

## ABSTRACT

SMELKER, C.L. Intensity thresholds for post exercise hypotension. MS in Adult Fitness/Cardiac Rehabilitation, December 2002, 48pp. (C. Foster)

Hypertension is a risk factor for heart disease that responds to both acute and chronic exercise. This study evaluates the effects of different exercise intensities on post exercise hypotension in hypertensive subjects. Subjects (N = 10) were healthy, mildly hypertensive individuals. Each performed a maximal test to allow for intensity prescription, then completed four randomly ordered 120min exercise trials and one control trial. The exercise trials consisted of 25 min cycling at 70, 80, 90, or 100% of the  $\text{VO}_2$  at the ventilatory threshold (VT) (e.g. quite easy to rather hard). Blood pressure was measured at 0, 30, 60, 90 and 120 minutes of each trial. There was a significant ( $p \leq 0.05$ ) reduction in systolic blood pressure at 30, 60, 90 and 120 min for 100%VT, at 60, 90 and 120 min for 90%VT, at 90 and 120 min for 80%VT and at 120 min for 70%VT. There was no change with diastolic blood pressure. Mean arterial pressure was significantly reduced at 60, 90 and 120 min for 90%VT and at 60 and 90 min of 100%VT. We conclude that exercise acutely lowers blood pressure in mildly hypertensive individuals and that a higher intensity exercise session may have a greater effect.

INTENSITY THRESHOLDS FOR POST EXERCISE HYPOTENSION

A MANUSCRIPT STYLE THESIS PRESENTED

TO

THE GRADUATE FACULTY

UNIVERSITY OF WISCONSIN-LA CROSSE

IN PARTIAL FULFILLMENT

OF THE REQUIREMENTS FOR THE

MASTER OF SCIENCE DEGREE

BY

CHRISTY L. SMELKER

DECEMBER 2002

COLLEGE OF HEALTH, PHYSICAL EDUCATION, RECREATION, AND  
TEACHER EDUCATION

UNIVERSITY OF WISCONSIN-LA CROSSE

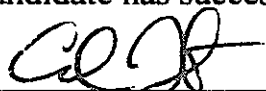
THESIS FINAL ORAL DEFENSE FORM

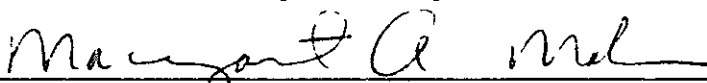
Candidate: Christy L. Smelker

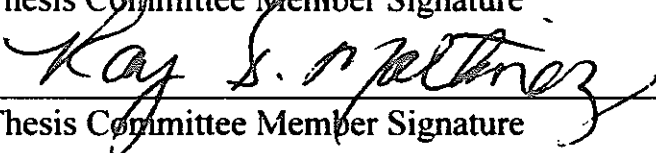
We recommend acceptance of this thesis in partial fulfillment of this candidate's requirements for the degree:

Master of Science in Adult Fitness/Cardiac Rehabilitation

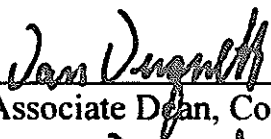
The candidate has successfully completed the thesis final oral defense.

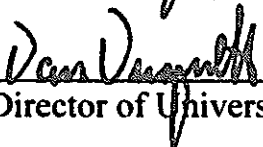
 5/15/02  
Thesis Committee Chairpersons Signature Date

 5/15/02  
Thesis Committee Member Signature Date

 5/15/02  
Thesis Committee Member Signature Date

This thesis is approved by the College of Health, Physical Education, Recreation, and Teacher Education.

 9/14/02  
Associate Dean, College of HPERTE Date

 9/14/02  
Director of University Graduate Studies Date

## ACKNOWLEDGEMENTS

I would like to express my deepest appreciation to the following individuals for aiding me in the completion of this research project:

To Dr. Carl Foster, thank you for your time, patience and efforts. Your guidance was instrumental in the success of this project.

To Dr. John Porcari, thank you for the time spent working on my thesis statistics. I could not have done it without you

To Dr. Peg Maher and Dr. Ray Martinez, thank you for participating on my thesis committee and for the many hours spent reading and correcting my thesis.

To my family and Kevin Ruskusky, thank you for always having confidence in me, supporting my efforts and encouraging me to never give up. Thank you for everything you have done.

## TABLE OF CONTENTS

	PAGE
ACKNOWLEDGEMENTS.....	iii
LIST OF FIGURES.....	v
LIST OF APPENDICES.....	vi
INTRODUCTION.....	1
METHODS.....	3
Subjects.....	3
Protocol.....	4
Statistical Treatment.....	5
RESULTS.....	6
DISCUSSION.....	12
REFERENCES.....	17
APPENDICES.....	20

## LIST OF FIGURES

FIGURE	PAGE
1. Control Corrected Changes in Systolic Blood Pressure. . . . .	8
2. Control Corrected Changes in Diastolic Blood Pressure. . . . .	9
3. Control Corrected Changes in Mean Arterial Pressure. . . . .	10
4. Time versus Estimated Systemic Vascular Resistance. . . . .	11

## LIST OF APPENDICES

APPENDIX	PAGE
A. Informed Consent. ....	21
B. Review of Literature. ....	24

## INTRODUCTION

According to the National Institutes of Health, hypertension plays a part in approximately 700,000 deaths a year, from stroke, heart disease and kidney disease. Fifty million Americans have hypertension, as defined by systolic blood pressure (SBP) > 140 (mm Hg) and diastolic blood pressure (DBP) > 90 (mm Hg), of which approximately 30% are undiagnosed cases (1, 2). Ninety to ninety-five percent of hypertension cases are classified as "essential hypertension," which is also known as primary or idiopathic hypertension because the cause is unknown (3, 4).

