}$), older adults might require an elevated TPR (reduced systemic vascular conductance). Thus, some, if not all, of these regions might undergo greater vasoconstriction. Accordingly, Taylor et al (1992) found older men demonstrated an augmented whole-forearm vasoconstriction response during brief cycling exercise at the same percentage of peak oxygen consumption relative to young controls. In fact, the average BF to forearm skin during steady-state exercise were not different in the two groups, suggesting that the greater reductions in BF to the forearms of the older men were mediated by correspondingly greater vasoconstriction in the non-active muscles of the forearm (Taylor et al., 1992). This was consistent with the augmented increases in norepinephrine concentrations in blood plasma obtained from antecubital veins of the older men during exercise (Dempsey, & Seals, 1995).
Plasma catecholamine (Sympathetic modulation of CV function): During maximal exercise, the sympathetic component is essentially the exclusive autonomic modulator, and a marked increase in catecholamine secretion occurs. The impact of β-adrenergic modulation of the HR and cardiac volume during exercise has been inferred when exercise is performed in the presence of β-adrenergic blockade. In young individuals, β-blockade does not influence the CO achieved during upright cycle exercise, but the hemodynamic profiles differ. In the presence of β-blockade, the increase in HR and the reduction in ESV are markedly less, but the EDV increases substantially, and this permits a larger SV than in the absence of β-blockade (Renlund et al., 1985). This hemodynamic pattern during acute β-blockade shows how the interaction among cardiac parameters maintains CO when a deficit in adrenergic modulation is present (Lakatta, 1993).

With age, there appears to be a diminution in the effectiveness of sympathetic modulation of CV responses to exercise, and this might contribute to many of the changes in the CV response to exercise in older adults (Fleg et al., 1995, 1990b), such as the decline in HR\textsubscript{max} and ejection fraction, and increase in EDVI and ESVI. For example, Fleg et al (1994) demonstrated that the influence of β-blockade on hemodynamic parameters in older adults during exercise is attenuated, supporting the notion that sympathetic control in older adults is diminished. The age-associated changes in EDVI and ESVI during upright cycle exercise are not apparent in the presence of propranolol, and the age-associated reduction in HR is markedly attenuated, due to a greater effect of β-adrenergic blockade to decrease the HR and increase cardiac dilation in younger than older subjects (Fleg et al., 1994). Furthermore, age differences in CO during exercise are smaller during β-adrenergic blockade (Conway, Wheeler, & Sannerstedt, 1971).
One of the possible explanations for an apparent diminution in the effectiveness of β-adrenergic modulation of CV performance during exercise in older individuals is that the secretion of high levels of norepinephrine (NE) or epinephrine (E) during exercise stress, as reflected in their plasma levels, may decline with advancing age (Lakatta, 1993). However, during exercise, or under other circumstances that require an adjustment in the CV performance variables from their basal levels, plasma concentrations of NE and E are increased rather than decreased in older versus younger subjects (Featherstone et al., 1987; Fleg, Tzankoff, & Lakatta, 1985; Goldsten'in et al., 1983). Although clearance of plasma catecholamine appears to be reduced in older individuals (Esler et al., 1981; Featherstone et al., 1987), excessive spillover into the plasma also occurs, and this, rather than a diminished clearance rate, best correlates with the increased plasma level (Featherstone et al., 1987). This lack of evidence for a reduced secretion of catecholamines during exercise in older individuals suggests that if a decline in tissue catecholamine occurs in humans, as it does with adult aging in animal models, it appears to be of little functional importance, at least for maintenance of neurotransmitter levels during short-term stress. It is noteworthy that older (66 yr average) endurance-trained individuals (cyclists) at a given submaximal work load also appear to have higher epinephrine and norepinephrine levels than younger (25 yr average) ones. However, when expressed at the same relative workload, no apparent age difference emerges (Lehmann, Schmid, & Keul, 1984). In contrast, in sedentary subjects, the age-associated increase in plasma NE at rest persists at all relative and absolute workloads, including the maximum workload (Fleg, Tzankoff, & Lakatta, 1985). In this regard, the observed similarities and differences in the hemodynamic pattern between younger and older
individuals at maximum exercise by Fleg et al. (1990b) also pertain to a common
exercise workload, e.g., 50% maximum (Fleg et al., 1995, 1990b).

Other explanation for the apparent age-associated reduction in adrenergic modulation
of CV function is that neurotransmitters are not as effective at the level of the target
organs. There is indeed a large body of convincing evidence to indicate that the HR,
vascular smooth muscle (VSM), and myocardial contractile responses to β-adrenergic
stimulation decline with age. One method to assess the postsynaptic response to
neurotransmitter is to infuse these substances at rest. In human, isoproterenol infusion
elicits a greater increase in the LV ejection fraction (Stratton et al., 1992) and CI
(Kuramoto et al., 1978, Stratton et al., 1992) in younger versus older individuals.
Infusion of E and NE into intact adults and senescent rats, or into young and adult cats
and rabbits, elicits a variety of complex changes in cardiovascular function (Frolkis et al.,
1979; Frolkis, Berzrokov, & Schevtchuk 1975). However, the specific adrenergic CV
effects cannot be ascertained from such studies (Lakatta, 1993).

Additionally, there is some evidence to indicate that parasympathetic control of CV
function changes with age, but the evidence is conflicting. Some studies in rats suggest a
more efficient cholinergic modulation with aging. The threshold of the negative
chronotrophic effects of vagus nerve stimulation and concentrations of acetylcholine
required to cause changes in myocardial contractility are decreased with advancing age in
rats (Frolkis et al., 1979; Frolkis, Berzrokov, & Schevtchuk 1975). Additionally,
significantly greater increases in guanosine 3’,5’-cyclic monophosphate in response to
submaximal doses of acetylcholine have been observed in hearts of 24- to 26-months old
rats compared with 6- to 8-months old rats. This difference persisted in the presence of
acetylcholinesterase blockade, suggesting that the mechanisms of the age-associated differences are at or distal to the receptor (Kulchitsskii, 1980).

Other studies have observed an enhanced sensitivity to the direct chronotropic action of acetylcholine in right atria isolated from aged versus younger Fischer rats but have attributed to an age-associated reduction in cholinesterase activity (Kennedy, & Seifen, 1990). In contrast, other observations indicate an age-associated reduction in the response to parasympathetic agonists. In one such study a decrease in HR reduction in old rats to both vagal nerve stimulation and to bolus injections of methacholine was observed (Kelliher, & Conahan, 1980). More recent studies have indicated that the number of cholinergic receptors in the LV decreases with age in rats, as does the response to acetylcholine (Chevalier et al., 1991). Finally, a marked reduction in acetylcholine content of atrial tissue from senescent rats has been demonstrated (Verkhratsky, 1988). In humans, as noted above, a decrease in HR variability at rest or in response to a postural stress in older individuals has been attributed, in part, to a reduction in parasympathetic tone with aging. While age-associated differences in the extent of parasympathetic tone affect the interpretation of most studies that have infused β-adrenergic agonist into young and old humans or animals (and vice versa for infusion or parasympathetic agonists), it has been demonstrated in senescent versus younger adult beagle dogs that the HR$_{\text{max}}$ increase in response to isoproterenol infusion in the presence of full vagal blockade with atropine decreases with aging (Yin et al., 1979). In contrast, the HR$_{\text{max}}$ that could be elicited by external electrical pacing, which was far in excess of that elicited by isoproterenol infusion, was not age related (Lakatta, 1993).
2.2.2 Acute Cardiovascular Response to Anaerobic Work

Anaerobic work is defined as any physical activity or task in which the energy required is supplied through anaerobic or nonrobic metabolic (phosphagen and glycolisis) pathways. This subset of activities includes, but is not limited to, static or isometric exercises (lifting or pushing heavy weights and contracting muscles against fixed objects) and dynamic resistance or strength-developing exercises (weightlifting). In static exercise, skeletal muscle contraction causes principally a change in tension with little change in length; that is, a sustained force production with muscles remaining at a constant length. There is no cycling or muscle pumping effect between an active and relaxed state. Resistance exercise (RE) (e.g. weightlifting) is often characterized by a combination of static and dynamic exercise. Typically, each movement begins with a static contraction (until force production exceeds the weight of the object to be lifted), and is followed by concentric and/or eccentric contractions and finally a brief relative relaxation before the initiation of the next repetition (for a review, see MacDougall, 1994; Mitchell & Wildenthal, 1974; Surgeon General Report, 1996).

Detailed reviews on responses to anaerobic work are available (Kraemer & Daniel, 1986; MacDougall, 1994; McCartney, 1999; Mitchell, 1990, 1985, 1983; Mitchell & Wildenthal; 1974), and are only briefly summarized herein. Accordingly, this subsection first provides an overview of the CV adjustments to static and dynamic RE in healthy-younger adults. This is followed by a discussion of CV adjustments during RE in older adults, that will be limited to dynamic RE performed with large muscle groups. CV adjustments to isometric exercise in older adults will not be discussed.
Circulatory and left-ventricular response to isometric exercise: Early studies using partially static exercise assumed that BF to working muscle could be subjected to mechanical occlusion of the artery by the contracting fibers, and reported increased VO\(_2\) and CO after the cessation of heavy exertion (Lindhard, 1920). Later, it was reported that mild tachycardia and dramatic increases in arterial BP accompany such exertion (Alam and Smirk, 1937 & 1938). In addition, Asmusen, & Hansen (1938) reported that isometric exercise in the leg produced increases in VO\(_2\), CO, HR and BP, and that the cessation of the exercise was normally accompanied by a rapid return to normal HR and BP even though CO and VO\(_2\) might remain elevated. However, Alam and Smirk (1937 & 1938) observed that the return of HR and BP toward control values could be delayed by circulatory occlusion to the exercising limb (Alam and Smirk, 1937 & 1938). In the years that followed, many of these earlier observations were confirmed, and the differences between the effects of static and dynamic exertion were further examined (Tuttle and Horvath, 1957). However, it was not until the 1960s, that an extensive and comprehensive investigation of the special characteristics of isometric effects was undertaken. The series of the papers that appeared from the laboratories of Lind, Donald, and colleges (1963; 1964; 1966; 1967; 1968; 1968; 1970), along with that of Freyschuss (1970) remain the primary sources of basic information on the physiology of CV responses to isometric exercise (Mitchell and Wildenthal, 1974).

Static or isometric exercise involves the development of muscular tension and strength, usually against resistance. A sustained static contraction of even a small muscle group, such as the forearm muscles during handgrip dynamometry, invokes a ‘pressure load’ on the heart (Mitchell and Wildenthal, 1974). This (static exercise) is characterized
by a moderate increase in HR and CO, minimal change in peripheral vascular resistance, and a substantial rise in mean arterial pressure due to an increase in SBP and DBP (Hanson and Nagle, 1987). Whereas other ventricular volumes show little change, there are increases in the end-systolic volume (Sullivan et al., 1992), and indexes of myocardial contractility (Sagiv et al., 1985; Sullivan et al., 1992; Vitcenda et al., 1990). The increased CO results from an increased HR (McCartney and McKelvie, 1996; McCartney, 1999; Mitchell and Wildenthal, 1974; Sagiv et al., 1985).

When static contractions are performed at increasing percentages of maximum-voluntary contraction force (MVC), there is a proportional increase in intramuscular compression that eventually will exceed muscle perfusion pressure and occlude local blood flow (BF). Measurements of the intensity at which BF becomes impeded by the contraction range from approximately 40% - 60% MVC and varies considerably among muscle groups (Mitchell et al., 1980; Bonde-Petersen et al., 1975). Up to this point, BF is maintained by a marked pressor response that serves to adjust perfusion pressure in relation to the increasing intramuscular pressure, although the mechanisms that activate and control this response are not fully understood (see below for details). This increase in BP is largely the result of an increase in CO and to a lesser extent a reflex vasoconstriction in vascular beds other than those of the exercising muscles (Nutter & Wickliffe, 1981; Shepherd et al., 1981). In addition, if the desired force production is sufficient to elicit a Valsalva maneuver, the elevation in intrathoracic pressure also will be transmitted directly to the arterial tree (MacDougall et al., 1992). Because of the mechanical compression of the blood vessel in the contracting muscles, peripheral resistance does not decrease as in dynamic exercise and DBP increases in proportion to
the increase in SBP resulting in large increases in mean BP (Bezucha et al., 1982; Kimbol, & Brundil, 1976; Lind et al., 1964; Seals et al., 1983). Thus, the increases in SBP and DBP, and more specifically the hemodynamic responses to static exercise, are: almost directly proportional to the relative intensity of the contraction (Freyschuss, 1970; Lind, McNicol, & Donald, 1966; Lind and McNicol, 1967; MacDougall et al., 1992; McArdle et al., 1991; Mitchell, 1990; Seals et al., 1983); affected by the size of the active muscle mass (Blomqvist et al., 1981; Edwards & Wiles, 1981; Humphreys & Lind, 1963; MacDougall et al., 1992; McCloskey & Streatfeild, 1975; Mitchell, 1990, 1981; Seals & Hagberg, 1984; Shepherd et al., 1981); related to the duration over which the contraction is sustained (Lind, McNicol, & Donald, 1966; Mitchell, 1990; Seals & Hagberg, 1984; Shepherd et al., 1981); and influenced by the joint angle at which the work is done (MacDougall et al., 1992, MacDougall, 1994).

Although the mechanisms that activate and control this response are not fully understood, it has been suggested that the graded response to static exercise might involve either a central command or feed-forward behavior (Goodwin, McCloskey, & Mitchell, 1972; McCloskey, 1981, Mitchell, 1985, 1990), or a reflexive-peripheral feedback from the active muscles (Alam, & Smirk, 1937, 1938; Kao, & Ray, 1954; Mitchell, 1985, 1990; Patterson, 1928), or both. The cerebral or “central command” factor is a direct effect of the motor cortex on medullary CV centers. The BP increase is partially related to the intensity of the “command” (i.e., the subjective effort exerted), which probably affects the number of motor units recruited. One peripheral mechanism involves the somatic afferent fibers, which activate the exercise pressor response in a manner similar to that in dynamic exercise. The reflexive-peripheral feedback from the
active muscles may be additional peripheral reflex factors initiated by metabolic or
chemical agents (e.g., K ions) liberated in the muscle tissues. Alternatively, it has been
suggested that both central command and reflective peripheral mechanisms
simultaneously might be responsible for the pressor response during static exercise
component originating in the supraspinal areas of the brain and a peripheral or reflex
neural component originating in the contracting muscles and transmitting along group III
and IV muscle afferent to cardiovascular control centers (Leonard et al., 1985;

**Circulatory and left-ventricular response to resistance exercise:** Resistance
exercise, or weight-lifting exercise, is a combination of static and dynamic contractions,
the proportions of each varying in accordance with the degree of effort required to lift the
weight. At the initiation of the movement, there is a static contraction until the muscle
force exceeds the weight of the object to be lifted. This is followed by a dynamic
concentric (shortening) contraction to raise the weight, a dynamic eccentric (lengthening)
contraction to lower it, and a variable relaxation phase between successive lifts (Haslam
et al., 1988; Lentini et al., 1993; MacDougall et al., 1985; McCartney, 1999; McCartney
et al., 1993; McCartney and McKelvie, 1996). According with these RE phases
(concentric, lock, and eccentric), different circulatory and left-ventricular responses have
been established, and some contributing factors have been identified (Lentini et al., 1993;
MacDougall et al., 1985, 1992; McCartney, 1999; Miles et al., 1987).

The first investigation (MacDougall et al., 1985) to measure HR and intrabrachial
artery pressures during heavy RE in young subjects involved single-arm, and single- and
double-leg exercise to failure at intensities ranging from 80 to 100% of the one-repetition maximum (1-RM) (MacDougall et al., 1985). The results demonstrated that SBP and DBP rose rapidly to extremely high values during the concentric contraction phase for each lift and declined with the eccentric contraction. The findings also revealed large, pulsatile swings in arterial pressure throughout each repetition, and in one subject performing double-leg press the peak SBP and DBP reached 480/350 mmHg. The major mechanisms underlying the dramatic increases in arterial BP were assumed to be a potent pressor response, mechanical compression of the skeletal muscle vasculature, and an elevated intrathoracic pressure coincident with the Valsalva maneuver. The work of MacDougall et al (1985) was followed-up by others investigations (e.g., Miles et al., 1987; Lentini et al., 1993) who examined the changes in ventricular volumes during exercises in young men lifting heavy weights until failure. Miles et al (1987) used impedance cardiography during knee extension exercise to demonstrate that the average CO was unchanged throughout the protocol. However, the analysis of the changes in CO and SV throughout different phases of the lift revealed that both CO and SV were smaller during the concentric phase than the eccentric phase. There were also increases in HR, arterial pressure, and total peripheral resistance. The increase in arterial pressure was due to an increase in both SBP and DBP. Since CO remained relatively constant; it was suggested that BP changes were due to an increase in total peripheral resistance. Thus the maintenance of CO throughout the exercise period, despite the decrease in SV, was attributed to the increase in HR.

A more recent study (Lentini et al., 1993) extended previous observations by including a more detailed analysis of the changes in arterial pressure and LV volumes
throughout the various phases of bilateral leg press exercise, performed at 95% of the 1
RM to failure. During the initial concentric phase, the CV response was similar to that
during a static effort. There were significant increases in mean arterial pressure and total
peripheral resistance, and EDV and ESV declined by 30% and 50% respectively. The 17-
mL reduction in SV was not significantly different from rest, and an almost fourfold
increase in the peak SBP to EDV ratio indicated a notable enhancement of myocardial
contractility. In the lockout phase, there was a decline in SBP, DBP and HR and an
increase in EDV, and SV to control values. It was only the ESV that had not returned
close to resting levels, remaining significantly lower (16mL). During the eccentric phase,
responses were qualitatively similar to those in the concentric phase, but of a lesser
magnitude. SBP, DBP and HR tended to increase, whereas EDV, ESV, and SV tended to
decrease. The large increases in SBP (270/183 mmHg) during the early concentric phase
were countered by a substantial increase in myocardial contractility and by a reduction in
LV transmural pressure resulting from a large increase in intrathoracic pressure (58±24
mmHg) coincident with the Valsalva maneuver (Lentini et al., 1993). Such rapid changes
in arterial pressure, cardiac volumes, and myocardial contractility during the course of a
single-repetition of leg press exercise were attributed to the varying degrees of effort
(central command) required in each phase of the lift, being greatest in the concentric
(weakest) phase and least during lock-out (strongest phase). These results are consistent
with several other reports (Amussen, 1981; Hanson and Nagle, 1987; Sullivan et al.,

The assessment of hemodynamics immediately following RE in healthy young adults
and athletes has also been addressed (Fleck, 1988; Gettman et al., 1978; Stone et al.,
For example, Brown et al. (1991) using echocardiography to assess changes in cardiac function after the completion of moderate-resistance exercise (60% of 1-RM) report an increase in SV and contractility. Unfortunately, arterial BP was not reported. The absence of hemodynamic data makes it difficult to determine the extent to which changes in SV were due to alterations in preload, afterload, or contractility, particularly when one considers the potential influence of a rapid decrease in systemic arterial BP that is thought to occur as RE is terminated (Wieck et al., 1990). Furthermore, it is not recommended the use of immediate post-exercise echocardiography to assess maximal function; even though, functional parameters remain near maximally or even supra-maximally elevated into the recovery period.

More recently, MacDougall et al. (1992) demonstrated that the magnitude and pattern of CV responses to RE are influenced by several important factors. These include: (1) number or repetitions, (2) absolute or relative load, (3) muscle mass or mode or exercise (leg press versus leg extension), (4) joint angle, and (5) salva (for a review, see McCartney, 1999).

The first factor mediating the CV response to RE is the number of repetitions. It was observed that the peak arterial pressures in the initial repetition of a set of lifts exceed those of the next two or three repetitions, then the pressures rise progressively, reaching the highest values during the final repetitions of a set ending in failure. This finding has been corroborated by Lentini et al. (1993). Over the same time period, HR may attain levels of 160-170 beats/min. Subsequently, within 1-2 seconds after the final lifts, the pressures decrease to resting values or below.
The pattern of change in arterial BP throughout a set of lifts was explained as follows. In the first repetition the initial concentric contraction is not preceded by an eccentric contraction and thus does not benefit from the stretch-shortening effects (Komi, 1992). In subsequent repetitions, the eccentric contraction reduces the voluntary effort (thus the motor unit activation) required in the following concentric contraction and the arterial BP is lower (Sale et al., 1993 & 1994). As the muscle becomes fatigued, arterial BP progressively rises, due most likely to a combination of factors. Those factors include a greater voluntary effort, a greater recruitment of motor units and recruitment of accessory muscles, and an increased use of the Valsalva maneuver. As to the former, feedforward and feedback mechanism activity (i.e. from muscle ergoreceptor and nociceptor) (Mitchell, 1985) are likely involved in the recruitment of additional motor units. The rapid post-exercise drop in pressure that accompanies this type of dynamic RE may reflect an abrupt perfusion of the vasodilated exercising musculature that had previously been partially occluded. In addition, reflexive to a temporary, baroreceptor mediated pressure responses may contribute to a subsequent to very high pressures during the final lift (MacDougall et al., 1985).

The second contributing factor to CV responses to RE is absolute and relative load. Within subjects, HR and arterial BP responses increase in proportion to the load. The values attained during a single maximum lift, however, are less than those recorded in a set of heavy lifting to failure. Between-subject comparisons among individuals with markedly different lifting capacities indicate that it is the relative, rather than absolute load, which predicts the circulatory response (MacDougall et al., 1992). This supports the argument that a feed-forward central command mechanism, reflecting the central activity
for the recruitment of motor units (Mitchell, 1990), is the major contributor to the circulatory response during RE (MacDougall et al., 1992; McCartney et al., 1993).

The third determining factor is the effect of *muscle mass*. It has been found that within individuals there appears to be a positive but nonlinear relationship between the circulatory responses and the muscle mass engaged in the lifting (MacDougall et al., 1992; McCartney et al., 1993). For example, HR and arterial BP are higher during bilateral-leg press than unilateral-leg press exercises but nowhere near double. On the other hand, comparisons between-subjects suggest that variations in muscle mass do not predict interindividual differences in the circulatory response to RE. For example, MacDougall et al (1992) found similar arterial BP during a 10-RM of double-leg press lifting despite extremely large interindividual variability in total quadriceps cross-sectional area and the total weight lifted. This indicates that the circulatory responses to RE are determined in large part by the relative intensity of effort, as this would have been the same for each subject during the completion of an equal number of repetitions to failure.

The fourth factor is *Joint Angle*. The effect of joint angle on the circulatory response to RE has been investigated in studies using double-leg press exercise (e.g., Lentini et al., 1993). At the beginning of the movement, the knee joint angle is 90°, and the leg extensors are at their weakest point on the strength curve. It is in this early phase that the rise in arterial pressure is the greatest. At lockout, the knee joint angle is 170°, the strongest position for the leg extensors, and the arterial pressure is reduced almost to resting. During the eccentric lowering phase, the arterial pressure rise again, but significantly less than during the initial concentric contraction. The most likely
explanation for this observation is related to the relative effort required in each phase of
the lift. Muscles can generate appreciably less force during a concentric contraction than
when contracting eccentrically (Komi, 1973). Thus, lifting a given weight will require a
greater relative effort than lowering it, and this will be reflected in the magnitude of the
circulatory response (Lentini et al., 1993; MacDougall et al., 1992; McCartney et al.,
1993; Sale et al., 1994; Sale et al., 1993).

The fifth factor mediating the CV response to RE is Valsalva maneuver. It has been
observed that at higher intensities of lifting (~80% or more of 1-RM), the Valsalva
maneuver becomes almost obligatory and helps to stabilize the trunk and facilitate the
necessary force production. In very heavy lifting to failure, the intrathoracic pressure may
exceed 100 mmHg, equivalent to 60% or more of the level that can be generated in a
maximum voluntary Valsalva maneuver (MacDougall et al., 1992). During lifting to
failure with submaximal loads, the Valsalva is used increasingly as the muscles become
fatigued. The rise in intrathoracic pressure resulting from the Valsalva maneuver is
transmitted directly to the arterial tree, causing an immediate increase in arterial pressure
(Hamilton et al., 1944). For this reason, and because it may also reduce venous return, the
Valsalva maneuver is often contraindicated during RE and while performing isometric
efforts (AACPR, 1995; ACSM, 1991). However, more recent evidence suggests that an
initial, brief Valsalva may be beneficial during RE. Lentini et al (1993) reported mean
values of intrabrachial artery pressure of 270/183 mm Hg during the early concentric
contraction phase of double-leg press exercise at 95% of 1-RM in healthy young men. At
the same time, the intrathoracic pressure increased to 59 ± 25 mm Hg as a consequence
of the Valsalva maneuver. Under most circumstances, the SBP would be representative
of the LV afterload, as intrathoracic pressure is usually low (Braunwald, 1997). In this investigation, however, it was concluded that the rise in intrathoracic pressure resulted in a lower LV transmural pressure, hence afterload, than would be predicted from the arterial pressure measurements alone (Lentini et al., 1993). As increases in intrathoracic pressure are also transmitted directly to the cerebrospinal fluid (Hamilton et al., 1944), a brief Valsalva may also serve a protective effect by reducing the transmural pressure across cerebral vessels (MacDougall et al., 1992).

Lastly, it should be noted that studies in healthy individuals (Lentini et al., 1993; MacDougall et al., 1985; MacDougall et al., 1992; McCartney et al., 1993) and patients with coronary artery disease (Haslam et al., 1988; Wiecek et al., 1990) have reported qualitatively similar findings during RE. Within subjects, the HR and arterial BPs increase over repetitions, in proportion to the relative load and the amount of muscle mass activated. The effect of muscle mass becomes more obvious during lifting at higher intensities (For a review, see McCartney and McKelvie, 1996). Furthermore, although high-intensity RE poses a potential risk to hypertensive patients and to those with CV disease, research data suggest that the risk is relatively low (Gordon et al., 1995) and that hypertensive persons may benefit from RE (Tipton 1991; American College of Sports Medicine, 1993; Surgeon General Report, 1996).

In summary, RE has been described as a series of near-maximal static contractions to overcome the inertia of the weight interspersed by forceful dynamic concentric and eccentric contractions of the muscle group (MacDougall et al., 1985). The changes in cardiac volumes, BP, and contractility observed are consistent with this description (Lentini et al., 1993; Amussen, 1981; Hanson and Nagle, 1987; Sullivan et al., 1992;
Vitcenda et al., 1990). Thus, muscle contraction during RE consists of both a static and
dynamic component so that it produces a different hemodynamic response. Most of the
studies of the hemodynamics of RE have involved healthy young adults and athletes,
with measurements performed after exercise (Fleck, 1988; Gettman et al., 1978; Stone et
al., 1983), whereas other studies have examined the response during RE in healthy
individuals (Lentini et al., 1993; MacDougall et al., 1985; Miles et al., 1987) or patients
with coronary artery disease (Haslam et al., 1988; McKelvie et al., 1990; Wiecek et al.,
1990). These latter studies (those performing the measurements during exercise) have
noted a phasic BP response with an increase in pulse pressure during exercise that differs
from that observed during static exercise (MacDougall et al., 1985; Miles et al., 1987). In
a study where intra-arterial BP was measured in experienced weight lifters performing
near-maximal leg press exercise to fatigue (MacDougall et al., 1985), significant
increases in systolic, diastolic, pulse pressure (up to 480/350 Torr) were observed. In
addition, it has been observed that the performance of a brief Valsalva maneuver is an
integral and unavoidable component of maximal or near-maximal contractions. The
increased intrathoracic pressure that results from this maneuver exaggerates the increase
in BP and would be expected to compromise cardiac filling (MacDougall et al., 1992).

2.3 General Comments about Efficacy of Exercise Training

It has been well established that both aerobic exercise training (Clausen, 19977;
Dempsey, & Seals, 1995; Ehasani, 1987; Ekblom, 1968; Fleg, 1986; Hagberg, 1987,
1994; Kraemer, & Daniels, 1986; Lakatta, 1993; Saltin, & Åstrand, 1967; Saltin, 1986)
and anaerobic exercise training (Fiatarone et al., 1992, McCartney et al., 1993; Sale et al.,
1994) induce specific physiologic adaptations. The magnitude of these adaptations
depends largely on type of muscular activity or mode of exercise, the intensity of
training, the frequency and duration of the exercise training sessions, and the body’s
initial level of fitness (for a brief review, see Surgeon General Report, 1996).

2.3.1 Chronic Cardiovascular Adaptations to Aerobic Exercise Training

The CV adaptations to aerobic exercise training in young (Bevergard, & Shepherd, 1967;
Clausen, 1977; Ekblom, 1968; Rowell, 1974; Saltin, & Åstrand, 1967; Scheuer, & Tiptor,
1977) and older adults (Dempsey, & Seals, 1995; Ehsani, 1987; Fleg et al., 1986;
Hagberg, 1987, 1994; Lakatta, 1993; Saltin, 1986; Spirdusso, 1996) has been previously
reviewed, and is summary herein.

Cardiovascular Adaptations to Aerobic Exercise Training in Younger Adults:

Maximal oxygen consumption (VO$_{2\text{max}}$) is the measurement most commonly used to
assess CV fitness. Revisiting the Fick equation, we see that VO$_{2\text{max}}$ is equivalent to the
product of maximal CO and (a – v) O$_2$ difference:

\[
\text{VO}_2\text{max} = \text{Cardiac Output}_{\text{max}} \cdot (a - v) \text{O}_2 \text{ difference}_{\text{max}}
\]

Since cardiac output is equal to HR times SV, oxygen consumption at any level of
exercise can be calculated as:

\[
\text{VO}_2 = (HR \cdot SV) \cdot (a - v) \text{O}_2 \text{ difference}
\]

Thus, aerobic exercise training might result in a change in VO$_{2\text{max}}$ by altering any one of
these three variables: HR, SV, and (a – v) O$_2$ difference. Accordingly, a number
adaptation to aerobic exercise training in healthy young and middle-aged individuals
have been documented (Bevegard, & Shepherd, 1967; Clausen, 1977; Crawford, Petru, &
Rabinowitz, 1985; Ekblom, 1968; Pollock et al., 1972; Rowell, 1974; Saltin, & Åstrand,
1967), including central and peripheral CV mechanisms (Blomqvist, & Saltin, 1983; Clausen, 1969, 1976; Crawford, Petru, & Rabinowitz, 1985; Lakatta, 1993).

Cardiac output at rest and during submaximal exercise is essentially unchanged following aerobic exercise training (Åstrand, & Rodahl, 1986; Brooks, Fahey, & White, 1995; Keul et al., 1982). However, at or near maximal rates of work, CO is increased substantially, up to 30 percent or more (Saltin, & Rowell, 1980; Spina et al., 1993, 1992). After endurance training, SV is increased at rest, during submaximal exercise, and during maximal exercise (Seals et al., 1994; Spina et al., 1993, 1992). Finally, post-training HR is decreased at rest and during submaximal exercise and is usually unchanged at maximal rates of work (Clausen, 1977; Pollock et al., 1972; Spina et al., 1993, 1992; Yoshida et al., 1982).

Factors contributing to the increase in SV as a result of an aerobic exercise-training program include changes in contractility, cardiac loading conditions, or both (Ross, 1976). Hence, the training-induced increase in SV can be due to: (1) an enhanced inotropic state, which can be the result of enhanced sensitivity to catecholamines, a larger preload (EDV), or more elusive intrinsic changes in the myocardial tissue; (2) a larger total BV and EDV, which not only contributes to contractility via Frank-Starling, but also by definition suggests a greater pre-systolic LV volume; (3) a reduced LV end-systolic wall stress; or (4) any combination of these variables (Ross, 1976; Spina, 1999).

An enhanced sensitivity to catecholamines (β-adrenergic mediated increases in LV contractile function and diastolic filling) can lead to the greater SV observed in the trained state (Hopkins et al., 1996; Mole 1998; Spina et al., 1992; Wyatt et al., 1978). Studies in experimental animals (Mole, 1998; Wyatt et al., 1978) and humans (Hopkins
et al., 1996; Spina et al., 1992) support this hypothesis, but other studies do not (e.g., Martin, Coyle, & Ehsani, 1984; Stratton et al., 1992). For example, Mole (1998) found that at the same concentration of isoproterenol, exercise-training evoked a greater rate of tension development of rat cardiac muscle. Wyatt et al (1978) demonstrated improvement of both myocardial contractility and adenylate cyclase activity in response to isoproterenol after training in cats. In response to 12 weeks of aerobic exercise training, Spina et al (1992) observed a larger increase in LV fractional shortening and greater decrease in end-systolic dimension at comparable changes in wall stress with no concomitant changes in preload induced by isoproterenol. On the other hand, Stratton et al (1992) did not observe any change in the inotropic response to isoproterenol after training in young men. Martin, Coyle, & Ehsani (1984) did not find any changes in the fractional shortening response to one dose of epinephrine in highly trained subjects after cessation of training (For a review, see Spina, 1999). Differences in protocols, subject participating, and/or exercise-training program may reconcile these disparate findings.

Additionally, increases in $SV_{\text{max}}$ might be as a result of a hypertrophy of the cardiac muscle fibers (i.e., increase in size of each fiber), allowing a greater force to be exerted with each beat of the heart (George, Wolfe, & Burggraf, 1991; Surgeon General Report, 1996).

It has been reported that aerobic exercise training increases plasma volume approximately by the same percentage that it increases SV (Covertino, 1991; Green, Jones, Painter, 1990; Surgeon General Report, 1996). An increased plasma volume increases the volume of blood available to return to the right heart and, subsequently, to the LV. Thus, there is an increase in EDV. (Seals et al., 1994). This acute increase in the
LVEDV (ventricular dilatation) also contributes to greater pre-load and greater myocardial elastic recoil (an enhanced inotropic state).

A larger EDV might be also the result of LV volume overload or eccentric hypertrophy (proportional increase in chamber size and wall thickness with maintenance of a normal LV wall thickness-to-radius ratio) (Grossman, Jones, & McLauren, 1975; Maron, 1986; Spina, 1999). Thus, it has been hypothesized that eccentric hypertrophy plays a major role for the increase in SV at maximal exercise in response to endurance training (Blomqvist, & Saltin, 1983; Clausen et al., 1976). Cross-sectional (e.g., Hopkins, Spina, & Ehasani, 1996; Longhrst et al., 1980; Milliken et al., 1988) and longitudinal (e.g., Celemajer, 1997; Colan et al., 1985; Ehsani, Hagberg, & Hickson, 1978; Spina et al., 1992) studies have demonstrated a greater LV mass in highly trained competitive athletes that explains for 40% of the variance in VO$_{2\text{max}}$, and a relationship between the training-induced increase in end-diastolic dimension at rest, and SV at VO$_{2\text{max}}$. At present there does not appear to be a consensus regarding mechanisms that may account for training-induced eccentric hypertrophy. However, it has been suggested that the increase in volume that the left ventricle shows may play a role for the increase in sarcomeres in series that characterize volume-overload or eccentric hypertrophy. In addition, the increase in blood volume in response endurance exercise training (Covertino, 1991) might mediate the LV hypertrophy. However, it is not known how much of a role the increase in blood volume plays for mediating changes in the adaptive hypertrophic process (For a review, see Spina, 1999).

Augmented LV filling (Matsuda et al., 1983; Levy et al., 1993) mediated by increased sensitivity to cathecholamines (Hopkins et al., 1996; Mole 1998; Spina et al., 1992;
Wyatt et al., 1978), greater blood volume (Covertino, 1991), enhanced venomotor tone, altered diastolic properties of the myocardium (Colan et al., 1985; Lorell, & Grossman, 1987; Nixon et al., 1982), or a combination of these factors can contribute to the higher SV at maximal exercise in the trained state (Spina, 1999). For example, Matsuda et al (1983) and Levy et al (1993) demonstrated an augmentation in early diastolic filling in endurance athletes, and an enhanced LV filling dynamics during exercise (reflected in an increase in absolute values for peak filling rate) in young men in response to endurance training, respectively. Subsequently, the same group of researchers reported that the improvement in LV filling after training was not mediated by enhanced responses to β-adrenergic stimulation (Stratton et al., 1994). More recently, Hopkins et al (1996) demonstrated a higher ratio of peak early filling (E) velocity to peak late filling velocity (A) of the transmitral Doppler flow in response to dobutamine in endurance athletes, as compared to sedentary controls at similar heart rates. However, in contrast to Stratton et al, Hopkins et al results suggest that the improvement in LV filling dynamics is compatible with enhanced sensitivity to sympathetic stimulation in the trained stated (For a review, see Spina, 1999).