Left untreated hypertension can cause a cascade of problems. Once it has developed, it can last a lifetime. Many of the outcomes of hypertension, such as a heart attack or stroke, occur before it is discovered. Hypertension increases the workload on the heart requiring it to need more oxygen to sustain normal function. It also increases the sheer stress on the endothelial lining of the vessels leading to initiation of the atherosclerotic process (5). It greatly increases the risk of heart disease and stroke, the first and third leading causes of death in the United States (US). Cardiovascular disease is estimated to take nearly one million lives each year in the US, ending one life every 33 seconds (3). Uncontrolled hypertension is also a leading cause of heart failure, kidney disease, and blindness. The risk of developing hypertension is thirty to fifty percent greater in those who are less active and less fit. Aging also increases the risk of hypertension. In general, sixty percent of Americans ages sixty and older have hypertension (3).

Current research suggests that lifestyle modifications, such as losing weight, increasing physical activity, reducing alcohol consumption and modifying the diet by reducing sodium intake and increasing potassium and calcium intakes, can play an essential role in controlling hypertension. Achievable goals with these approaches include enabling some patients to decrease the amount of medication needed or avoid drug therapy altogether. The side effects of these non-drug therapies are very beneficial, including lowered risk of stroke, cardiovascular and kidney disease, prevention of some cancers and increased health (5).

Regular physical activity is one of the most effective lifestyle modifications for preventing and/or controlling hypertension. According to the National Institutes of Health and the Working Group Report on Primary Prevention of Hypertension, the mechanism for a post exercise-induced reduction of blood pressure is uncertain but may be related to a decrease in cardiac output or peripheral vascular resistance (2). Exercise may lower blood pressure through a variety of other mechanisms, involving effects on the sympathetic nervous system, insulin sensitivity, electrolyte balance, neural and baroreceptor reflex mechanism, and vascular structure (6). Evidence from both observational and experimental studies supports the value of low to moderate intensity physical activity of as an intervention for the primary prevention of hypertension.

This study evaluates the effects of different exercise training intensities on post exercise hypotension (PEH). PEH is the reduction of arterial blood pressure after a single session of exercise. PEH has been observed following dynamic exercise performed at intensities between 40 – 70% of  $\text{VO}_2$  peak (7 – 19). Although mechanistic studies are

lacking, PEH has been attributed to a reduction in vascular resistance. Other possible factors involved in mediating PEH include decreases in stroke volume, cardiac output and sympathetic discharge. The effect of different exercise intensities on the development of PEH is not well documented. Accordingly, the purpose of the present study was to compare the effects of exercise performed at varying intensities in relation to the power output at the ventilatory threshold on the post exercise reduction in blood pressure in mildly hypertensive individuals.

## METHODS

### Subjects

The subjects for this study were 10 volunteers, (2 females, 8 males) from the La Crosse community and the University of Wisconsin-La Crosse Exercise and Health Program. Each participant was apparently healthy and clinically stable. Six were borderline hypertensive, and four had a clinical diagnosis of hypertension and were on medical therapy. None of the subjects had documented coronary artery disease, prior myocardial infarction, symptoms suggestive of angina pectoris or a history of revascularization procedures. The subjects were instructed to take all medications as usual. Prior to the study, all subjects provided informed consent. The study was approved by the University of Wisconsin-La Crosse Institutional Review Board for the Protection of Human Subjects.

Initially each subject performed a maximal  $\text{VO}_2$  test to allow for the subsequent sessions intensity prescription and also for screening for occult coronary artery disease. Following this, each subject was asked to schedule five appointments. Each of these

appointments was 120 minutes in duration. Participants were scheduled at the same time of day for each appointment. Subjects were asked not to consume any food or drink for two hours prior to each testing session, and to refrain from alcohol intake 24 hours prior to each testing session. None of the subjects used tobacco. Characteristics of the study subjects are shown in Table 1.

Table 1. Characteristics of the Study Population. (N = 10)

<u>Characteristic</u>	<u>Value</u>
Age (years)	51 ± 11
Weight (kg)	86 ± 20
Height (cm)	172 ± 9
Average resting SBP (mm Hg)	134 ± 12
Average resting DBP (mm Hg)	82 ± 6
<u>Average resting MAP (mm Hg)</u>	<u>99 ± 7</u>

Data is expressed as mean ± standard deviation.

### Protocol

All studies were conducted in the Human Performance Laboratory at the University of Wisconsin-La Crosse. The researcher measured all blood pressures by auscultatory method with the subject seated and their arm supported and elevated at heart level. Each subject was studied during four randomly ordered exercise sessions, performed at least 48 hours apart. The subject also participated in a control trial consisting of 120 minutes of continuous seated rest with blood pressure and heart rate values obtained at 0, 30, 60, 90, and 120 minutes.