In summary, the increase in maximum SV, and consequently in maximum CO, is due to a cardiac dilatation at end-diastole and end-systole, an increase in cardiac mass or eccentric hypertrophy (Grossman, Jones, & McLauren, 1975; Maron, 1986; Milliken, 1988), and an enhanced circulating blood volume (Covertino, 1991; Rerych et al., 1990). Cardiac alargement occurs at both end-diastole and end-systole; the former is greater than the latter, resulting in an augmentation of SV (Ginzton et al., 1989, Lakatta, 1993).
Another important CV adaptation to aerobic exercise training (peripheral adaptations) is an enhanced (a – v) O₂ difference at maximal exercise (Adams, McHenry, & Bernaouer, 1977; Ekblom, 1969; Rowell, 1974; Saltin et al., 1968). It has been suggested that the increase in (a – v) O₂ difference at maximal exercise is mediated by changes in muscle blood flow), and/or adaptations in the muscle tissue itself. (Kraemer, & Daniels, 1986).

Changes in muscle BF are a potential mechanism resulting in a widening of the (a - v) O₂ difference at maximal exercise. It has been found that BF to active muscle during submaximal exercise is either unchanged or slightly reduced. (Brooks, & Fahey, 1984; Clausen, 1977). However, during maximal exercise, the BF to exercising muscle is increased in endurance-trained subjects (Brooks, & Fahey, 1984; Clausen, 1977; Kraemer, & Daniels, 1986). Thus, it has also been suggested that a change in the oxidative properties of the muscle tissue itself might explain the increase in (a –v) O₂ difference (Spina, 1999). These muscle adaptations include an increase in the size and number of mitochondria (Davies et al., 1981; Holloszy, & Booth, 1976), and a large increase in the activity of oxidative enzyme (Holloszy, 1973; Holloszy, & Coyle, 1984; Spina et al., 1996). This is consistent with observations of changes in muscle phenotype from Type IIb to type IIa characteristics (Spina et al., 1996), and with the vascular adaptations to endurance exercise training, such as proliferation of capillaries per squared millimeter (capillary density) (Anderson, & Henriksson, 1977; Apell, 1980), and the widening of the (a – v) O₂ difference (Spila, 1999).

Resting SBP and DBP in normotensive individuals are generally unaffected by endurance training, although mean pressure may decrease as a result of the acquired
resting bradycardia (MacDougall, 1994). Aerobic training attenuates the BP response during submaximal exercise so that at the same absolute power output, post-training systolic and mean pressures are lower than their pre-training values (Clausen, 1969; Choquette & Ferguson, 1973; Fagard, & Tipton, 1994; Hagberg, 1990; Hagberg et al., 1986; Schwartz, & Hirth, 1995; Seals & Hagberg, 1984; Surgeon General Report, 1996; Van Hoof et al., 1999). However, at the same relative exercise intensity and maximal exercise, BP responses remain unchanged but the responses occur at a higher power output (MacDougall, 1994). With hypertensive persons, several investigations (Clausen, 1969; Choquette & Ferguson, 1973; Hanson & Nedde, 1970), although no all (Gilder et al., 1989; Cleroux et al., 1987), also have noted significant decreases in resting SBP and DBP after a period of endurance training (For a review, see Hagberg, 1990; Tipton, 1984; MacDougall, 1994). Further, greater declines in BP have been reported in persons with high blood pressure. For example, after an aerobic exercise training program, it has been reported that resting BP (systolic/diastolic) decreases on average –3/-3 mmHg in person with normal BP (Van Hoof et al., 1999); in borderline hypertensive persons, the decrease is –6/-7 mmHg (Kukkonen et al., 1982); and in hypertensive persons, the decrease is –10/-8 mmHg (Fagard, & Tipton, 1994; Hagberg et al., 1983; Schwartz, & Hirth, 1995; Surgeon General Report, 1996).

Although the decline in BP is likely to be multifactorial, the most widely endorsed mechanism is the effect of aerobic exercise training on sympathetic nervous system (SNS) tone (Hagberg et al., 1989; Jennings et al., 1986; Schwartz, & Hirth, 1995). That is, a reduction in plasma norepinephrine level after training in subjects endurance trained (e.g., Hagberg et al., 1989; Jennings et al., 1986). However, other investigators have
suggested that the exercise associated-decline in BP is secondary to a reduction in weight, which, in various studies, has ranged from no change to a 3 kg weight lost (Schwartz et al., 1991). Thus, a reduction in epinephrine and norepinephrine (sympathetic drive), and/or changes in body weight and body fat, which might result in a decrease in total peripheral resistance, appear to be the mechanisms mediating the decline in arterial BP at the same absolute workload after moderate endurance exercise training (Clausen, 1977; Schwartz, & Hirth, 1995; Surgeon General Report, 1996).

Additionally, it should be mentioned that aerobic exercise training also results in some cellular and metabolic adaptations in the muscle tissue itself (as mentioned previously). This issue is beyond the scope of this review; however, the most relevant cellular and metabolic adaptations to aerobic exercise training are listed below, and the reader is referred to previous detailed reviews (Holloszy, 1973; Holloszy, & Booth, 1976; Howard, 1982). These cellular and metabolic adaptations include: an increase in the density of capillaries supplying muscle fibers (Appell, 1980), increased mitochondrial densities (Davies et al., 1981), increased mitochondrial enzyme activity (Brooks and Fahey, 1984; Davies et al. 1981; Holloszy, & Booth, 1976; Holloszy, 1973); changes in the substrates utilized for aerobic metabolism; increase in myoglobin content of muscle; increased activity of the electron-transport chain components; and some studies also indicate that training can result in changes in muscle fiber type distribution patterns (For a review, see Kraemer, & Daniel, 1986).

Lastly, it should be mentioned that there are some determining factors that need to be considered when evaluating the response of the individuals to a training program. That is, how the characteristics of the individuals (initial fitness level, gender, body composition,
health status, and age) and the training itself (mode of exercise, intensity, frequency, and duration of the training) affect the magnitude of this adaptation. For example, it has been documented that the lower the initial level of fitness is, the greater the percent increase in VO$_{2\text{max}}$ in the participants as a result of an aerobic exercise training (Saltin et al. 1968); women on average have lower values in VO$_{2\text{max}}$ than men do (Wilmore et al., 1977); and VO$_{2\text{max}}$ decline in with age (Åstrand et al. 1973).

The characteristics of the training itself (mode of exercise, exercise intensity, exercise duration, and exercise frequency) also affect the response in the individual to aerobic exercise training. For example, it has been observed greater improvements in VO$_{2\text{max}}$ as a result of very intense and strenuous training program (Hickson, Bomze, & Holloszy, 1977; Karvonen et al., 1957) than those observed after a moderate intensity-training program (Pollock, 1973; Daniel et al., 1979; Wilmore et al., 1970).

Similar results on aerobic fitness have been reported when manipulated the duration and frequency of exercise training. For example, Mileses et al. (1976) and Wilmore et al. (1970) found greater improvements in VO$_{2\text{max}}$ for participants in the longest-duration (45 minutes) training program as compared with the improvements attained for participants in the shortest-duration (15 minutes) training program. In other studies, Pollock et al. (1969) and Pollock (1978) comparing different frequencies of the training per week (2 versus 4; and 1 versus 3 versus 5, respectively) report a greater improvement in aerobic fitness (i.e., VO$_{2\text{max}}$) in the training programs with higher frequency per week. Pollock et al. found twice as much of an increase in VO$_{2\text{max}}$ (35 versus 17 percent) in the 4-days per week group, and Pollock an increased improvement in VO$_{2\text{max}}$ as training increased from 1 to 3 to 5 days per week. However, Pollock also noted a marked increase in the
incidence of injury as the frequency increased beyond 3 days per week and when the
duration per day increased above 30 minutes.

In summary, aerobic exercise training results in a number of CV adaptations that
improve not only maximal performance but also the ability to do prolonged exercise.
These CV adaptations include increases in maximal CO, SV, diastolic filling, and LV
volume overload hypertrophy (Astrand, 1960; Blomqvist, & Saltin, 1983; Clausen, 1976;
Levy et al., 1993; Longhurst et al., 1980; Saltin et al., 1968; Schaible, & Scheuer, 1985).
These adaptative responses, in concert with the adaptations in skeletal muscle (Anderson,
& Henriksson, 1977; Holloszy & Coyle, 1984), increase VO$_{2\text{max}}$ in young healthy
subjects in response to endurance exercise training (Spina, 1999). The increase in the
VO$_{2\text{max}}$ is associated mainly with an increase in the maximum CO, and a lesser extent, to
an increase in maximum (a – v) O$_2$ difference. Generally, at rest and submaximal
workloads, HR is decreased and SV is increased as compared with the untrained state. At
maximum exercise, HR$_{\text{max}}$ is unchanged or slight increased, while SV is increased.
Skeletal muscle adaptations include increase in muscle respiratory capacity, increase in
mitochondrial density and enzyme activity, and lower blood lactate levels at a given
workload. Resting arterial blood pressure, blood pressure at the same absolute workload,
and peak blood pressure is reduced after an aerobic exercise-training program in healthy
and diseased younger adults. The magnitude of CV adaptations depends on the
characteristics of the individuals and the exercise-training program (Kraemer, & Daniels,
1986; Spina 1999; Lakatta, 1993).

**Cardiovascular Adaptations to Aerobic Exercise Training in Older Adults**: Chronic
CV adaptations to aerobic exercise training in older adults have been studies using both
cross-sectional approaches with already highly trained older endurance athletes (primarily men) (Dehn, & Bruce, 1972; Dill et al., 1967; Hagberg, 1987; Heath et al., 1991), and longitudinal investigations assessing the effects of physical exercise training on previously sedentary older adults (Hagberg, 1994; Hagberg et al., 1989; Kohrt et al., 1991; Makrides et al., 1990; Seals, & Chase, 1989; Seals et al., 1984a).

According to evidence from some early longitudinal studies indicating a lack of increase in VO$_{2\text{max}}$ in older men and women as a result of endurance training (Adams, & DeVries, 1973; Benestad, 1965; DeVries, 1970; Niinimaa, & Shephard, 1978), it was thought that older adults (over 60 years of age) lost the ability to adapt to aerobic exercise training. However, those studies used very short and low-intensity aerobic exercise training, so that an insufficient training stimulus applied to the CV system is suggested as the reason why the exercise training did not induce any increase in VO$_{2\text{max}}$ (Hurley, Hagberg, 1998; Spina, 1999).

On the other hand, follow-up cross-sectional studies (those comparing young, middle-aged, and older individuals), and longitudinal studies (those performing exercise training programs in sedentary older adults) report usual adaptations (e.g., increases in VO$_{2\text{max}}$) to aerobic exercise training (Ehsani et al., 1991; Kohrt et al., 1991; Ogawa et al., 1992; Spina, 1999; Stratton et al., 1994). For example, cross-sectional studies show that master athletes certainly exhibit typical adaptations to aerobic exercise training (Fleg et al., 1988; Heath et al., 1981; Kavanagh, & Shephard, 1977; Pollock et al., 1987). Those typical adaptations in master athletes include: a lesser accumulation of subcutaneous fat; a better preservation of muscle mass, lean tissue, and increased peripheral O$_2$ utilization; a greater work performance at a given target HR; an aerobic capacity of nearly twice that...
of sedentary older individuals; and an equivalent VO$_2$ in highly trained middle-aged men (52-59 yr old) as compared to younger men (25-30 yr) (Wahren et al., 1974, for a review, see Kraemer, & Daniel, 1986; Lakatta, 1993; Spina, 1999). Therefore, cross-sectional studies with older master athletes and sedentary control have found that estimated maximum CO (assuming that the SV measured at submaximum exercise is the same as that at maximum exercise) during treadmill exercise is increased in master athletes versus sedentary age-matched controls (Hagberg et al., 1985); estimated maximum SV during exercise (i.e. the O$_2$ pulse, VO$_2$/HR) in master athletes is increased compared with their sedentary counterparts (Heath et al., 1981); and HR$_{max}$ during cycle or treadmill exercise does not differ between the master athletes and sedentary older individuals (Fleg et al., 1988; Heath et al., 1981; for a review, see Lakatta, 1993).

In fact, later published data (e.g., from longitudinal studies) in older sedentary men and women (60-80 yr of age) performing aerobic exercise training have demonstrated that older adults maintain the ability to adapt to prolonged and relative intense aerobic exercise training, as evidenced by an increase in the O$_2$ transport capacity of the CV system (Beere et al., 1999; Ehsani et al., 1991; Hagberg et al., 1989a, 1989b; Kohrt et al., 1991; Meredith et al., 1989; Ogawa et al., 1992; Stratton et al., 1994). These studies have reported in response to aerobic exercise training an enhanced VO$_{2max}$ in old men and women that has been similar in relative magnitude to that observed in young people (20-30%) (Devries, 1970; Ehsani, 1987; Haber, Honiger, Klicpera, & Niederberg, 1984; Hagberg et al., 1989a, 1989b; Kavanagh, & Shephard, 1977; Raven, & Mitchell, 1980; Seals et al., 1984; Spina et al., 1993; Spina, 1999; Lakatta, 1993). For example, Seals et al reported a 12% increase in VO$_{2max}$ in healthy (60-69 years of age) men and women
after 6 months of training at 50% of VO\textsubscript{2max}, and a further increase of 18% after a subsequent 6 months of training at 75-85% of VO\textsubscript{2max}, totaling to a 30% increase in VO\textsubscript{2max} after a year of training (Seals et al., 1984). Hagberg et al reported a 28% increase in VO\textsubscript{2max} in 60-69 years of age hypertensive men and women with 9 months of training at 70-75% of VO\textsubscript{2max} (Hagberg et al., 1989b). Hagberg et al also found a 22% increase in VO\textsubscript{2max} in healthy 70-79 years of age men and women with 6 months of training at 70-75% of VO\textsubscript{2max} (Hagberg et al., 1989a). Hence, prolonged and relatively intense (> 75% of VO\textsubscript{2max}) aerobic exercise training results in substantial increases in VO\textsubscript{2max} in older men and women at a similar degree (Kohrt et al., 1991).

Central and peripheral CV adaptations mediating the enhanced aerobic capacity in older men and women have been reported as a result of aerobic exercise training (Beere et al., 1999; Ehasani et al., 1991; Ogawa et al., 1992; Schocken et al., 1983; Seals et al., 1984; Spina, 1993). However, the results in these studies (e.g., Beere et al., 1999; Seal et al., 1984; Spina et al., 1993) have report differences in the relative magnitude the change in each of these mechanisms, which appears to be primarily due to gender-related differences in the mechanisms of adaptations to aerobic exercise training. For example, after a low- (6 months) and higher-intensity (6 months) training program with older (60-69 yr of age) men (n=8) and women (n=3) for 12 months, Seals et al (1984) found that an increase in VO\textsubscript{2max} by ~ 30% (25.4 to 32.9 mL/kg/min) was achieved primarily by an increase in estimated maximal (a – v) O\textsubscript{2} difference and estimated maximal SV, with little increase in estimated maximum CO during treadmill exercise. However, Seals et al did not perform comparison between men and women, so that it is not possible make conclusion about this issue. Therefore, given that the larger number of old men
participating in Seals et al study, the results reported appear to agree with findings in subsequent studies examining gender-related differences in CV adaptations to aerobic exercise training. Therefore, after a 3-month period of exercise training with cycle ergometry in older and younger men, Beere et al (1999) found that with training the older and younger groups increased VO$_2$ by 17.8% and 20.2%, respectively, but peak CO did not change in both groups, and systemic (a – v) O$_2$ difference increased 14.4% in the older group and 14.3% in the younger group and accounted for changes in peak oxygen consumption. In this last study, SV at maximal exercise is not reported, so that if there was a decline in HR with exercise training, an increase in maximal SV should have happened to maintain peak CO unchanged.

In contrast, in a study designed to examine gender-related differences in the CV adaptations to aerobic exercise training in older adults, Spina et al found that after a high-intensity exercise training for 9-12 months in older (60-69 yr of age) men (n=15) and women (n=16) the training-induce increase in VO$_{2\text{max}}$ was 19% and 22% in the men and women, respectively. SV at peak treadmill exercise in older (64 yr) men increases by 15%, which was accompanied by a 7% increase in arteriovenous O$_2$ difference. Thus, two-third of the increase in VO$_{2\text{max}}$ was accounted for by an increase in maximal CO, and one-third was accounted for by a wider (a – v) O$_2$ difference. On the other hand, the increase in VO$_{2\text{max}}$ in women in response to training was solely the result of an enhanced (a – v) O$_2$ difference at maximal exercise, with no evidence of central adaptations.

Given the gender-related differences in CV adaptations to aerobic exercise training in older adults, follow-up studies tried to determine the mechanisms mediating the specific central and peripheral changes by sex with aerobic exercise training in older adults (e.g.,
Ehsani et al., 1991; Spina et al., 1993). Hence, studies examining this issue documented that LV systolic function improves with aerobic exercise training in older men. For example, in a study in which 10 sedentary men (64 yrs of age) underwent an aerobic exercise training program for 12 months, that induced a 23% increases in VO₂max, Ehsani et al (1991) found that before training LV ejection fraction response during supine cycle ergometer exercise increased modestly (4.3%) from rest to peak exercise. However, after training, the increase in ejection fraction during exercise was greater (10.7%), and similar to the increase observed in young sedentary men. There was a greater decrease in ESV after training, as compared to before, despite a similar increase in SBP with a leftward shift in the ESV/SBP relationship, indicative of enhanced inotropic state. In addition, the exercise training induced proportional increases in LV EDV and posterior wall thickness, measured by echocardiography, with no change in wall thickness to radius ratio, suggestive of LV volume overload or eccentric hypertrophy (Spina, 1999).

Similar results have been found in other studies. For example, Stratton et al (1994) found an increase in ejection fraction and CO at peak cycle ergometer exercise after 6 months of endurance exercise training program in older men. Makriedes et al (1990) found that peak CO during upright cycle-ergometer-exercise, measure with the CO₂ rebreathing procedure is augmented in response to training in men between the ages of 60-70. Schulman et al (1996) studied sedentary older men before and after training and older endurance athletes prior to and after cessation of training. Endurance training induced an increase in VO₂max and the reserve of cardiac and SV indexes during peak exercise. In the endurance trained men, VO₂max, peak EDV index, SV index, and cardiac index were decreased in response to cessation of training (Lakatta, 1993; Spina, 1999).
On the other hand, other studies have been led to determine CV adaptations to aerobic exercise training in older women (Spina et al., 1993). For example, in a study examining LV systolic performance by electocardiographic-gated blood pool imaging during supine exercise in 10 women (63 yr of age), before and after the same training program as the men, Spina et al found that VO$_{2\text{max}}$ was increased by 21% in response to training. However, LV ejection fraction at rest and during peak exercise did not change in response to training, and the changes in ejection fraction from rest to peak exercise were similar before and after training. Stroke volume and CO at peak exercise also did not change in response to training. Basal EDV was unchanged as a result of training, indicative of lack of cardiac enlargement. Thus, the results suggest that older women undergo similar increases in VO$_{2\text{max}}$ in response to training as men without changes in LV systolic performance and eccentric hypertrophy (Spina et al., 1993; Spina 1999).

In addition to previous findings, Coggan et al (1992) demonstrated that the percent increase in skeletal muscle capillarization induced by training was the same as the percent increase in VO$_{2\text{max}}$ in older women. Therefore, the women in this study (Coggan et al., 1992) demonstrated also an increase in the activity of mitochondrial marker enzymes. These two findings suggest that the increase in VO$_{2\text{max}}$ in response to aerobic exercise training in older women is due to peripheral adaptations that lead to enhanced (a –v) O$_2$ content difference at maximal exercise (Coggan et al., 1992; Spina, 1993). It was concluded that the mechanisms responsible for the training-induced increase in VO$_{2\text{max}}$ in older men were similar to those found in young people (central and peripheral CV mechanisms); while, the older women adapted to the exercise training with an
increase in O\textsubscript{2} extraction with no increase in cardiac output (peripheral CV mechanism) (For a review see, Spina et al., 1999).

Other gender-related CV adaptation to aerobic exercise training observed in older men has been an improvement in peak early filling during exercise in response to 6 months of endurance exercise training (Levy et al., 1993). Similar result was observed in a study examining the adaptive response of LV filling dynamics to training among men and women (Spina et al., 1996). In this study older men (65 yr) and women (64 yr) were assessed before and after 9 months of aerobic exercise training using radionuclide ventriculography imagine at rest and during supine exercise. It was found that LV filling dynamics was influenced by gender. That is, the older men showed the adaptive improvement in LV filling, whereas the older women did not (Spina et al., 1996). It should be noted that a reduced ability to fill the ventricle adequately during exercise could also contribute to the lower CO and VO\textsubscript{2max}. In contrast to this finding, some cross-sectional studies (e.g., Jungblut et al., 2000) have found no improvements in LV diastolic filling characteristics using M-mode and Doppler echocardiographic data obtained at rest from older highly trained endurance athletes (65-75 yr of age) and sedentary control group subjects (65-73 yr of age) with no cardiovascular disease. However, this data were collected after exercise, and it is known that hemodynamic response after exercise might not represent the CV response during maximal exercise (MacDougall et al., 1992; McCartney et al., 1993; Wieck et al., 1990). Therefore, longitudinal studies e.g., (Levy et al., 1993) have demonstrated improvements in LV diastolic filling dynamic at rest.

In summary, in older men the training-induced increase in VO\textsubscript{2max} is attained by an increase in both maximum CO (Makriedes et al., 1990; Stratton et al., 1994) and (a – v)
O$_2$ difference (Beere et al., 1999; Ehsani et al., 1991). This increase in CO following is achieved by an increase in SV, due to an increase in EDV. After training, a greater reduction in ESV during exercise augments the ejection fraction achieved during exercise. Because arterial pressure during the supine exercise testing is not affected by conditioning, the enhanced ejection fraction and reduced ESV after conditioning have been interpreted to reflect an increase in myocardial contractility induced conditioning (Ehsani et al., 1991). Therefore, increases in (a –v) O$_2$ difference have been observed following exercise training (Seals et al., 1984). In contrast, the training effect to increase VO$_{2\text{max}}$ in women appears to be achieved by an increase in exercise arteriovenous O$_2$ difference (peripheral mechanism), since neither peak SV nor maximum HR increased (Seals et al., 1984; Spina et al., 1993).

Other CV adaptations as a result of aerobic exercise training in older adults include changes en blood volume and blood flow, and blood pressure. For example, it has documented that physical training (1) increase total blood volume (Jones et al., 1999), (2) reduce vascular resistance (Hagberg et al., 1985; Jensen-Urstand et al., 1999; Martin et al., 1990), (3) increase the ability to redirect blood to working muscles and the muscle’s ability to extract oxygen (aerobic metabolism) from the blood (Larson & Bruce (1987); and (4) increase peripheral blood flow (Beere et al., 1999; Hagberg et al., 1985; Martin et al., 1990). It has been suggested that these changes increase the preloading of the heart and amplifies stroke volume (Hagberg et al., 1985; Jones et al.; 1999; Weisfeldt, Gerstenblith, & Lakatta, 1985). Thus, SV is increased in exercisers and CO is maintained even though their HR$_{\text{max}}$ decreases as they age (Landin et al., 1985; Mahler, Cunnigham, & Curfman, 1986; Zauner, 1985; Spirduso, 1996).
The effects of normal physical activity (Reaven et al., 1991) and aerobic exercise training (Brauth et al., 1994; Seals et al. (1999; Van Hoof et al., 1989) on resting BP, casual and ambulatory BP, and BP during submaximal and maximal exercise have been studied in normotensive and hypertensive older men and women (Brauth et al., 1994; Seals et al., 1999; Van Hoof et al., 1989). Studies examining the former issue (effects of normal physical activity on BP) have been done to determine differences in BP between physically active and no physically active men and women. For example, epidemiological studies showed either somewhat lower BP or no difference in BP in active compared with sedentary individuals (For a review, see ACSM, 1993; Montoye et al., 1972). Similar results have been evident in studies following-up University of Pennsylvania alumni who played intramural sports less or more that 5 hours per week, or athletes and nonathletes. Therefore, Reaven et al found in one study with 641 Caucasian women between the ages of 50 and 89 yr, who were divided into physical activity categories of light (58%), moderate (24%), heavy (6%), or no activity (12%) that as the activity intensity increased, systolic blood pressure decreased. The SBP was approximately 20 mmHg lower in the heavy-activity group than in the no-activity group. Lower rates of hypertension were also associated with higher physical activity. (ACSM, 1993; Reaven et al., 1991).

Similar findings have been also reported in studies assessing the BP lowering effects of endurance exercise training on normotensive and hypertensive older adults (Brauth et al., 1994; Seals et al., 1999; Van Hoof et al., 1989). For example, in a study assessing the effect of endurance exercise training on BP at rest, during exercise and during 24-hours in sedentary men, Van Hoof et al found that during exercise SBP was lower after training
when measured at the same submaximal workload. However, when workload was expressed as a percentage of peak VO₂, SBP was not different before and after training (Van Hoof et al., 1989). Brauth et al in a study examining the effects of exercise intensity on resting BP in normotensive older adults (60-79 yr of age) during 6 months of exercise training found that walking exercise lowers resting BP in normotensive older adults, and produces a moderate physical conditioning benefit similar to that observed in younger persons (Brauth et al., 1994). Seals et al comparing four groups (pre- and post-menopausal sedentary, and pre- and post-menopausal endurance-exercise trained women) of healthy normotensive women found that 24-h SBP and pulse pressure were not different in the endurance-trained group with age. In the sedentary group, both 24-h SBP and pulse pressure were higher in (~10mmHg) in postmenopausal women than in premenopausal women (Seals et al., 1999).

Similar lower BP measurements induced for exercise training have been found in studies assessing the effect of physical training on BP in hypertensive persons (Hagberg et al., 1989; Ishikawas, 1999). For example, Ishikawa et al comparing sedentary hypertensive subjects (who performed exercise training at mild intensity for 8-wks with a combination of various exercises) to other hypertensive control subjects found that BP was reduced in all exercise subgroups classified by age and gender. Therefore, no changes were observed in any of these variables in the control group subjects (Ishikawas, 1999). In other study, Hagberg et al examined the effects of 9 months of low- or moderate-intensity exercise training in hypertensive men and women (mean age 64 ± 3 yr) found that DBP decreased 11 to 12 mmHg in both group. SBP decreased 20 mmHg in the low-intensity group with training (Hagberg et al., 1989a).
The mechanisms by which exercise training lowers BP are unclear. However, potential mechanisms include: decrease in plasma norepinephrine levels, increase in circulating vasodilators substances, amelioration of hyperinsulemia, and alteration in renal function (ACSM, 1997).

Lastly, it should be noted that the absolute and relative magnitude of the VO$_{2\text{max}}$ augmentation and the underlying central and peripheral mechanisms vary with relative fitness before training, with intensity, duration and frequency of aerobic exercise training (Ehsani, 1987; Kavanagh, & Shephard, 1977; Raven, & Mitchell, 1980), and with the experimental paradigm in which performance was measured (Raven, & Mitchell, 1980). It is clear that the reduction in maximum HR in older individuals is not affected by physical conditioning, regardless of duration or intensity.

2.3.2 Chronic Cardiovascular Adaptations to Anaerobic Exercise Training


Chronic Cardiovascular Adaptations to RE Training in Younger Adults: In healthy younger adults, cross-sectional (Brown et al., 1983; Fleck et al., 1987; Longhust et al., 1980; Pearson et al., 1986), and longitudinal (Kanakis, & Hickson, 1980; Lusiani et al., 1986; Ricci et al., 1982) studies have documented structural and functional changes in
the CV (Kanakis, & Hickson, 1980; Lusiani et al., 1986; McCartney et al., 1993; Ricci et al., 1982) and musculoskeletal (e.g., muscle fibers, connective tissue, and bone modeling) systems. In addition, neural adaptations, body composition changes, and changes in endocrine functions have also been reported.

The CV adaptations to RE training include changes in morphology, functional adaptations (systolic and diastolic cardiac function), HR and BP and acute responses to resistance exercise (Effron, 1989; Fleck, 1988).

**Morphological adaptations:** Studies determining morphological heart adaptations to RE have examined changes in LV wall thickness (Brown et al., 1983; Pearson et al., 1986), LV dimension and volume (Kraemer, Deschenes, & Fleck, 1988; Longhurst et al., 1980), and LV mass (Brown et al., 1983; Pearson et al., 1986) in different caliber weight-trained athletes as compared to control subject, or in short-term longitudinal study participants. The major exercise-induced change in cardiac dimension is an absolute increase in *left ventricular wall* or cardiac hypertrophy (Brown et al., 1983; Fleck et al., 1987; Snoeckx et al., 1982; for a review see Effron, 1989; Fleck, 1988). Cardiac hypertrophy have been reported in cross-sectional (Brown et al., 1983; Fleck et al., 1987; Longhurst et al., 1980; Pearson et al., 1986), and short-term (10-20 wk) longitudinal studies (Kanakis, & Hickson, 1980; Lusiani et al., 1986; Ricci et al., 1982) showing increases in echocardiographic imagine of diastolic posterior LV wall thickness (Brown et al., 1983; Pearson et al., 1986), and diastolic intraventricular septum (Brown et al., 1983). It has been suggested that that the amount of thickness depends upon the training intensity (Efron, 1989; Fleck, 1988). For example, national and international weight lifters have a greater degree of LV wall hypertrophy and LV mass as compared with
amateur and recreational weight lifters (Brown et al., 1983; Fleck et al., 1987; Snoeckx et al., 1982; for a review see Effron, 1989; Fleck, 1988).

It should be noted, however, that even though the absolute increase of LV wall thickness has been suggested as an evident CV adaptation to RE in resistance trained athletes as compared to control subjects, this difference is greatly reduced or nonexistent if examined relative to body surface area (BSA) and lean body mass (LBM) (Fleck, 1988). Therefore, the caliber of weight-trained athlete appears to have little effect upon LV wall thickness.

Other cardiac indexes used to determine the morphological heart changes with RE have been *LV dimension and volume*, which have been determined for LV internal dimension during systole (LVIDs) and diastole (LVIDd). Most of the cross-sectional studies indicate that LV internal dimension and volume during systole and diastole have not increased in resistance-trained athletes as compared to controls (Kraemer, Deschenes, & Fleck, 1988; Longhurst et al., 1980). A few cross-sectional studies have reported increased absolute LVIDs (Menapace et al., 1982) and LVIDd (Colan et al., 1985; Menapace et al., 1982; Pearson et al., 1986) in resistance-trained athletes versus control subjects. However, if corrected for BSA and LBM, any cross-sectional studies have demonstrated increased LVIDs or LVIDd. Thus, this appears to indicate that RE training does not induce cardiac changes indicative of a volume overload on the heart (Fleck, 1988). Similar findings have reported short-term longitudinal studies with young adult men, which indicate no increases in systolic LV internal dimension (Kanakis, & Hickson, 1980) and diastolic LV internal dimension (Kanakis, & Hickson, 1980; Lusiani et al., 1986; Ricci et al., 1982; for a review, see Effron, 1989; Fleck, 1988).
Another cardiac index thought to determine morphological change is left ventricular mass. Most of cross-sectional studies comparing resistance-trained individuals to control subjects report (absolute and relative to body surface area) increase in LV mass (Brown et al., 1983; Longhurst et al., 1980; Pearson et al., 1986). However, relative to LBM, a few studies have demonstrated that weight lifters have greater LV mass than controls subjects (e.g., Longhurst et al., 1980; Pearson et al., 1986). For example, Longhurst et al found an increased LV mass relative to lean body mass in national caliber athletes as matched for age, height, and weight (Longhurst et al., 1980). Short-term longitudinal studies report absolute (Kanakis, & Hickson, 1980; Ricci et al., 1982) and relative to BSA and LBM (Lusiani et al., 1986) increases in LV mass with RE training, supporting the results based on cross-sectional studies.

LV mass increases have been attributed to an augmented left ventricular afterload with resistance exercise (Fleck, 1988). It has been considered that static exercise increases ventricular afterload through elevated SBP. Elevated BP increases LV mass (Effron, 1989; Fleck, 1988) and wall thickness in patients with hypertension. However, the results are not consistent in weight lifters as in patients with chronic hypertension or aortic stenosis may be due to the difference in which CV responses dynamic resistance exercise; that is intermittent nature of the hemodynamic changes (Effron, 1989; Fleck, 1988)

**Functional adaptations:** Studies determining CV functional adaptations to RE training have examined changes in stroke volume (SV), and systolic and diastolic function in weight-trained athletes as compared to control subjects, or short-term longitudinal participants. Studies examining stroke volume have reported that RE has no
substantial effect on stroke volume. For example, Pearson et al showed an increase in absolute SV in weight lifters as compared with control but no change was observed if SV was normalized to BSA or LBM. Brown et al found no difference in SV in recreational or competitive weight lifters (Brown et al., 1987). Fleck reported in a meta-analysis study on 5 studies reporting SV measurements that an increase in absolute SV in competitive weight lifters but not in recreational weight lifters. When normalized for BSA or LBM, no weight lifter of any type showed any difference in SV index as compared with untrained controls (For a review, see Effron, 1989).

Most of the cross-sectional studies assessing systolic function with echocardiographic examination indicate that in RE trained athletes are not different from sedentary control at rest. Percent shortening fraction (Dickhuth et al., 1979; Menapace et al., 1982; Pearson et al., 1986; Salke, Rowland, & Burke, 1985), ejection fraction (Longhurst et al., 1980b), and velocity of circumferential shortening (Dickhuth et al., 1979; Longhurst et al., 1980b) are not different at rest in resistance trained athletes as compared to are not different from sedentary control at rest. Only one study (Colan et al., 1985) has reported percent fractional shortening in resistance-trained athletes to be greater than control group. Therefore, short-term longitudinal studies report equivocal results. While Lusiani et al reported no change in percent fractional shortening (Lusiani et al., 1986), Kanakis, & Hickson reported an increase in the same index (Kanakis, & Hickson, 1980). However, in summary, it is suggested that cross-sectional and short-term indicate that RE training appears not to alter LV systolic function. On the other hand, LV function did not deteriorate after RE training in spite of the LV mass increase (For a review, see Effron 1989; Fleck, 1988).
An additional parameter assessed to examine functional adaptations to RE has been *diastolic function*. Cross-sectional studies have found an enhanced diastolic function in weight-trained athletes as compared to sedentary control subjects (Colan et al., 1985; Pearson et al., 1986). For example, Colan et al looked at echocardiographic indexes of LV diastolic function in weight lifters, the peak rate of wall thinning and rate of diastolic dimensional change was increased despite of greater absolute and normalized to body surface area LV mass (Colan et al., 1985). Therefore, other diastolic function index assessed (peak rate of chamber enlargement normalized for end-diastolic dimension, peak rate of wall thinning normalized for percent of thinning, and time from aortic valve closure to peak rate of chamber enlargement and to peak rate of wall thinning) did not differ between the athletes and control group (Colan et al., 1985).

Using Doppler flow studies to evaluate diastolic function in weight lifters, Pearson et al found no change in either peak E/A ratio or the E/A integral. However, there was a small but significant increase in the atrial flow velocity (A) (Pearson et al., 1986). Thus, it is suggested that RE does not have effect upon diastolic function (Colan et al., 1985; Pearson et al., 1986). Increases in absolute diastolic indexes may occur, but when the effects of ventricular size and systolic function are considered, diastolic function is normal (Fleck, 1988).