The four randomly ordered exercise trials consisted of aerobic exercise on a cycle ergometer at an intensity level of 70, 80, 90, or 100% at the  $\text{VO}_2$  of the ventilatory threshold (VT) (e.g., ranging from quite easy to rather hard). Individual power output requiring these values for  $\text{VO}_2$  were derived using the American College of Sports Medicine cycle ergometer equations (9). Blood pressure (BP) and heart rate (HR) were measured following five minutes of seated rest after the subject arrived in the laboratory. This initial BP was recorded as the 0 minute value for the trial. Immediately after the initial BP was obtained, the subject exercised for 25 minutes. HR and rating of perceived exertion (RPE) were monitored during exercise to document exercise intensity along with exercising BP measured at 10, 20 and 25 minutes. The Category Ratio Rating of Perceived Exertion scale (e.g., 0 – 10) was used to measure RPE (20). At the conclusion of the 25 minute exercise session, a five minute active cool-down was allowed, after which a resting seated BP was taken. This BP served as the 30 minute measurement. Additional resting BP was measured at 60, 90, and 120 minutes.

Systemic vascular resistance (SVR) was estimated according to Grossman and Barry,  $[\text{SVR} = 80(\text{Mean Arterial Pressure} - 5)/\text{Cardiac output}]$  (21). It was assumed that at rest, cardiac output is equal to  $[(\text{VO}_2 \text{ (L/min)} * 5) + 5]$  (21). Due to the 30 minute BP measurement being only 5 minutes post-exercise we felt that the assumption of a resting cardiac output was not justified. Accordingly, only the 0, 60, 90, and 120 minute time periods could be used during recovery to analyze estimated SVR. Mean Arterial Pressure (MAP) was calculated as  $1/3(\text{SBP} - \text{DBP}) + \text{DBP}$ .

### Statistical Treatment

Statistical analysis was performed using repeated measures ANOVA to examine the main effects of exercise intensity on SBP, DBP, MAP, HR, and SVR from rest to 30, 60, 90, and 120 minutes (5, 30, 60, and 90 minutes post exercise). Alpha was set at  $\leq 0.05$  to achieve statistical significance. Tukey's post hoc tests were used to evaluate pairwise differences when main effects were significant by ANOVA.

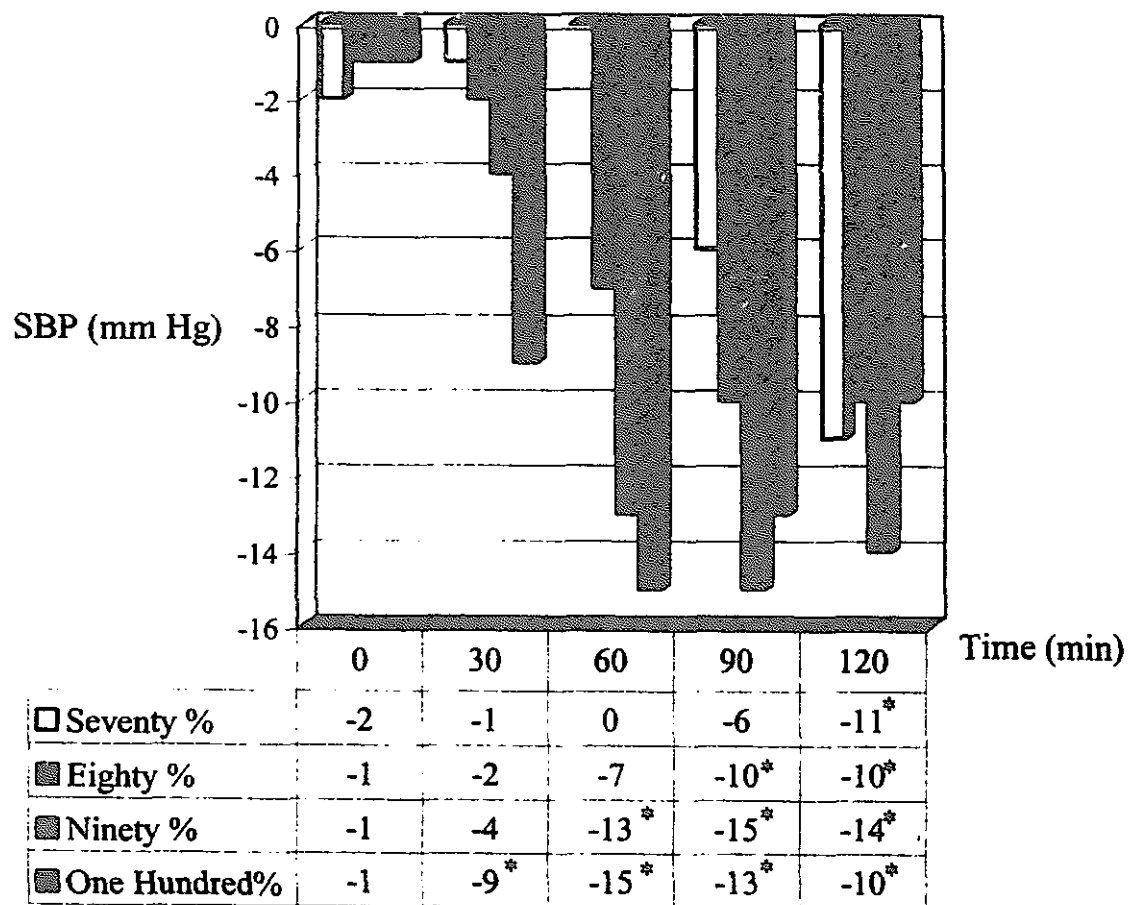
### RESULTS

For all four interventions (70, 80, 90, and 100% of the  $VO_2$  at the ventilatory threshold) there was a significant decrease in SBP following exercise. At 70% VT, there was a significant decrease in SBP from rest at the 120 minute time point. At 80% VT, there was a significant decrease in SBP from rest at the 90 and 120 minute time points. At 90% VT, there was a significant decrease in SBP from rest at the 60, 90, and 120 minute time points. At 100% VT, there was a significant decrease in SBP from rest at the 30, 60, 90, and 120 minute time points (Figure 1). There was no significant change in DBP from rest with any of the interventions (Figure 2).