Other CV adaptations to RE training that have been assessed are resting *HR and BP*. Resting HR (Colan et al., 1985; Fleck, & Dean, 1987) in cross-sectional studies comparing highly trained weight lifters and sedentary subjects (Colan et al., 1985; Fleck, & Dean, 1987; Longhurst et al., 1980a, 1980b; Menapace et al., 1982; Pearson et al., 1986) report no difference between these two groups in this parameter. Some short-term
longitudinal studies involving young men report a decrease in resting HR (e.g., Baechle, 1976; Kanakis et al., 1980; Stone et al., 1983a), while other studies only show a trend toward such an effect (Lusiani et al., 1986; Ricci et al., 1982; Seals et al., 1994; Stone et al., 1983b). The possible mechanisms mediating a training-induced bradycardia might be related to an increased parasympathetic drive or decreased sympathetic influence on the heart (Frick, Elovainio, & Somer, 1967).

In regard to resting BP, the results are not completely conclusive. The majority of cross-sectional studies examining in highly trained weight lifters show no difference in resting BP from sedentary control subjects (Fleck, & Dean, 1987; Longhurst et al., 1980a, 1980b; Menapace et al., 1982; Pearson et al., 1986). However, increased (Snoecky et al., 1982) and decreased (Smith, & Raven, 1986) resting SBPs have been also reported in weight lifters. Short-term studies involving young males report no change in resting SBP and DBP (Baechle, 1976; Lusiane et al., 1986) and a decrease in resting SBP, but no change in resting DBP (Stone et al., 1983). It appears to be that majority of cross-sectional and longitudinal studies indicate that RE training has not effect on resting BP (Fleck, 1988).

Additionally, changes in acute CV response to resistance exercise have been documented. The established pressor response (HR and BP) to dynamic RE (Lentini et al., 1993, Lewis et al., 1985; MacDougall et al., 1985, 1992) has been reported to be lower in trained bodybuilders than in novice resistance-trained and non-resistance exercise trained individuals (Fleck, & Dean, 1987) when performing RE at 60, 70, 80 and 90% of 1-RM, and at one repetition maximum (Fleck, 1988; Fleck, & Dean, 1987). However, the evidence that highly trained weight lifters have a blunted circulatory
response to RE compared with nontrained control is not at all conclusive (McCartney, 1999; Fleck & Dean, 1987), and more research in this area has been suggested (McCartney, 1999).

Short-term longitudinal studies comparing RE trained and control groups have found that for a given absolute workload, all responses (e.g., HR, BP) were markedly reduced after training in the trained group (Seal et al., 1994). In this study, after 19-weeks of RE training, there were reductions in SBP, DBP, and RPP of 17-27% during 10-20 repetitions lifting the same absolute weight as before training (Sale et al., 1994). This training adaptation has been reported whether HR and BP responses are expressed in terms of peak values or in magnitude of response (e.g., peak value minus resting values).

Lastly, it should be noted that RE training programs do not appear to be effective in increasing VO\textsubscript{2max}. Exceptions would be relatively untrained people, in whom increases ranging from 5% to 8% in aerobic power result from resistance training (Gettman et al. 1978; Fleck, 1988; Stone et al. 1983). Even increases in VO\textsubscript{2max} from circuit weight training programs (light loads, short rests, and multiple exercise) are lower than what would be expected from conventional endurance training programs (Baechle, 1994).

**Musculoskeletal adaptations:** The classical response observed in most studies examining RE training is an increase in muscular strength (Atha, 1981; Clarke, 1973; Delorme, 1946, 1945; Fleck, & Schutt, 1983; Gettman et al. 1978; McCartney et al., 1991; McDonagh, & Davies, 1984; Sale et al., 1994; Stone et al. 1983; for a review, see Kraemer, & Daniels, 1986). This increase in muscle strength has been suggested to be mediated by an increase in the cross-sectional area of muscle or muscle size (Brooks, Fahey, & White, 1995; Gollnick, 1983; Häkkinen et al., 1988; Ikai, & Fukunaga, 1970;
<table>
<thead>
<tr>
<th>Variable</th>
<th>Result following resistance training</th>
<th>Result following endurance training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Performance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Muscle strength</td>
<td>↑</td>
<td>=</td>
</tr>
<tr>
<td>Muscle endurance</td>
<td>↑ high power output</td>
<td>↑ low power output</td>
</tr>
<tr>
<td>Aerobic power</td>
<td>= / ↑ slightly</td>
<td>↑</td>
</tr>
<tr>
<td>Max. rate of force production</td>
<td>↑</td>
<td>= / ↓</td>
</tr>
<tr>
<td>Vertical jump</td>
<td>Ability ↑</td>
<td>Ability =</td>
</tr>
<tr>
<td>Anaerobic power</td>
<td>↑</td>
<td>=</td>
</tr>
<tr>
<td>Muscle fibers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fiber size</td>
<td>↑</td>
<td>= / ↑ slightly</td>
</tr>
<tr>
<td>Capillary density</td>
<td>= / ↓</td>
<td>↑</td>
</tr>
<tr>
<td>Motochondrial density</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Fast Heavy-chain myosin</td>
<td>↑ in amount</td>
<td>= / ↓ in amount</td>
</tr>
<tr>
<td>Enzymes activity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Creatine phosphokinase</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Myokinase</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Phosphofructokinase</td>
<td>↑</td>
<td>#</td>
</tr>
<tr>
<td>Lactate dehydronase</td>
<td>= / #</td>
<td>#</td>
</tr>
<tr>
<td>Metabolic energy stores</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stored ATP</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Stored creatine phosphate</td>
<td>↑</td>
<td>↑</td>
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<tr>
<td>Stored glycogen</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Stored triglycerides</td>
<td>May ↑</td>
<td>↑</td>
</tr>
<tr>
<td>Connective tissue</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ligament strength</td>
<td>May ↑</td>
<td>↑</td>
</tr>
<tr>
<td>Tendon strength</td>
<td>May ↑</td>
<td>↑</td>
</tr>
<tr>
<td>Collagen content</td>
<td>May ↑</td>
<td>#</td>
</tr>
<tr>
<td>Bone density</td>
<td>= / ↑</td>
<td>↑</td>
</tr>
<tr>
<td>Body composition</td>
<td></td>
<td></td>
</tr>
<tr>
<td>%body fat</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Fat-free mass</td>
<td>↑</td>
<td>=</td>
</tr>
</tbody>
</table>

(Adapted from Baechle, 1994).
MacDougall, et al., 1977; McDonagh, & Davies, 1984; McDonagh, Hayward, & Davies, 1983; McArdle, Katch, & Katch, 1991), which occurs primarily through an increase in cross-sectional area of both type I and type II fibers (Brooks, Fahey, & White, 1995; MacDougall, 1986; MacDougall et al., 1980, 1979); and an increase in the size and number of actin and myosin filaments, and the addition of sarcomeres to existing muscle fibers, which mediate the increase in muscle fiber size (Goldberg et al., 1975; Gollnick et al., 1981; MacDougall et al., 1980; Martin et al., 1981; Dons et al., 1979).

Other musculoskeletal adaptations (for more information, see table 3) have been a decrease in mitochondrial volume density and capillary density as a result of a dilution of these elements in the increased cell volume with training (Luthi et al., 1986; MacDougall et al., 1975, 1979); and increases in energy substrate levels and their availability in muscle (ATP, creatine phosphate, creatine, and glycogen increased in the triceps brachii), and the capacity for intramuscular lipid storage. (Tesch, 1987; Tesch, Komi, & Hakkinen, 1987; Baechle, 1994; Kraemer, & Daniels, 1986). In addition, though not entirely conclusive, some evidence suggests a greater absolute amount of connective tissue in the muscle has been found in trained bodybuilders, but the evidence is not completely conclusive (Conroy et al., 1992; Stone, 1988). Lastly, an elevated level of bone mineral in both axial and appendicular skeletons has been found in elite strength athletes involved in competitive forms of resistance exercise (Colleti et al., 1989; Conroy et al., 1992; Granhed, Johnson, & Hansson, 1987; Nilsson, & Westlin, 1971; For a review, Baechle, 1994; Kraemer, & Daniels, 1986).
CHAPTER III. STRENGTH TRAINING AND THE CARDIOVASCULAR SYSTEM IN THE ELDERLY

Physical training interventions designed to optimize health and reduce health care costs in older men and women should address cardiovascular function as CVD collectively represent the major cause of death and disability in older adults (Brock, Guralnick, & Brody, 1990). In addition, physical training interventions frequently address declines in the musculoskeletal function that occur with aging, and that are associated with deterioration in physical functional capacity (Lindle et al., 1997; Lynch et al., 1999) and increased risk of falls (Campbell, Borrie, & Spears, 1989) and hip fracture (Aniansson et al., 1984; Hurley, & Hagberg, 1998; Pollock, & Evans, 1999; Pollock, & Vincent, 1996).

Resistance exercise training is well established as an effective method for developing muscular strength and endurance, and is prescribed for fitness and the prevention and rehabilitation of orthopedic injuries (AACVPR, 1991; ACSM, 1998, 2000; Atha, 1981; Feigenbaum, & Pollock, 1999; Fleck, & Kraemer, 1997; Hurley, & Roth, 2000). Furthermore, RE training, more recently, has been also considered as a modality that might be used for health purposes (Huley, & Roth, 2000; Pollock et al., 2000; Surgeon General Report, 1996).

The results in studies including RE training in older adults fitness and health programs have demonstrated positive effects preserving and enhancing muscular strength and endurance (muscular fitness) (Fiatarone et al., 1994; Reyes et al., 2000; Taunton et al., 1997), which, in turn, might help to prevent falls, improve mobility, and counteract muscle weakness and frailty (Fiatarone et al., 1994; Taunton et al., 1997). Consequently, muscular fitness in older men and women might allow the performance of activities of daily living with less effort, extend functional independence during the latter years.
(ACSM, 2000), thereby allowing the older adult to avoid a primary risk factor of CV disease, a physical inactive or sedentary life.

While one can argue that the benefits of RE extend to lower risk of CV diseases, it is also of interest to examine the extent to which RE improves CV function in older adults. Thus, the following discussion identifies the benefits of RE with a particular emphasis on CV function.

3.1. List of Benefits of Strength Training

Resistance exercise training above all induces structural and functional adaptations in the musculoskeletal system (e.g., muscle fibers, connective tissue, and bone modeling). These musculoskeletal adaptations include: (1) **an increase in muscular strength**, classical response observed in most studies examining RE training (Atha, 1981; Clarke, 1973; Delorme, 1946, 1945; Fleck, & Schutt, 1983; Gettman et al. 1978; McCartney et al., 1991; McDonagh, & Davies, 1984; Stone et al. 1983; for a review, see Kraemer, & Daniels, 1986); (2) **an increase in the cross-sectional area of muscle or muscle size** (Brooks, Fahey, & White, 1995; Gollnick, 1983; Häkkinen et al., 1988; Ikai, & Fukunaga, 1970; MacDougall, et al., 1977; McDonagh, & Davies, 1984; McDonagh, Hayward, & Davies, 1983; McArdle, Katch, & Katch, 1991), which occurs primarily through an increase in cross-sectional area of both type I and type II fibers (Brooks, Fahey, & White, 1995; MacDougall, 1986; MacDougall et al., 1980, 1979); (3) **an increase in the size and number of actin and myosin filaments, and the addition of sarcomeres** to existing muscle fibers, which mediate the increase in muscle fiber size (Goldberg et al., 1975; Gollnick et al., 1981; MacDougall et al., 1980; Martin et al., 1981; Dons et al., 1979); (4) **a decrease in mitochondrial volume density and capillary**
density as a result of a dilution of these elements in the increased cell volume with training (Luthi et al., 1986; MacDougall et al., 1975, 1979); and (5) increases in energy substrate levels and their availability in muscle (ATP, creatine phosphate, creatine, and glycogen increased in the triceps brachii), and the capacity for intramuscular lipid storage. (Tesch, 1987; Tesch, Komi, & Hakkinen, 1987; Baechle, 1994; Kraemer, & Daniels, 1986); (6) a greater absolute amount of connective tissue in the muscle has been found in trained bodybuilders, but the evidence is not completely conclusive (Conroy et al., 1992; Stone, 1988); and (7) an elevated level of bone mineral in both axial and appendicular skeletons has been found in elite strength athletes involved in competitive forms of resistance exercise (Colleti et al., 1989; Conroy et al., 1992; Granhed, Johnson, & Hansson, 1987; Nilsson, & Westlin, 1971; For a review, Baechle, 1994; Kraemer, & Daniels, 1986).

Resistance training also induces neural adaptations, body composition changes, hormonal influence, and CV adaptations. Neural adaptations have been suggested as the primary mechanism (optimization of recruitment patterns) underlying the initial muscle strength gains taken place in the absence of tissue hypertrophy during the first 2 or 4 weeks of a training program (Digby Sale, 1988; Moritani, & DeVries, 1979). Further, a person becomes more adept in effectively using the muscle mass, independent of any improvement that occur with training in muscle cross-sectional area with training. (Edgerton, 1976; Ikai, & Steinhaus, 1961). This may include increased inhibition of antagonistic muscles and better co-contraction of synergistic muscles, increased activation of synergistic muscles, inhibition of neural protective mechanisms, and...

The *body composition changes* most common observed because of RE training are an increase in fat-free or lean body mass and a decrease the percentage of body fat (Baechle, 1994; Gettman et al. 1978; Goldberg, 1989; Hurley & Kokkinos, 1987; Kraemer, Deschenes, & Fleck, 1988). Such changes may be accompanied by changes in hormone concentrations in response to RE training (e.g., testosterone, human growth hormone) (e.g., Kraemer et al., 1992, 1991; Weiss, Cureton, & Thompson, 1983), but the results of studies in this area are not conclusive (Fahey et al., 1976; Skierska et al., 1976 (Baechle, 1994; Kraemer, and Daniels, 1986).

Resistance exercise training also induces *cardiovascular adaptations* (Kanakis, & Hickson, 1980; Lusiani et al., 1986; McCartney et al., 1993; Ricci et al., 1982). These include: (1) *Cardiac hypertrophy*, which have been reported in cross-sectional (Brown et al., 1983; Fleck et al., 1987; Longhurst et al., 1980; Pearson et al., 1986), and short-term (10-20 wk) longitudinal studies (Kanakis, & Hickson, 1980; Lusiani et al., 1986; Ricci et al., 1982) showing increases in echocardiographic imagine of diastolic posterior LV wall thickness (Brown et al., 1983; Pearson et al., 1986), and diastolic intraventricular septum (Brown et al., 1983); (2) *unchanged LV dimension and volume*, which have been reported in cross-sectional studies indicating that LV internal dimension and volume during systole and diastole have not demonstrated to be increased in RE athletes as compared to controls (Kraemer, Deschenes, & Fleck, 1988; Longhurst et al., 1980), as well in short-term longitudinal studies reporting no increases in systolic LV internal dimension (Kanakis, & Hickson, 1980) and diastolic LV internal dimension (Kanakis, & Hickson,
1980; Lusiani et al., 1986; Ricci et al., 1982); and (3) increased LV mass: which has been reported in cross-sectional studies showing absolute and relative increased LV mass in RE trained individuals compared to controls (Brown et al., 1983; Pearson et al., 1986).

Studies examining CV functional adaptations to RE training have observed including: (1) an greater absolute stroke volume in RE training athletes as compared to sedentary control subjects (Pearson et al., 1986). However, SV was not observed to be altered by exercise if corrected by body surface area or lean body mass (Fleck, 1988); (2) systolic function (percent shortening fraction, ejection fraction, and velocity of circumferential shortening) at rest in RE training athletes is not different from sedentary control individuals (Dickhuth et al., 1979; Longhurst et al., 1980a; Menapace et al., 1982; Pearson et al., 1986); (3) an enhanced diastolic function in cross-sectional studies examining cardiac hypertrophy in weight lifters (Colan et al., 1985; Pearson et al., 1986); and (4) no differences in resting heart rate (Colan et al., 1985; Fleck, & Dean, 1987), and blood pressure (Longhurst et al., 1980a, 1980b; Menapace et al., 1982; Pearson et al., 1986) in cross-sectional studies comparing highly trained weight lifters and sedentary subjects, and in training intervention studies involving young men (Lusiani et al., 1986; Seals et al., 1994).

In addition, it has been observed changes in acute CV response to RE. The well-established pressor response (HR and BP) to dynamic RE (Lentini et al., 1993, Lewis et al., 1985; MacDougall et al., 1985, 1992) has been reported to be lower in trained bodybuilders than in novice RE trained and non-resistance exercise trained individuals (Fleck, 1988) when performing RE at 60, 70, 80 and 90% of 1-RM, and at one repetition maximum. Similar results have been reported in longitudinal studies comparing RE
trained group for a short-term (e.g., 19 wks) to control group. That is, for a given absolute weight lifted, all response (e.g., HR, BP) were markedly reduced after training in the trained group (Seal et al., 1994).

Lastly, it should be noted that RE is not very effective in increasing VO$_{2\text{max}}$. Exceptions would be relatively untrained people, in whom increases ranging from 5% to 8% in aerobic power result from resistance training (Gettman et al. 1978; Fleck, 1988; Stone et al. 1983). Even increases in VO$_{2\text{max}}$ from circuit weight training programs (light loads, short rests, and multiple exercise) are lower than what would be expected from conventional endurance training programs (Baechle, 1994).

### 3.2 Risks of Resistance Exercise Training

Data regarding potential risks associated with RE testing and training in healthy young adult women, older men and women, and in moderate-to-high-risk cardiac patients, as well as patients with neuromuscular disorders and end-stage renal disease data are lacking (McCartney, 1999; Pollock et al., 2000). However, an approach of the risks and safety associated to RE testing and training might be suggested considering: (1) the results of safety of RE from studies in healthy and low- to moderate-risk CV patients (i.e., persons without resting or exercise-induced evidence of myocardial ischemia, severe LV dysfunction, or complex ventricular dysrythmias); (2) the physiological response to RE as compared to dynamic aerobic exercise; and (3) the well established risks associated with exercise testing (stress test) and vigorous dynamic exercise (ACSM, 2000; McCartney, 1999; Pollock et al., 2000).

Studies in healthy adults (MacDougall et al., 1992, 1985; McCartney et al., 1993; Lentini et al., 1993; Sale et al., 1994, 1993; Taaffe et al., 1999), older adults (Ades et al.,
1996; Brown et al., 1990; Charette et al., 1991; Fiatarone et al., 1994, 1990; Frontera et al., 1990, 1988; Yarashesli et al., 1993), and low-risk cardiac patients (Haslam et al., 1988; Kelemen et al., 1986; McCartney, 1998; Steward et al., 1998; Weicek et al., 1990) reported few orthopedic complications and no CV events (Pollock et al., 2000).