MAP was significantly decreased at 90% VT at the 60, 90, and 120 minute time points. At 100% VT, there was a significant decrease in MAP from rest at the 60 and 90 minute time points (Figure 3).

Estimated SVR was significantly decreased from control at 90% VT at the 60, 90, and 120 minute time points. At 100% VT, there was a significant decrease in estimated SVR from rest at the 90 minute time point (Figure 4).

Subjective exercise intensity was rated on the RPE scale as follows: 70% VT =  $3 \pm 1$ , 80% VT =  $3.5 \pm .5$ , 90% VT =  $4 \pm 1$  and 100% VT =  $5 \pm 1$ . These correspond to between moderate to hard on the RPE scale.



\* = Significantly different than control,  $p < 0.05$

Figure 1. Control Corrected Changes in Systolic Blood Pressure

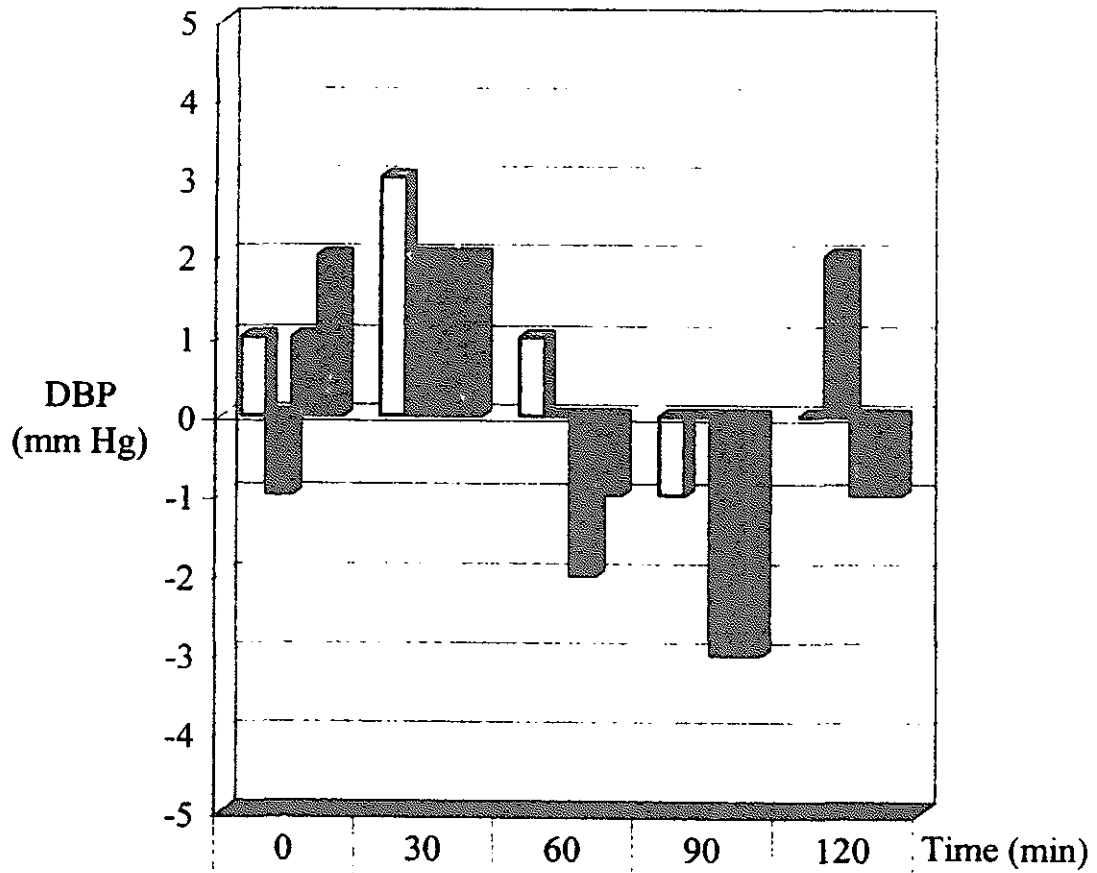


Figure 2. Control Corrected Changes in Diastolic Blood Pressure

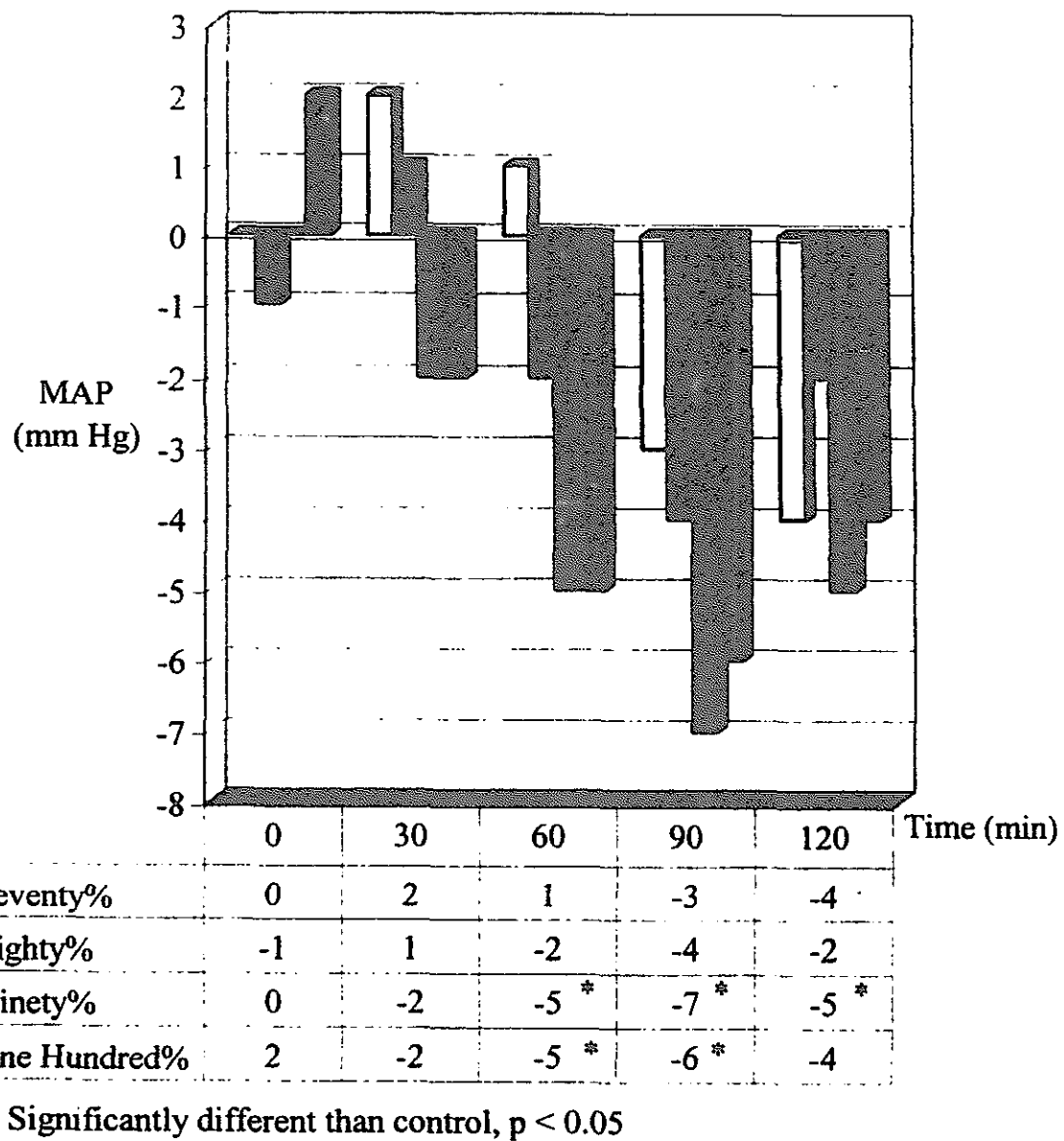
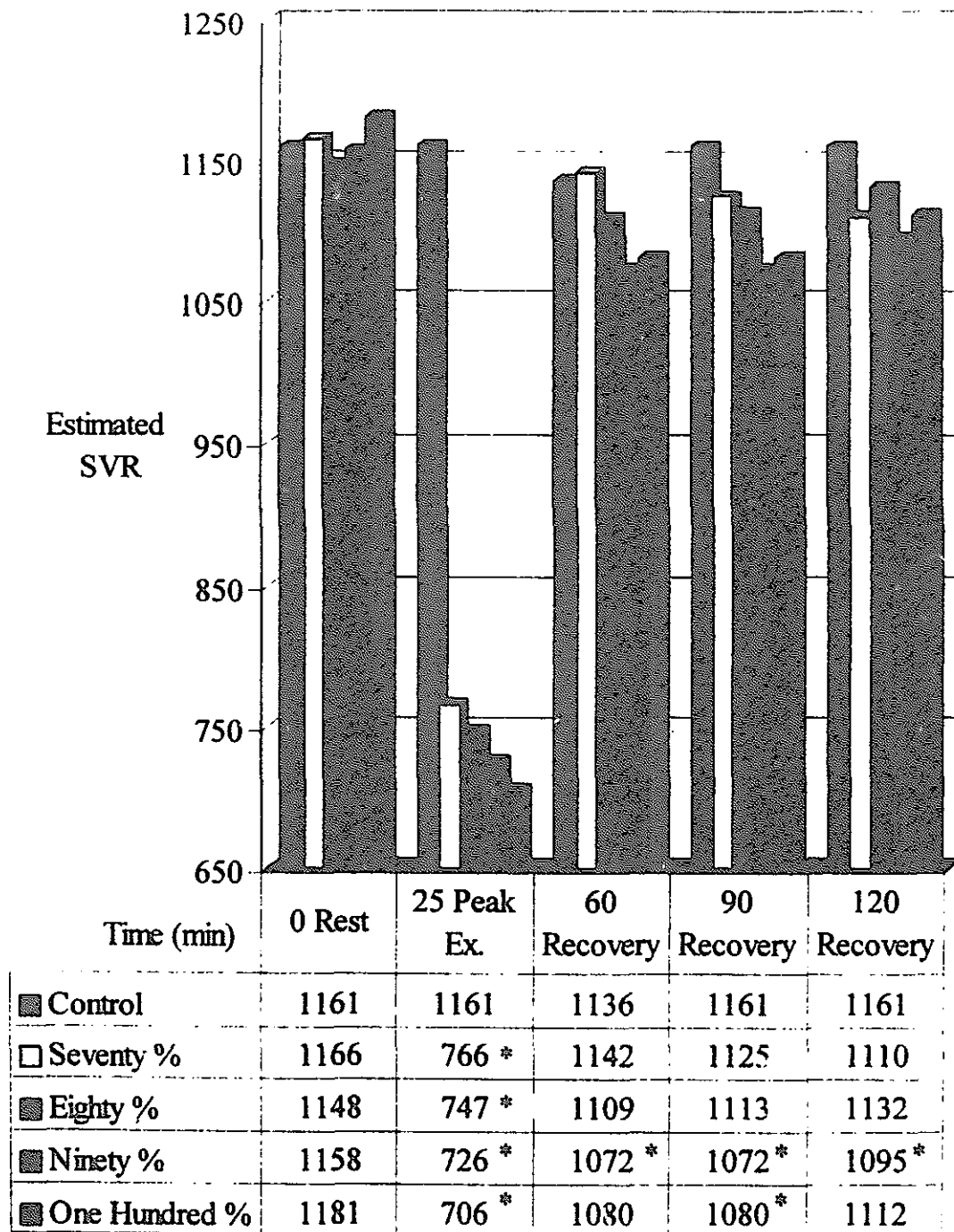