Accordingly, Gordon et al (1995) reported not significant CV events after determining the maximum weight that could be used to complete 1-repetition (i.e., 1-repetition maximum, 1-RM) strength testing (bench press, leg press, and knee extension) in 6653 healthy subjects aged 20 to 69 years who had undergone a preliminary medical examination and maximal treadmill testing (All participants had resting blood pressure ≤160/90 mm Hg). More recently, in a study evaluating both physiological and biological CV tolerance to RE in 65 healthy older adults (32 men/33 women, age=65-80 yrs), Bermon, Rama, & Dolisi found no significant increase in cardiac troponin I (cTnI) blood concentration secondary to exercise, despite a exercise-induced increases in SBP, DBP, and HR. They concluded the RE could be conducted in healthy older adults without clinical, electrical, and biological sign of myocardial ischemia (Bermon, Rama, & Dolisi, 2000).

The safety of RE training in studies with mild hypertension (Harris, & Holly, 1987) and cardiac patients (Haslam et al., 1988; Weicek et al., 1990) has been also reported. For example, Haslam et al, and Wiecek et al found intra-arterial BP during weight lifting in cardiac patients to be within a clinically acceptable range at 40% and 60% of 1-RM, and reported that all patients completed the weightlifting exercises without dyspnea, chest pain, significant dysrhythmias, or ischemic electrocardiographic changes (Haslam et al., 1988; Weicek et al., 1990).
Additionally, RE or circuit weight training used in physical-conditioning regimens of men with CAD, who had already been aerobically trained for 3 months or more, reported improvements in muscular strength and endurance at high (80% of 1-RM) and moderate (30% to 40% of 1-RM) training intensities, as well absence of anginal symptoms, ischemic ST-segment depression, abnormal hemodynamic, complex ventricular dysrhythmias, and CV complications. This suggests that strength testing and training are safe for clinically stable men with coronary disease who are actively participating in a rehabilitative program (Pollock et al., 2000). Lastly, it has been reported that many men can safety perform static-dynamic activity equivalent to carrying up to 30 pounds by 3 weeks after an acute MI (Wilke et al., 1985), although conventional participation guidelines have suggested that surgical and post-myocardial infarction (MI) patients should avoid RE training for at least 4 to 6 months (Kelemen, 1989; Sparling et al., 1990), Thus, it is possible that RE training could be initiated sooner, if low-weight programs are used (Pollock et al., 2000).

The second thought considered in an approach/rationale of risks of RE training might be the physiological response to RE as compared to dynamic aerobic exercise. For example, the physiological response to dynamic aerobic exercise is an increase in VO$_2$ and HR that parallels the intensity of the imposed activity and a curvilinear increase in SV. There is a progressive increase in SBP, with maintenance of or a slight decrease in the DBP, and a concomitant widening of the pulse pressure. Blood is shunted from the viscera to active skeletal muscle, where increased oxygen extraction widens the systemic (a – v) O$_2$ difference. Thus, aerobic exercise imposes primarily a volume load on the myocardium (Lind, & McNicol, 1967). Conversely, the acute circulatory response to RE
seems to yield a favorable balance between myocardial oxygen supply and demand; in patients with coronary artery disease, this probably accounts for the observations of reduced signs and symptoms of ischemia, and fewer wall motion abnormalities, than during dynamic activities such as walking and cycling (Butler et al., 1987; Haslam et al., 1988; Vander et al., 1986).

Lastly, it could be considered in this approach/rationale, without discarding the CV response differences in these two types of exercises, the well-documented risks associated with exercise testing (stress test), and vigorous exercises during dynamic exercise (ACSM, 2000; Cobb, & Weaver, 1986; Mittleman et al., 1993; Willich et al., 1993). For example, regarding the safety of peak or symptom-limit graded exercise testing, it has been estimated from a summary of 12 different reports involving nearly 2 million exercise tests (ACSM, 2000), that during or immediately after an exercise test the risk of death is less than or equal to 0.01%; acute myocardial infarction (MI) is less than or equal to 0.04%; and a complication requiring hospitalization (including acute MI and/or serious arrhythmias) is less than or equal to 0.2%. Therefore, the risks associated with submaximal physical fitness testing (e.g., submaximal cycle ergometer test), which has been administrated to thousands of adults (ages 18 to 65 years) in worksite health promotion programs and community health and fitness centers, appears to be even lower when no deaths, MIs, or lasting morbid events have been reported. (ACSM, 2000).

On the other hand, risks associated with vigorous dynamic exercise, given the increased myocardial demands, may precipitate CV events in persons with know or occult heart disease. The risk of CV complications appears to be increased during vigorous exercise compared with that observed at other time. For example, the death rate
of a survey reporting 1 jogging death per year for every 7620 joggers in Rhode Island, which corresponds to 1 death per 396,000 man-hours of jogging (Thompson et al., 1988), was 7 times the estimated death rate from heart disease during more sedentary activities (Koplan, 1979); for patients with CAD, the relative risk of developing cardiac arrest during vigorous exercise may be more than 100-fold greater than what might be expected to occur spontaneously (Cobb, & Weaver, 1986); and the relative risk of acute MI is also 2 to 6 times higher during vigorous physical exertion (6 or more metabolic equivalent, METs) than during other activities (Mittleman et al., 1993; Willich et al., 1993).

All above information about vigorous dynamic exercise appears to contradict the held belief that regular exercise reduces risks of CV events. However, the relative risk of cardiac arrest during exercise compared with that observed at other time is only 5 times greater among men with high levels of habitual physical activity, while it is 56 times greater among sedentary men (Siscovick et al., 1984). The overall risk of cardiac arrest among habitually active men is only 40% of that for sedentary men (ACSM, 2000).

In summary, RE testing and training are widely utilized as part of fitness programs for individuals of all ages, and appears to be remarkably safe. Therefore, the risks associated with RE testing and training appear to be lower than those associated with exercise testing (stress test) and vigorous dynamic exercise given the differences in CV responses to these two exercises. However, there is some evidence that RE training might be potential hazardous for a small percentage of the population. Haykowsky et al (1996) have documented three cases of nonfatal subarachnoid hemorrhage associated with weight-lifting training. It is likely that these individuals harbored a previously innocuous intracranial aneurysm, which ruptured in response to a significant increase in cerebral
arterial transmural pressure precipitated by the lifting. For the estimated 1% of the population with an undetected intracranial aneurysm, RE training may be inappropriate (Haykowsky et al., 1996), but at the present time routine detection of this defect is unavailable (For a review, see McCartney, 1999; Pollock et al., 2000, 1994).

3.3 Cardiovascular Benefits of Resistance Exercise Training in the Elderly

It is well established that aerobic exercise training induces substantial benefits on the CV system, but the extent of these effects is not completely defined. While many experts in the field (e.g., Hurley, & Roth, 2000) suggest that RE training can induce changes on CV functions in older adults (i.e., cardiovascular fitness, as assessed by time to exhaustion on a treadmill test), the extent to which this conclusion is actually supported by evidence appears to depend on how improvements on CV function or fitness are defined and the degree to which the magnitude of change constitutes a real benefit in older adults (Hurley, & Roth, 2000).

Resistance exercise training appears to induce exercise adaptations in older adults, similar to those in general healthy adult populations. These adaptations influence the CV system (McCartney et al., 1993), and musculoskeletal system (Fiatarone et al., 1994, 1990; Frontera et al., 1990) among others (see Table 4). With respect to CV function, the results of many studies have suggested that RE training programs do not increase VO\(_{2}\max\) in older adults (e.g., Ades et al., 1996; Hagberg et al., 1989a; Parker et al., 1996; Smutok et al., 1993; for a review, see Hurley, & Hagberg, 1998). However, few studies have revealed increase in VO\(_{2}\max\) (e.g., Frontera et al., 1990; Hagerman et al., 2000). Among the best studies in this area is that of Hagberg et al, who compared the effects of endurance or RE training on VO\(_{2}\max\) and the CV responses to exercise of old men and
women (age=70-79 yrs) and found that RE training neither elicited changes in VO$_{2\text{max}}$ nor effected CV responses to sub-maximal or maximal treadmill exercise (Hagberg et al., 1899a). Parker et al examined the effects of a 16-week RE training program on CV responses during a submaximal walk and a weight-load walking test in healthy older women (age=60-77 yrs) and reported no change in VO$_{2\text{max}}$ (Parker et al., 1996). Ades et al in a 12-week randomized controlled trial, compared RE training with a nonexercising control group and found that neither group showed a change in peak aerobic capacity or in whole-body composition (Ades et al., 1996). Smutok et al in a 20-week training program compared three different (RE, aerobic, and control) groups found that VO$_{2\text{max}}$ did not change in the RE training group.

On the other hand, Frontera et al reported a significant 5% increase in VO$_{2\text{max}}$ after 12-weeks RE training program in healthy older men (age=60-72 yrs). However, VO$_{2\text{max}}$ was measured during a cycle-ergometer exercise test, and since non-cyclists are usually unable to achieve true VO$_{2\text{max}}$ on a cycle-ergometer, it has been suggested that unlikely a true VO$_{2\text{max}}$ was measured on these subjects. It should be noted that in contrast to VO$_{2\text{max}}$ on the treadmill, cycle-ergometer performance appears to be limited by non-cardiovascular factors, such as leg strength or power (Hickson, Rosenkoetter, & Brown, 1980; Marcinik et al., 1991). Supporting the possibility that true VO$_{2\text{max}}$ did not change in the study of Frontera et al (1990) are the data of Hickson, Rosenkoetter, & Brown (1980), who found that 10-wks of RE training in young men resulted in a significant increase in peak VO$_2$ tested on a cycle-ergometer, but did not change true VO$_{2\text{max}}$ measured during a maximal treadmill exercise test (Hickson et al., 1980). Similar finding was found by Marcinik et al study, in which time to exhaustion on a cycle-ergometer was increased by
33% with circuit RE training, despite no significant change in treadmill VO$_{2\text{max}}$. More recently, Hagerman et al also found that 16-wks of RE training in older men (60-75 yrs) showed a 9% increase in relative peak VO$_2$ and time to exhaustion (endurance performance) in a treadmill test (Bruce protocol) (Hagerman et al., 2000). However, when peak VO$_2$ values were corrected for gains in fat free mass, the increment was reduced to a 5.7% increase, which might be explain by normal biological or methodological variation (Hurley et al., 1988; Hurley et al, 1984).

Additionally, it should be noted that RE among untrained people increases aerobic in the range of 5% to 8%, which appears to indicate that changes in aerobic metabolism and performance are not optimally affected with RE. Even increases in VO$_{2\text{max}}$ from circuit weight training programs (light loads, short rests, and multiple exercise) are lower than what would be expected from conventional endurance training programs (For a review, see Baechle, 1994).

Thus, according with the results above reported, RE training appears to induce little or no change on CV function (central or peripheral mechanisms) that would improve oxygen uptake in older adults. However, RE training might elicit other more generalized adaptations that might benefit the CV functions of older men and women. For example, despite no changes in VO$_{2\text{max}}$, Ades et al found that 12 week of RE training increased treadmill walking endurance at 80% VO$_{2\text{max}}$ by 38% in 65- to 79-year-old women, which was related to increased leg strength (Ades et al., 1996). Parker et al reported that 16-week of RE training decreased HR, BP, and RPP during submaximal and weight-loaded treadmill walking test in 60- to 77-year-old women (Parker et al., 1996). Thus, RE training appears to increase endurance performance, despite little or no changes in
VO$_{2\text{max}}$, which may suggest that VO$_{2\text{max}}$ may be good index of aerobic capacity, but it might not be a perfect predictor of endurance performance (Costill, Thomason, & Roberts, 1973).

The specific mechanisms for these submaximal CV adaptations to RE training are not well understood, but it has been suggested that potential explanation may be related to: (1) increases in muscular strength (Hickson et al., 1988; Hickson, Rosenkoetter, & Brown, 1980); and (2) changes in fiber type recruitment (i.e., greater rate of type I and a reduced rate of type II muscle fiber recruitment), and less occlusion of blood flow and increased lactate threshold (Hickson et al., 1980; Marcinik et al., 1991; for a review, see Hurley, & Roth, 2000). Additionally, it has been suggested that RE training fails to produce substantial improvements in aerobic capacity because RE stimulates a catecholamines increases approximately 7-fold higher than aerobic training in the same individual at the same oxygen cost. This results in HR elevations that are disproportionate to the low aerobic demands of the muscle during training, leading to an oxygen pulse during exercise below the threshold necessary to elicit the kind of CV adaptations observed with aerobic exercise training (Hurley et al., 1984, for a review, see Hurley, & Roth, 2000). However, some studies have failed to established increases in plasma concentration of catecholimines.
<table>
<thead>
<tr>
<th>Author</th>
<th>Design</th>
<th>Participants</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hagerman et al. 2000</td>
<td>16-wk, 2x/wk, 3-set, 6-8 rep RT, randomly RT or CON</td>
<td>N=18 sedentary men, age=60-75 yrs</td>
<td>RT group ↑ muscle strength, endurance performance, &amp; VO\textsubscript{2max}, ↓ % fat, ⇔ HR, BP, capillary density, &amp; serum lipids.</td>
</tr>
<tr>
<td>Haykowsky et al. 2000</td>
<td>16-wk, 3x/wk, 60-80% 1RM RT, randomly RT or CON</td>
<td>N=20 men, mean age= 68±4 yrs</td>
<td>RT group ↑ upper &amp; lower body muscle strength, ⇔ LV cavity size, wall thickness, mass, or systolic function.</td>
</tr>
<tr>
<td>Martel et al. 1999</td>
<td>24-wk, 3x/wk. Single group pre/post</td>
<td>N=21; men &amp; women, mean age= 69 ± 1 yrs</td>
<td>↑ Upper &amp; lower body muscle strength, ↓ resting SBP, &amp; DBP.</td>
</tr>
<tr>
<td>Ades et al. 1996</td>
<td>12 wks, 3x/wk, 3-set, 8-rep RT, randomly RT &amp; CON.</td>
<td>N=24 men &amp; women, mean age=70.4 ± 4 yrs</td>
<td>RT group ↑ leg strength and walking. ⇔ Peak VO\textsubscript{2max}, &amp; body composition</td>
</tr>
<tr>
<td>Parker et al. 1996</td>
<td>16-wk, 3x/wk, 1-2-set, 12-15 rep RT. Single group pre/post</td>
<td>N=14 women; age=60-70 yrs</td>
<td>↑ Isotonic and isometric strength. ↓ HR, BP, &amp; RPP during submaximal, &amp; weight-load walking. ⇔ VO\textsubscript{2max}</td>
</tr>
<tr>
<td>McCartney et al. 1993</td>
<td>12-wk, 3x/wk, 2-4-set, 10-15 reps. RT, single group pre/post</td>
<td>N=15 men. Mean age=66.3 ± 4 yrs</td>
<td>↑ Upper &amp; lower body muscle strength. ↓ HR, SBP, DBP, MAP, RPP at absolute load, but ⇔ at the same relative load.</td>
</tr>
<tr>
<td>Smutok et al. 1993</td>
<td>20-wk RT or AT; randomly RT, AT or CON.</td>
<td>N=37 untrained men, mean age=50 ± 9yrs.</td>
<td>RT group ⇔ VO\textsubscript{2max}, lipoprotein or lipid profile, &amp; BP. plasma glucose level, &amp; insulin level during fasting.</td>
</tr>
<tr>
<td>Cononie et al. 1991</td>
<td>24-wks, 3x/wk, 1 set, 8-12-rep, Randomly RT, AT, or CON</td>
<td>N=49 men &amp; women, mean age=70-79 yrs</td>
<td>RT group ↑ muscle strength. ⇔ VO\textsubscript{2max}, CO, BP, peripheral vascular resistance, catecholamines, angiotensin I &amp; II</td>
</tr>
<tr>
<td>Frontera et al. 1990</td>
<td>12-wks, 3x/wk, 3-set, 80% 1RM, single group pre/post</td>
<td>N=12 healthy men, age=60-72 yrs</td>
<td>↑ Leg muscle strength, VO\textsubscript{2MAX}, capillary density, &amp; citrate synthase activity. ⇔ Hemoglobin, plasma volume, &amp; TBV.</td>
</tr>
<tr>
<td>Hagberg et al. 1989a</td>
<td>26 wks. 3 groups: RT, AT, CON</td>
<td>N=28 men &amp; women; age=70-79 yrs.</td>
<td>RT group ⇔ VO\textsubscript{2max} nor max or submaximal CV response. ↑ Upper and lower body muscle strength.</td>
</tr>
</tbody>
</table>

↑= Increase; ↓= decrease; ⇔ = no change; RT= resistance training; CON= control group; AT= aerobic training, TBV= total blood volume
In regard to effects of RE training on LV morphology changes (morphological heart changes) in older adults, in a study with 20 healthy older adults (mean age= 68 ± 4 years) using 2-dimensional echocardiography, Haykowsky et al found that 16-wks of RE training was not associated with changes in LV cavity size, wall thickness, mass, or systolic function after 4, 8, 12, and 16 weeks of exercise in older adults (Haykowsky et al., 2000). These findings are similar to those found in short-term longitudinal studies with healthy younger adults reporting unchanged systolic LV internal dimension (Kanakis, & Hickson, 1980), diastolic LV internal dimension (Kanakis, & Hickson, 1980; Lusiani et al., 1986; Ricci et al., 1982), and systolic LV function (Martel et al., 1999; for a review, see Effron, 1989; Fleck, 1988). However, LV wall thickness and mass findings are opposite as compared to those found in healthy younger adults, which report increases in diastolic posterior LV wall thickness (Brown et al., 1983; Pearson et al., 1986), diastolic intraventricular septum (Brown et al., 1983) and LV mass (Brown et al., 1983; Pearson et al., 1986). Thus, it appears that short-term RE training does not have any effect in morphological structure of the heart in healthy older adults. However, long-term longitudinal training studies, as well cross-sectional studies assessing morphological changes with RE training in healthy older adults are lack.