Figure 3. Control Corrected Changes in Mean Arterial Pressure



\* = Significantly different than control,  $p < 0.05$

Figure 4. Time versus Estimated Systemic Vascular Resistance

## DISCUSSION

PEH was observed at all intensity levels in our study. There were differences between intensities relative to the time of onset of PEH, the magnitude of PEH and the duration of PEH. The most dramatic reduction in SBP was found following the higher intensity bouts and persisted for a longer duration during the post exercise recovery. The largest differences were observed at 100% VT with a significant reduction in SBP at 30, 60, 90, and 120 minutes post exercise. SBP was lower for the entire recovery period with the greatest reduction, -15 mm Hg, at 30 minutes post exercise (60 minute time period). Subsequent measurements at 60 and 90 minutes post-exercise (90 and 120 minute time periods) showed a gradual return to resting values, but SBP were still -10 mm Hg lower than control at the 120 min time period.

At 90% VT, SBP was not significantly reduced until 30 minutes post-exercise (60 minute time period), then stayed consistently lower for the remainder of the recovery period (60 – 120 minute time period). During 80% VT, there was a significant decrease in SBP at 60 minutes post-exercise (90 minute time period). SBP remained consistently lower for the remainder of the recovery period (90 & 120 minute time periods). With 70% VT there was not a significant decrease in SBP until 90 minutes post-exercise (120 minute time period).

These data are consistent with the findings of Hagberg et al., who found that at higher exercise intensities (70% compared to 50% of  $\text{VO}_2$  max) there was a greater decrease in SBP (-13 mm Hg compared to -8 mm Hg) for a longer period of time (three hours compared to one hour) (15). Different from the present results, Hagberg et al.

found that at 50% VO<sub>2</sub> max, SBP was significantly lower (-8 mm Hg) immediately post exercise and remained lower for a duration of an hour. We observed a -14 mm Hg decrease at 90% VT which corresponded to 52% VO<sub>2</sub> max. In our data the onset of PEH occurred at 30 minutes post-exercise and remained lower for the duration of recovery. At 70% VT (40% VO<sub>2</sub> max) there was no reduction in PEH until the 120 minute time period (90 minute post-exercise).

Our data evaluates cuff BP measured in the laboratory over a two hour period. Several studies have evaluated PEH with ambulatory BP monitoring and have found no difference in the magnitude of PEH in relation to exercise intensity (12, 22). Marceau et al. found that 10 weeks of closely supervised exercise training at 50% and 70% of maximal oxygen uptake induced comparable decreases in average 24-hour blood pressure (22). Pescatello et al. using ambulatory BP monitoring, found that SBP was reduced for 8 hours following exercise, independent of exercise intensity (40% compared to 70% VO<sub>2</sub> max) (12). The results from these studies suggest that the effects of PEH may be sustained. However, it is fair to point out that at the 120 minute observation in the present data, the magnitude of PEH was similar in all four exercise conditions (-10 to -14 mm Hg relative to control). To the degree that our single late observation is measuring the same hemodynamic consequences in Pescatello's study it may be that exercise intensity is less important to long term PEH.

Differences in findings between the present and previous studies can be accounted for by differences in methodology. Marceau et al. compared average 24-hour BP (22). Pescatello et al. compared average BP collected over 13 hours (12). Both of these studies

measured BP over a longer period of time by auscultation and also by ambulatory BP monitoring, whereas the present data were relatively acute (5 to 90 minutes post-exercise). It is not clear what accounts for the inconsistent hemodynamic changes observed after a single bout of exercise. It is clear that additional data are needed to define more completely the nature of systemic and regional hemodynamic alterations mediating PEH, as well as the role of differing methodologies in determining the nature of these responses (23, 24)

Contrary to the previous studies, Gilders et al. found that after 16 weeks of exercise conditioning (e.g. chronic rather than acute exercise) there was no change in 24-hour ambulatory BP or in casual BP measurements (19). They also found no changes in cardiac output or total SVR (19). The divergent responses in their study may be accounted for by differences in methodology and control of BP measurements. Gilders et al. measured ambulatory BP on the days when no exercise was performed, which could account for the lack of PEH (19). On the other hand, Pescatello et al. measured ambulatory BP on days where subjects exercised (12).

The majority of previous studies reported a significant reduction in DBP (11 – 13, 22, 24) whereas, we did not. There is some evidence suggesting that normotensive individuals have a more pronounced and prolonged DBP response to acute exercise (24). Our findings were consistent with others who also found no change in DBP (15).

In our study, MAP was significantly reduced for the longest duration at 90% VT, first evident at 30 minutes post-exercise and continued throughout the recovery period (60 – 120 minute time periods). The greatest reduction, -7 mm Hg, occurred at 60

minutes post exercise (90 minute time period). MAP was also reduced significantly at 100% VT at 30 and 60 minutes post-exercise (60 and 90 minute time periods). There was no reduction in MAP at 70% and 80% VT. The MAP response, again demonstrates that higher intensity exercise has a more dramatic effect on reducing BP. Consistent with these observations, Paulev et al. reported MAP to be reduced an average of -8 mm Hg for at least 4 hours after submaximal cycling exercise in borderline hypertensive humans (25).

The estimated SVR was found to be significantly reduced the most during 90% VT at 30 minutes post-exercise with the effects lasting through recovery (60 – 120 minute time periods). At 100% VT there was a significant reduction at 60 minute post-exercise (90 minute time period).

A reduction in BP is attributable to a reduction in cardiac output or SVR, or both. A cumulative reduction in SVR is proposed to result from acute bouts of exercise, which decrease total SVR. Rueckert et al. found that immediately after exercise vascular resistance was significantly lower than pre-exercise (26). As SVR gradually normalized they then found a significant decrease in cardiac output, which contributed to the decline in blood pressure (24). The early decrease in SVR may be due to a number of factors including the effect of exercise-induced metabolite formation, flow-mediated vasodilation, reduced alpha-adrenergic responsiveness and thermodilation (26). Moreover, Floras recently reported reductions in SBP, DBP, MAP, calf vascular resistance, and SVR, and tachycardia-mediated increases in cardiac output after submaximal treadmill exercise (27). Sustained decreases in limb (forearm and calf)

vascular resistance and SVR have been reported more consistently after exercise in both normotensive and hypertensive subjects (23). These observations suggest that a sustained vasodilation in skeletal muscle and other arterial beds may contribute to PEH (23).

The factors that cause a decline in SVR could account for the greater reduction in PEH that was found at higher intensities in our study. Higher intensities may mediate a greater formation of exercise-induced metabolites and higher flow may mediate a greater vasodilation in the periphery. As this type of vasodilation is time-dependent, there may be a longer period of PEH as the body gradually returns to resting levels after exercise. Rueckert et al. also found that a significantly lower cardiac output contributed to the reduction in blood pressure after exercise (26). They found that a change in HR was the primary determinant of changes in post-exercise cardiac output. Early after exercise, cardiac output was maintained by an increased HR, but from 50 minutes to two hours the increase in HR was not sufficient to prevent a significant decline in cardiac output (26).

In the present study, although cardiac output was not directly measured, HR was found to be elevated immediately post-exercise and gradually returned to resting levels, or below, by 60 minutes post-exercise (90 minute time period). This could account for the significant reduction in SBP (-11 mm Hg) that occurred 90 minutes post-exercise (120 minute time period) at 70%VT.

The magnitude of the PEH response observed in mildly hypertensive subjects in our study is significant and should likely be considered clinically important. However, more investigation is required to determine if the intensity of the exercise session and

duration of the response is sufficient in real-life conditions to contribute to the reduction in arterial blood pressure observed after chronic exercise.

## REFERENCES

1. Joint National Committee on Detection, Evaluation, and Treatment of Blood Pressure (1997). The Sixth Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. *Arch. Intern. Med.* 157:2413-2446.
2. Guyton AC (1991). *Textbook of Medical Physiology*. (Eighth Edition). Philadelphia, PA: Saunders.
3. American Heart Association (1998). Heart and stroke statistical updates. Dallas, TX: American Heart Association.
4. National High Blood Pressure Education Program (1993). Working group report on primary prevention of hypertension. National Institutes of Health. U.S. Department of Health and Human Services. NIH Publication No. 93-2669.
5. Hagberg JM (1997). Physical activity, physical fitness, and blood pressure. *Human Kinetics*. 112-119.
6. Schuster-Decker R, Foster C, Porcori JP, Maher MA (2002). Acute effects of dynamic exercise and nutritional supplementation on blood pressure in mildly hypertensive patients. *Clin. Exerc. Physiol.* 4:17-21.
7. Rowland TW (1998). The biological basis of physical activity. *Med. Sci. Sports Exerc.* 30:392-399.
8. Grimm RH, Grandits GA, Culter JA, et al (1997). Relationships of quality-of-life measures to long-term lifestyle and drug treatment in the treatment of mild hypertension study. *Arch. Intern. Med.* 157:638-664.
9. American College of Sports Medicine (2000). *Guidelines for Exercise Testing and Prescription*. (Sixth Edition). Baltimore, MD: Lippincott Williams & Wilkins.
10. Halliwill JR (2001). Mechanisms and clinical implications of post-exercise hypotension in humans. *Exerc. Sports Sci. Rev.* 29:65-70.
11. MacDonald JR, MacDougall JD, Hogben CD (1999). The effects of exercise duration on post exercise hypotension. *J. Human Hypertens.* 14:125-129.