In the same study, Haykowsky et al report no change in systolic function at resting condition (Haykowsky et al., 2000), which is in agreement with short-term studies with younger adults reporting the same results (Lusiani et al., 1986). However, at the same time is opposite to studies with younger adults reporting increases in systolic function indexes (e.g., Kanakis, & Hickson, 1980). Thus, short- and long-term longitudinal studies are needed before any conclusions can be drawn.
The same result is reported regarding resting HR and BP, that is no change with short-term RE training (Hagerman et al., 2000; Haykowsky et al., 2000). Such as inference is consistent with results of cross-sectional and longitudinal studies with healthy younger adults. With respect to morphological changes, there is a lack of intervention about the influence of RE in systolic and diastolic function.

Lastly, there are effects of RE training on the acute LV response to resistance exercise in older adults. Short-term longitudinal studies have demonstrated that RE training attenuates the pressor response to dynamic RE in older adults (Hagerman et al., 2000; Martel et al., 1999; McCartney et al., 1993). For example, McCartney et al found that after 12 weeks of RE training, the 1RM increased by 24 (legs) to 54% (arms) and there was a marked attenuation of HR and arterial BP during exercise when subjects lifted the same absolute load. However, after training, during lifting at 60, 80, and 100% of the post-training 1RM, the HRs and BPs were the same as those during pre-training testing when the same relative, but lighter, absolute loads were used. It was suggested that the circulatory response to weight lifting was mediated by a feedforward “central command” mechanism coupled to the relative intensity rather than to the absolute level of force (McCartney et al., 1993). Parker et al in the same study examining the effects of a RE training on CV stress during a submaximal walk and a weight-load walking test in healthy older women reported that RE training decreased HR, SBP, and RPP during the performance of these two treadmill walking tests (Parker et al., 1996). The most common RE training-induced adaptation in the musculoskeletal system (Fiatarone et al., 1990; 1994; Frontera et al., 1990) is an increase in muscle strength. Muscle strength assessed with upper and lower 1-RM strength test has been suggested as a result of an increase in
muscle fiber size (hypertrophy). For example, Fiatarone et al found that RE training would help to preserve and enhance muscular strength and endurance (muscular fitness), which, in turn, might help to prevent falls, improve mobility, and counteract muscle weakness and frailty (Fiatarone et al., 1994; Taunton et al., 1997). Consequently, muscular fitness in older men and women might allow the performance of activities of daily living with less effort.

In summary, in addition to preserving and enhancing muscular strength and endurance, short-term RE in older adults appears to induce CV benefits similar to those in healthy younger adults, without any greater risk of adverse responses than that associated with vigorous dynamic exercise. Therefore, RE may allow older adults to perform daily activities with lower pressor responses with a prolonged time to fatigue, and thereby extend functional lifespan.
CHAPTER IV. CURRENT TRENDS IN STRENGTH TRAINING PRESCRIPTION FOR THE ELDERLY

4.1 Historical Evolution of Physical Activity Recommendations

Between 1978 and 1998, several exercise recommendations and position statements about how to perform specific physical activities for the purpose of obtaining fitness and health benefits were presented by different public and private organizations including the American Medical Association (AMA), American College of Sports Medicine (ACSM), the American Heart Association (AHA), Centers for Disease Control and Prevention (CDCP), the President's Council on Physical Fitness and Sports (PCPFS), and the Healthy People 2000.

These private and public organizations issued position statements in an effort to contribute to the standardization of guidelines for recommending physical activity and exercise programs for the purposes of improving health and/or physical fitness. These recommendations and position statements were based on clinical experience and on scientific evidence supporting the beliefs of the benefits of physical activity, and a growing understanding of how physical activity affects health and physiologic function (Cureton 1947, 1969; Taylor, Anderson, & Keys, 1957; for a review, see Surgeon General Report, 1996). Among the earliest to appear were the ACSM exercise guidelines, which focused primarily on aspects of physical fitness (e.g., cardiorespiratory fitness, and body composition) (ACSM, 1978). More recent guidelines from the ACSM (1995, 1998), Pate et al (1995), and the Surgeon General Report (1996), included the possibility of using lower level of exercise intensity to achieve health benefits.

In a more detailed evolution of physical activity recommendation (Surgeon General Report, 1996), additional information indicated that the first recommendations for
physical activity to achieve fitness and health benefits were based on systematic comparisons of effects from different profiles of exercise training (Cureton, 1947; Karvonen, Kentala, & Mustala, 1957; Christensen, 1960; Yakolav et al., 1961; Reindell, Roskamm, & Gerschler, 1962). Later in the 1960s and 1970s, based on clinical and scientific data available at that time, different investigators and organizations have recommended specific physical activity programs or exercise prescriptions for improving physical performance capacity or health (President's Council on Physical Fitness 1965; AHA 1972, 1975; ACSM, 1975). For example, the 1978 ACSM position statement recommended a frequency of exercise training of 3-5 days per week, an intensity of training of 60-90 percent of $HR_{\text{max}}$ (equivalent to 50-85 percent of $VO_2_{\text{max}}$ or HR reserve), a duration of 15-60 minutes per training session. Rhythmical and aerobic use of large muscle groups through such activities as running or jogging, walking or hiking, swimming, skating, bicycling, rowing, cross-country skiing, rope skipping, and various endurance games or sports. In so doing, the 1978 ACSM position statement primarily addressed cardiorespiratory fitness and body composition.

Table 5. ACSM Position Stand, 1978

<table>
<thead>
<tr>
<th>Edition</th>
<th>Frequency</th>
<th>Intensity</th>
<th>Duration</th>
<th>RE training</th>
</tr>
</thead>
<tbody>
<tr>
<td>1978</td>
<td>3-5 d/w</td>
<td>60-90% $HR_{\text{max}}$</td>
<td>15-60 min</td>
<td>Not address</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50-85% $VO_2_{\text{max}}$/HRR</td>
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</table>

[Adapted from Surgeon General Report, 1996]

In the 1990s, the ACSM recommendations and position statement (1990, 1995) added the development of muscular strength and endurance as a major objective. In the 1990 edition, the recommended frequency, intensity, and mode of exercise remained similar, but the duration was slightly increased from 15-60 minutes to 20-60 minutes per session,
and moderate-intensity RE training (one set of 8-12 repetitions of 8-10 different exercises at least 2 times per week) was suggested to develop and maintain muscular strength and endurance.

**Table 6. ACSM Position Stand, 1990, 1995**

<table>
<thead>
<tr>
<th>Edition</th>
<th>Frequency</th>
<th>Intensity</th>
<th>Duration</th>
<th>RE training</th>
</tr>
</thead>
<tbody>
<tr>
<td>1990</td>
<td>3-5 d/w</td>
<td>60-90% HR$_{\text{max}}$</td>
<td>20-60 min</td>
<td>1 set, 8-12 reps, 8-10 exercise, 2 d/w</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50-85% VO$_{2\text{max}}$/HRR</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1995</td>
<td>3-5 d/w</td>
<td>60-90% HR$_{\text{max}}$</td>
<td>20-60 min</td>
<td>1 set, 8-12 reps, 8-10 exercise, 2 d/w</td>
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<td></td>
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<td>50-85% VO$_{2\text{max}}$/HRR</td>
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<tr>
<td></td>
<td></td>
<td>40-50% VO$_{2\text{max}}$/HRR</td>
<td>20-30 min</td>
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[Adapted from Surgeon General Report, 1996]

In the 1995 edition, the recommended frequency, intensity, duration, and mode of exercise remained similar for healthy adults. However, in this edition (1995) is recognized that activities at moderate-intensity (40-50% VO$_{2\text{max}}$/HRR) during a shorter time (20-30 min) might have health benefits independent of cardiorespiratory fitness in unfit individuals. Hence, an important distinction was made between physical activities as it related to health versus fitness. It is recognized that the quantity and quality of exercise needed to obtain health-related benefits may differ from what is recommended for fitness benefits. Therefore, it is now clear that lower levels of physical activity than recommended by that position statement may reduce the risk for certain chronic degenerative diseases and yet may not be of sufficient quantity or quality to improve some physiological parameters (e.g., VO$_{2\text{max}}$). ACSM recognizes the potential health benefits of regular exercise performed more frequently and for longer duration, but at lower intensities than prescribed in this position statement (Pate et al., 1995).

Over time, interest developed in potential health benefits of more moderate forms of physical activity, and attention began to shift to alternative physical activity regimens.
(Haskell 1984; Blair Kohl, Gordon 1992; Blair 1993). For example, between the 1970s and the mid-1990s, exercise training studies conducted on middle-aged and older persons and on patients with lower functional capacity demonstrated that cardiorespiratory and health-related benefits can be obtained at more moderate levels of activity intensity than previously realized. As results of these findings, Pate et al. (1995), and Surgeon General Report (1996), without leaving to recognize the importance of fitness components, reported the possibilities of using other level of physical activity intensity, as understood that benefits of less vigorous activity are also possible to achieve better health and fitness status, and that cardiorespiratory fitness gains are similar when physical activity occurs in several short sessions (e.g., 10 minutes) as when the same total amount and intensity of activity occurs in one longer session (e.g., 30 minutes). Thus, the most recent CDC-ACSM guidelines recommend that all adults perform 30 or more minutes of moderate-intensity physical activity on most, and preferably all, days -either in a single session or "accumulated" in multiples bouts, each lasting at least 8-10 minutes (Pate et al., 1995).

In summary, the availability of scientific data has driven the evolution of exercise recommendations and position statements across time toward specific approaches (e.g., physical fitness, or health) and to promote the importance of a well-rounded exercise program for adult fitness and health programs. Therefore, given the enormous physiological and psychological differences (e.g., age, health status, physical fitness) among individuals, both physical fitness- and health-approach of the physical activity recommendations might be essential and mandatory to achieve desired outcomes. Further, influence of age, health status, and individual interest, abilities, and limitations; as well that the physiological and perceptual response to acute exercise and chronic
adaptations to exercise training vary in terms of magnitude and rate among individuals all are the significant relevance in exercise prescription.

4.2 Current Physical Activity Recommendations

The American College of Sport Medicine (ACSM, 1998) recommendation and position stand focus mainly toward body composition, cardiorespiratory fitness, muscle strength and endurance, and flexibility. However, it is also recognized that other ways of exercising (e.g., low intensity-long duration session, multiple session of short duration) might improve and maintain health status and fitness level, according to initial fitness level, presence of medications, risk of cardiovascular disease or orthopedic injury, individual variability relative to skill and enjoyment, functional capacity.

The exercise program consists of warm-up (~10 min), endurance phase (20 to 60 min), recreational activities (optional), and a cool-down (5 to 10 min). It is recommended that while endurance training activities should be performed 3 to 5 days per week, complementary flexibility and RE training may be undertaken at a reduced frequency (2 to 3 days per week). Flexibility training can be also included as part of the warm-up or cool-down, or undertaken at a separate time. Resistance exercise training is often performed on alternate days when endurance training is not; however, both activities can be combined into the same workout. Endurance and RE training should be prescribed in specific terms of intensity (load), duration, frequency, and type of activities.

Increase in cardiorespiratory fitness is measured by assessing the change in VO$_{2\text{max}}$, which is directly related to the frequency, duration, and intensity of exercise. Thus, it is recognized that the improvement in VO$_{2\text{max}}$ (e.g., ranging from 10 to 30%) would depend on the interaction of these prescriptive components and initial fitness level.
Accordingly, the following recommendations for each component of exercise training are given: **Mode of exercise**: It is recommended that exercise involves the use of large muscle groups over prolonged periods and is rhythmic and aerobic in nature (e.g., walking, hiking, running, machine-based stair climbing, swimming, cycling, rowing, combined arm and leg ergometry, dancing, skating, cross-country skiing, rope skipping, or endurance game activities). **Exercise intensity**: Intensity and duration of exercise determine the total caloric expenditure during a training session, and are inversely related. That is, similar increase in cardiorespiratory endurance may be achieved by a low intensity, long duration session as well as a higher intensity, shorter duration session. ACSM recommends an intensity of exercise corresponding to between 55 and 65% (55/65%) to 90% of \( HR_{\text{max}} \), or between 40 and 50% (40/50%) to 85% of oxygen uptake reserve (\( VO_{2R} \)) or HR reserve (HRR). The \( VO_{2R} \) is the difference between \( VO_{2\text{max}} \) and resting \( VO_2 \). Similarly, the HRR is the difference between \( HR_{\text{max}} \) and resting HR. The lower intensity values (i.e., 40-49%) of \( VO_{2R} \) or HRR and 55-64% of \( HR_{\text{max}} \), are more applicable to individuals who are quite unfit. **Exercise duration**: The duration of an exercise session interacts with the intensity to result in the expenditure of a sufficient number of calories to achieve health, fitness, and weight management goals. The duration of exercise recommended by the ACSM reflects that interaction (20 to 60 minutes of continuous or intermittent [minimum of 10-minute bouts] aerobic activity accumulated throughout the day). **Exercise frequency**: Although deconditioned persons may improve cardiorespiratory fitness with only twice-weekly exercise, optimal training frequency appears to be achieved with 3 to 5 workouts per week. The additional benefits of more frequent training appear to be minimal, whereas the incidence of lower extremity injuries
increases abruptly. Consequently, the ACSM recommends an exercise frequency of 3 to 5 days per week. *Caloric thresholds for adaptation:* The interaction of intensity, duration, and frequency determines caloric expenditure from the activity. However, the thresholds necessary to bring about significant improvement in \( \text{VO}_{2\text{max}} \), weight loss, or reduced risk of premature chronic disease may be different. The ACSM recommends a target range of 150 to 400 kcal of energy expenditure per day in physical activity and/or exercise (Haskell, 1994; Surgeon General Report, 1996). The lower end of this range represents a minimal caloric threshold of approximately 1000 kcal per week from physical activity and should be the initial goal for previously sedentary individuals. Based on the dose-response relationships between physical activity and health and fitness, individuals should be encouraged to move toward attainment of the upper end of the recommended range (e.g., 300 to 400 kcal per day from activity) as their fitness level improve during the training program. *Rate of progression:* The recommended rate of progression in an exercise-conditioning program depends on functional capacity, medical and health status, age, and individual activity preferences and goals. For apparently healthy adults, the endurance aspect of the exercise prescription has 3 stages of progression: initial conditioning stage, improvement stage, and maintenance stage. The *initial conditioning stage* should include light muscular endurance exercises and moderate level aerobic activities (40 to 60% of HRR) in order to avoid muscle soreness, discomfort, and injury; *in the improvement stage*, the goal is to provide a gradual increases in the overall exercise stimulus to allow for significant improvements in cardiorespiratory fitness; and, *in the maintenance stage*, the goal is to maintain for a long-term the cardiorespiratory fitness developed during the improvement stage.
Musculoskeletal flexibility: Flexibility exercises are recommended to maintain adequate range of motion in all joints for optimal musculoskeletal function. Therefore, it is recognized that preventive and rehabilitative exercise programs should include activities that promote the maintenance of flexibility. Lack of flexibility is prevalent in the elderly and often contributes to a reduced ability to perform activities of daily living (ADL).

Muscular fitness: The ACSM (1990; 1995) recommends resistance exercise (RE) training of moderate intensity (i.e., sufficient to develop and maintain muscular fitness and fat-free mass) as an integral part of adult fitness and rehabilitative exercise programs. It is recognized that in addition to the development and maintenance of muscular strength and muscle mass, the physiological benefits of RE training include increases in bone mass and the strength of connective tissue. These adaptations are beneficial for middle-aged and older adults, and, in particular, postmenopausal women who may experience a more rapid loss of bone mineral density. Muscular strength and endurance are developed by what is known as the "overload" principle (i.e., by increasing the resistance to movement or the frequency or duration of activity to level above those normally experienced). Though controversy exists as to the single most effective method of enhancing muscular strength and endurance, it appears that Muscular strength is developed using weights that elicit maximal or nearly maximal muscle tension with relatively few repetitions. Muscular endurance, on the other hand, may be addressed using lighter weights with a greater number of repetitions. To elicit improvement in both muscular strength and endurance, most expert recommend 8 to 12 repetitions for healthy participants under 50 to 60 years of age and 10 to 15 repetitions for individuals older than 50 to 60 years of age. The intensity of RE training can be manipulated by varying the
weight, the number of repetitions, the length of the rest interval between exercises, or the number of sets of exercise completed. The following RE training guidelines are recommended for the apparently healthy adult: perform a minimum of 8 to 10 separate exercises that train the major muscle groups (arms, shoulders, chest, abdomen, back, hips, and legs); perform a minimum of 1 set of 8 to 12 repetitions of each these exercises to the point of volitional fatigue; perform these exercises 2 to 3 days per week.

_American Heart Association (AHA, 1995)._ The American Heart Association (AHA) is another national organization that has written position statements related to physical activity benefits, and recommendations for physical activity program and exercise prescription. In the position statement and exercise recommendations, AHA (1995) not only provides general and specific revised standards and guidelines for the exercise testing and prescription, but also some specific recommendations or training technique for individuals free from clinical manifestations of cardiovascular disease as well as those with know cardiovascular disease.

Some of the most relevant general standards and guidelines for exercise testing and prescription in both health status, healthy and diseased persons, are the following: (1) care must be taken to ensure that apparently healthy individuals who are beginning an exercise program do not have detectable disease and that subjects with know disease are stable with no evidence of new or changing symptoms, (2) medical clearance should be obtained before entry into exercise training programs unless the anticipated activity is low level (less than 50% of maximum capacity, e.g., moderate walking), (3) exercise testing is helpful in establishing guidelines for exercise training in apparently healthy adults and is mandatory for subjects with know or suspected cardiovascular disease, (4)
care should be taken to exclude from training subjects with evidence of unstable heart
disease such as angina, uncontrolled heart failure, or arrhythmia, and (5) training
programs for subjects with cardiovascular disease should be medically supervised until safe levels of activity have been established. It is required the physician presence.

Regarding the specific standards and guidelines for exercise testing and prescription,
and in consideration of important factors of risk associated with exercise (e.g., age,
presence of heart disease, and intensity of exercise), it is recommended that: (1) anyone who plans to begin an exercise program more vigorous than walking should have a current (within 6 to 12 months) physical examination; (2) individuals under the age of 40 years who have no symptoms of cardiovascular disease, no major coronary risk factors, and no physical findings (including murmurs and hypertension) can be considered free of disease, do not need an exercise test, and should not be restricted in their exercise program; (3) individuals 40 years of age or older or those with symptoms such as chest pain, abnormal physical examination suggesting heart disease, or two or more major coronary risk factors should have a symptom-limited, maximum exercise test (unless otherwise contraindicated), if they plan to participate in vigorous exercise (such as jogging or running); (4) if an exercise test is not done or is abnormal, these individual should restrict their activities to moderate intensities; and (5) due to that the risks of serious complications of physical activity are highest during vigorous exercise in individuals with heart disease, screening should ensure that cardiovascular disease is not present, and that the physical activity is limited to low or moderate intensities (walking or the equivalent), or that activity is medically supervised.
Related to training technique for individuals free from clinical manifestations of cardiovascular disease and individuals with known cardiovascular disease; in general, it is established that (1) any physical activity performed for training should be assessed in terms of intensity, frequency, duration, mode and progression. (2) Warm-up and cool-down: exercise at a low intensity for 5 to 10 minutes before (warm-up) and after (cool-down) the training session, such activities help stretch and warm up muscles and ligaments in preparation for the activity session. The cool-down period also prevents hypotension, which may occur with sudden cessation of exercise. (3) Mode of exercise: activities that cause the greatest increase in VO2max have certain characteristics that, when present, are said to qualify the exercise as endurance or cardiovascular. These characteristics include dynamic exercise, alternately contracting and relaxing the muscle (as opposed to isometric or resistive exercise), and large muscle group activities (e.g., walking or running, swimming, cycling, stair-stepping, and cross-country skiing). Subjects with cardiovascular disease should do such activities of higher intensity with care. (4) Frequency, duration and intensity: exercise must be performed at least three times per week for a minimum of 30 minutes per session at a minimum intensity of 50% to 60% VO2max. The intensity of activity needed to improve physical conditioning varies among individuals and may be as little as 50% of VO2max. An exercise intensity-duration relation is likely, so low-intensity exercise requires more time to increase functional capacity than higher intensity exercise. From a health and conditioning standpoint, the major advantage of moderate- and low-intensity exercise is less likelihood of complications, whereas vigorous exercise has the advantage of accomplishing the goal in less time. (5) Resistance exercises: Resistance exercise involves activities that use low
or moderate repetition movements against a resistance, generating a rise in muscle tension that ultimately yields increase in muscular strength. Weight lifting is the prototype resistance exercise. The increased muscle tension during such exercise leads to both a restriction in muscle blood flow during contraction due to compression of arterioles and capillaries that perfuse the muscle bed and a centrally mediated pressor response. As such, resistance training is encouraged as part of the overall exercise program for most subjects, and should be perform in a variety of body positions, both to isolate specific muscle groups and to ensure maximum orthopedic stability. To avoid the additive cardiovascular response of the Valsalva maneuver, subjects should be trainer to exhale during the contraction phase of the movements. Resistance training should include 8 to 10 exercises that train the major muscle group of the body, e.g., arms, shoulder, chest, abdominal, back/trunk, hip, and leg. The intensity of each weight training exercise can be adjusted by alterations in any of the following factors: weight load, number of repetitions by sets, number of sets, and rest period between sets. Generally, for subjects with cardiovascular disease, 2 to 3 days per week of resistance training using one set of 10 to 15 repetitions to moderate fatigue is recommended. Once 15 repetitions can be completed, the weight can be increased an additional 5%. Initial weight training activities are usually introduced during the first weeks of an outpatient program. They include the use of light calisthenics and 3- to 5-lb dumbbell weights. Later in the program, if subjects are medically stable, they can be cleared for regular weight training activities using barbells and weight machines. This sequence of range of motion exercise and strength training has been shown to be safe for use with both MI and CABs patients. During this early period, resistance training should only employ lightweight. Clinical
experience has shown that less than 5% of CABS subjects have any contraindications to this exercise.

AHA, in its 1995 position statement and exercise recommendation provides some individual guidelines for cardiovascular exercise, among them are those of: exercise only when feeling well; do not exercise vigorously soon after eating, wait at least 2 hours; adjust exercise to the weather; slow down for hills; wear proper clothing and shoes; understand personal limitations; select appropriate exercise; be alert for symptoms, such as discomfort in the upper body, faintness accompanying the exercise, discomfort in bones and joints either during or after exercise, chronic fatigue, sleeplessness, and aches and pains in the joints.

The specific recommendations to individuals with cardiovascular disease involve the following general principle of exercise prescription in two situations: (a) Prescription in the absence of ischemia or significant arrhythmia, the exercise intensity should approximate 50% to 80% of VO2max which can be ascertained by an exercise test (if a test id not done initially, the measure of 20 beats per minute above resting heart rate is adequate until testing is performed); and (b) prescription in the presence of ischemia or arrhythmia, an exercise test is essential for this type of prescription. The exercise test is performed in the usual fashion, but the conditioning work intensity is derived from the heart rate at 50% to 60% of heart rate maximum can be used if it falls at least 10 beats per minute bellow the abnormal level. Otherwise, the recommended training heart rate is 10 beats per minute less than that associated with the abnormality.

In addition, level of supervision is indicated as an important consideration. There are two general categories in this regards: (a) medically supervised programs that should be
provided for moderate- to high-risk subjects, because these individuals are at increased risk for complications associated with vigorous physical activity, and (b) non-medically supervised programs that should be provided for low-risk subjects.

*Healthy People 2000.* A position statement and physical activity recommendation are pointed out through the year 2000 objectives. These recommendations are made in consideration of the scientific evidence suggesting that regular physical activity can help to prevent and manage some disease, such as coronary artery disease, hypertension, noninsulin-dependent diabetes mellitus, osteoporosis, obesity, and mental health problems (e.g., depression and anxiety), as well as the observed inverse relationship between regular physical activity and rates of colon cancer, stroke, and back injury. On average, physically active people outlive those who are inactive, and regular physical activity can also help to maintain the functional independence of older adults and enhance the quality of life for people of all age.

The goals of healthy people 2000 include: (1) at least 30 percent the proportion of people age 6 and older in regularly (preferably daily), light to moderate physical activity for at least for 30 minutes; (2) to achieve at least 20 percent people aged 18 and older, and at least 75 percent the proportion of children and adolescents aged 6 through 17 in vigorous physical activity that promotes the development and maintenance of cardiorespiratory fitness at a frequency of 3 or more days per week, and for at least 20 minutes per session; and (3) to involve at least 40 percent of people age 16 and older in regular physical activities that enhance and maintain muscular strength, muscular endurance, and flexibility.
Other aspects of the Healthy People 2000 objectives include the promotion of physical activity and fitness programs in settings such as physical education classes and worksites.

*Center for Disease Control and Prevention and ACSM (CDC/ACSM):* In the recommendation from the Centers for Disease Control and Prevention (CDC) and the American College of Sports Medicine (Pate et al., 1995), the main objective is to encourage increased participation in physical activity among Americans of all ages by issuing a public health recommendation on the types and amounts of physical activity needed for health promotion and disease prevention. In general, it is recommended that every US adult should accumulate 30 minutes or more of moderate-intensity physical activity on most, preferably all, days of the week. This recommendation is proposed because the current low-participation rate may be due in part to the misperception of many people that to achieve health benefits they must engage in vigorous, continuous exercise. However, the scientific evidence demonstrates that accumulation of 30 minutes per day of moderate-intensity physical activity provides substantial health benefits.

In conclusion, a new understanding of the benefits of less vigorous activity is growing. During the last years, the ACSM, the CDC, the AHA, the PCPFS, and the INH have all recommended regular, moderate-intensity physical activity as an option for those who get little or no exercise. The Healthy People 2000 goals for the nation's health have recognized the importance of physical activity and have included physical activity goals. The 1995 Dietary Guidelines for Americans, the basis of the federal government's nutrition-related programs, included physical activity guidance to maintain and improve weight - 30 minutes or more of moderate-intensity physical activity on all, or most, days.
of the week. On the other hand, accumulating physical activity over the course of the day has been included in recent recommendations from the CDC and ACSM, as well as from the NIH Consensus Development Conference on Physical Activity and Cardiovascular Health. This report based on both well-established findings and newer research results that await replication and amplification, suggests to pay attention to recent findings from three studies showing that cardiorespiratory fitness gains are similar when physical activity occurs in several short sessions (e.g., 10 minutes) as when the same total amount and intensity of activity occurs in one longer session (e.g., 30 minutes).

In summary, all these current exercise recommendations and position statements recognize the benefits of the physical activity to the health, and its positive effects in reducing the risks of some diseases, so that physical activity is recommended to improve and maintain fitness, health and the quality of life. The rationale for each exercise recommendation is based on clinical experience and scientific data showing the effects and benefits of the physical activity, and in the growing understanding of how physical activity affects physiologic function in the body. So, there is a general agreement regarding the health benefits of regular physical activity. Lastly, as said previously, all these physical exercise recommendations promote the importance of a well-rounded physical training program including aerobic endurance exercise and RE training for adult fitness and health programs. Therefore, the general principles of exercise prescription apply to adults of all ages. Relative adaptations to exercise are also similar to other age groups. For example, the percent improvement in $\text{VO}_{2\text{max}}$ in older adults is comparable to that reported in younger populations (ACSM, 2000).
4.3 Exercise Prescription for Older Adults

Recommendations for exercise and physical activity prescription for older adults have been published by individual investigators (Evans, 1999; Feigenbaum, & Pollock, 1999; Kligman, & Pepin, 1992; Pollock et al., 2000, 1994) and organizations (e.g., ACSM, 1995, 1998; Surgeon General Report, 1996). These recommendations promote the importance of a well-rounded program including aerobic endurance exercise and resistance training for older adult fitness and health programs (Evans, 1999; Feigenbaum, & Pollock, 1999; Kligman, & Pepin, 1992; Pollock et al., 2000, 1994). The general principles of exercise prescription (components of the training session, cardiorespiratory fitness, energy expenditure goals, rate of progression, musculoskeletal flexibility, muscular fitness, maintenance of the training effect, and program supervision) apply to exercise prescription in fitness and health programs for older adults. However, there are some additional recommendations as to the manner in which these principles are applied (Pollock et al., 2000, 1994).

These additional recommendations include: (1) to understand older adults needs, goals, physical and health status, personal preferences, and available time, equipment, and facilities (Pollock, & Wilmore, 1990; Larson, & Bruce, 1987; Wheat, 1987); (2) even though the improvement of physical fitness could be an important consideration when designing exercise programs for older adults, enhancing the ability to perform daily activities and improving and maintaining the quality of life will be the most important goal; (3) the elderly participant is more fragile and thus more susceptible to fatigue, orthopedic injury, and possible CV problems (Pollock, & Wilmore, 1990; ACSM, 1991, 1995; Fletcher et al., 1990), thus the exercise prescription for the elderly will include
activities of low impact, that is, activities that produce less impact force on the
musculoskeletal/joint structure (Pollock, & Wilmore, 1990; Fletcher et al., 1990); and (4)
the prescription should be performed at a more moderate intensity and should be applied
more gradually, that is, the progression of the exercise should be slower to allow more
gradual adaptation to the training program (Pollock, & Wilmore, 1990; Wheat, 1987).

The components of a training program for the elderly are similar to those
recommended for young and middle-aged adult (Pollock et al., 1994). The program
should include warm-up, muscular conditioning, aerobics or endurance phase,
recreational activities, and cool-down periods. However, it is recommended that the total
program should not last more than 1 hour (Pollock et al., 1994; Pollock, 1988) inasmuch
as program of longer duration are associated with lower rates of adherence. The warm-up
period, given that in the elderly participants steady state levels of ventilation, BP, and HR
are reached at a slower rate in older adults, more emphasis should be placed on the warm-
up periods, which might last from 10 to 15 minutes (Smith, & Kampine, 1984). It is
recommended that stretching, low-level calisthenics, and low-level aerobic activity, such
as slow walking, cycling, or swimming, should be included in the warm-up. The cool-
down period: Similar to the warm-up, the cool-down should also include low-level
aerobic activities, such as slower walking and cycling, and stretching activities, to be
conducted for 10 to 15 minutes. Continuation of these low-level activities after higher
intensity training can act to maintain venous return despite peripheral dilation (blood
pooling). Emphasis on proper cool-down can also alleviate potential problems associated
with postexercise increases in catecholamines, hypotension, and heat dissipation
(Robbins, & Rubenstein, 1984). Cardiovascular fitness or endurance training: Older
adults should accumulate at least 30 minutes of moderate-intensity exercise on most and preferably all days of the week (ACSM, 2000). This can be accomplished with activities such as brisk walking, gardening, yard work, housework, climbing stairs, and active recreational pursuits. For older adults with higher fitness levels, longer duration and/or higher-intensity exercise may provide additional benefits. The muscular fitness phase: Resistance exercise (RE) training is essential for proper musculoskeletal development and maintenance and improves physical functional capacity and quality of life (Evans, 1999; Carpenter, & Nelson, 1999), especially in the elderly or more frail low fit individuals (Fiatarone et al., 1994). Similar to cardiorespiratory fitness, individualization of the RE training prescription is essential and should be based on the health and/or fitness status and specific goals of the participants. Therefore, due to the natural course of physiological degradation, elderly adults may be more fragile and thus more susceptible to fatigue, orthopedic injuries, and CV complications, and these factors need to be taken into consideration when prescribing RE training program (ACSM, 1998; Fletcher et al., 1995; Pollock et al., 2000, 1994, 1991). In addition, older adults are generally more sedentary as a group and by lowering the intensity of the activity and progressing more slowly than programs prescribed for younger adults may be more beneficial over the long term. It is recommended to perform at least 1 set of 8 to 10 exercises that use all major muscle groups. Each set should involve 10 to 15 repetitions that elicit a perceived exertion rating of 12 to 13 (somewhat hard). Resistance training should be performed at least twice a week, with at least 48 hours of rest between sessions. The muscular conditioning period should include a higher-level calisthenics and/or resistance training lasting a minimum of 15 to 20 minutes. The mode of exercise is also an important
consideration when designing RE programs for the elderly. From a safety standpoint, variable RE training with selectorized weight stacks are generally recommended for several reasons (Pollock et al., 1994; Perkins, & Kaiser, 1961; Wescott, 1987): (a) the initial weight can be applied at a low level and increased in small increments; (b) the equipment is usually designed to protect the lower back, thus reducing the risk of injury; (c) many machines are designed to avoid handgripping which reduces the risk of exercise-induced hypertension; (d) the machines are usually designed to allow the resistance to be applied evenly through the participant’s full range of motion; (e) many types of equipments can be double pinned to allow the subject to exercise through their pain-free ROM; and (f) many resistance machines do not require the participant to balance or control the weight, as do dumbbells and barbells, which may reduce the likelihood of injury (Pollock et al., 1994).

Even though RE training may be appropriate for elderly participants, the prescription should call for moderation. Although the purpose of the muscular conditioning period is strength development, the maintenance of functional capacity, fat-free weight, and bone mineral density are equally important.

When initiating a RE training program, start with the lightest weight on the stack. If one repetition maximum (1-RM) test or maximum torque type of test is administered, then use approximately 30% to 40% of maximum as the starting weight. If the participant can comfortably lift up to 15 repetitions and perceive it to be light (<12 on the rating of perceived exertion [RPE] 15-point category scale), add 5% the next time. The goal will be to complete 10 to 15 repetitions at a comfortably hard level (RPE = 14 to 16). If a participant cannot complete 10 repetitions, decrease the weight.
Intensity should progress slowly, allowing time for adaptation. Participants should not hold their breath when lifting and should usually exhale during the difficult part of the lift. It is important to use good form and go through the ROM in a slow, controlled fashion, usually taking 2 seconds to lift the weight and 4 seconds to let it down.
CONCLUSIONS

Designing investigations to examine age-related changes in the structure and function of the CV system is a formidable research challenge. The impact of other factors co-existing with age on CV function is but an example of methodologic problems that contribute to equivocal interpretations of the real effect of aging on the CV system. Therefore it is important to continue to investigate CV function in older adults and the influence of physical activity on the CV aging process. Such work is not meant to supplant previous investigations, but to reconcile disparate findings so that patterns of age-related effects, and adaptations to lifestyle or other interventions on CV function might start to emerge.

More than half of all deaths in older adults are due to CV disease. Physical inactivity is known to be a major risk factor for the development and progression of CV diseases in older adults, and it is also associated to physical functional impairment in older adults. Moreover, the aging process is associated to serious muscle atrophy and marked declines in physiological function, which might accelerate the possibilities for decline in physical function. On the other hand, data now indicate that normal physical activity and exercise (e.g., strength training) have positive effects reversing and attenuating the physical functional decline with age and associated factors. That means that physical activity and exercise might reduce the relative risk of CV disease that specific factors might have such as physical inactivity, and sedentary life.

Therefore, there has been a heightened interest in identifying physical activity interventions appropriate for optimizing health and reducing health care cost in older adults, with an important but not exclusive focus on reducing the risk of CV disease. To this point, there appears to be agreement that a well-rounded program including aerobic
endurance exercise and RE in fitness or health training program is beneficial for most older adults. Only recently, however, has support for an RE component emerged overcoming earlier concerns that RE in older adults might result in exaggerated pressor response. Therefore, the current ACSM, AHA, the Surgeon General Report, and other health organization have included RE in their position stand and physical activity recommendations for older adults.

Aside of the improvement and maintenance of musculoskeletal fitness, RE also appears to provide CV adaptations in healthy older adults including either no change or a reduced BP response to a given absolute workload and improvements in CV endurance performance. Similar findings are reported performing activities resembling activities of daily life (e.g. loaded-walking). Therefore, at a minimum, short-term RE training does not appear to have any adverse effects on CV structure and function at rest and during exercise. However, due to reduced number (mostly in men and in certain age) of short-term studies and the lack of long-term studies assessing the CV adaptations to RE, more research is needed before any general interpretation regarding this issue can be made. It is of particular interest to examine the effect of RE alone and in combination with aerobic training on systolic and diastolic function of the heart at rest and during physical activity. Additionally, future studies should also examine the effect of RE training on the hemodynamic recovery pattern immediately after exercise in older adults. This issue has not been addressed, but may be of particular importance as many adverse CV responses to exercise are manifest during the recovery period.
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