12. Pescatello LS, Fargo AE, Leach CN, Scherzer HH (1991). Short-term effect of dynamic exercise on arterial blood pressure. *Circulation*. 470:231-241.
13. Bennet T, Wilcox RG, MacDonald IA (1984). Post-exercise reduction of blood pressure in hypertensive men is not due to acute impairment of baroreflex function. *Clin. Sci.* 67:97-103.
14. Rowell LB (1986). Human circulation regulation during physical stress. London, England. *Oxford University Press*.
15. Hagberg JM, Montain SJ, Martin WH (1987). Blood pressure and hemodynamic responses after exercise in older hypertensives. *J. Appl. Physiol.* 63:270-276.
16. MacDonald JR, MacDougall JD, Interisano SA, Smith KM, Youngai EV, Tarnopolsky MA (1998). Hypotension following mild bouts of resistance exercise and submaximal dynamic exercise. *Eur. J. Appl. Physiol.* 79:148-154.
17. MacDonald JR, Hogben CD, Tarnopolsky MA, MacDougall (2001). Post exercise hypotension is sustained during subsequent bouts of mild exercise and simulated activities of daily living. *J. Human Hypertens.* 15:567-571.
18. Kaufman FL, Hughson RL, Schaman JP (1987). Effect of exercise on recovery blood pressure in normotensive and hypertensive subjects. *Med. Sci. Sports Exerc.* 19:17-20.
19. Gilders RM, Voner C, Dudley GA (1989). Endurance training and blood pressure in normotensive and hypertensive adults. *Med. Sci. Sports Exerc.* 21:629-636.
20. Borg G, Hassmen P, Langerstrom M (1985). Perceived exertion in relation to heart rate and blood lactate during arm and leg exercise. *Eur. J. Appl. Physiol.* 65:679-685.
21. Grossman W, Barry WH (1988). Cardiac Catheterization. In: Heart Disease: Third Edition Braunwald E, ed. *A Textbook of Cardiovascular Medicine*, pp. 242-267. Philadelphia, PA: WB Saunders Company.
22. Marceau M, Kouame N, Lacourciere Y, Cleroux J (1993). Effects of different training intensities on 24-hour blood pressure in hypertensive subjects. *Circulation*. 88:2803-2811.
23. Kenney MJ, Seals DR (1993). Postexercise hypotension: Key features, mechanisms, and clinical significance. *Hypertension*. 22:653-664.

24. MacDonald JR, MacDougall JD, Hogben CD (1999). The effects of exercise intensity on post exercise hypotension. *J. Human Hypertens.* 13:527-531.
25. Paulev P-E, Jordal R, Kristensen O, Ladeforged J (1984). Therapeutic effect of exercise on hypertension. *Eur. J. Appl. Physiol.* 53:180-185.
26. Rueckert PA, Slane PR, Lillis DL, Hanson P (1996). Hemodynamic patterns and duration of post-dynamic exercise hypotension in hypertensive humans. *Med. Sci. Sports Exerc.* 28:24-32.
27. Floras JS, Sinkey CA, Aylward PE, Seals DR, Thoren PN, Mark AL (1989). Postexercise hypotension and sympathoinhibition in borderline hypertensive men. *Hypertension.* 14:28-35.

APPENDIX A  
INFORMED CONSENT

## INFORMED CONSENT

### INTENSITY THRESHOLD FOR POST EXERCISE HYPOTENSION

I, \_\_\_\_\_, give my informed consent to participate in this study designed to evaluate different training intensities and their effects on blood pressure following exercise. I consent to the presentation, publication and other release of summary data from the study, which is not identifiable with myself.

I have been informed that this study will consist of five different phases each 120 minutes in duration. One phase will involve sitting at rest. The other four phases will involve exercising for 30 minutes on a cycle ergometer with different assigned intensities levels at 70%, 80%, 90% and 100% of the power output at the ventilatory threshold (i.e., the point where talking becomes uncomfortable) Which ranges from quite easy to rather hard. I have been informed that my blood pressure will be monitored at thirty minutes intervals throughout each testing session, at 0, 30, 60, 90 and 120 minutes. I have been informed that I should not to consume any food or drink (other than water) 2 hours prior to each testing session and should refrain from alcohol intake 24 hours prior to each testing session. I have been informed that I will be required to perform a preliminary maximal test as a screening procedure and to allow prescription of the intensity of the subsequent rides.

I have been informed that during the maximal testing and exercising phases, there are possible risks which include: dizziness, faintness, injury, fatigue, muscle and/or joint pain, and in some rare cases, serious complication. However, the probability of serious complications occurring is very small and has never occurred on the University of Wisconsin-La Crosse campus. I have been informed that the risk of medical complications during exercise testing in individuals thought to have heart disease is approximately 6/10,000 tests. In individuals, like myself, who are healthy, the risk of complications is less well documented but is thought to be very low.

I have been informed that the primary benefits of this study include a maximal exercise test and blood pressure monitoring while exercising. I have been informed that I will gain the benefits of fitness testing and will learn about my own blood pressure response to exercise. No monetary rewards will be given for participation in this study. I have been informed that the results of the study may improve the ability of health care professionals to more effectively treat hypertension.

I have been informed that the investigators will answer questions regarding the procedures throughout the course of the study.

I have been informed that I am free to decline to participate or to withdraw from the study at any time without penalty.

I have been informed that in the unlikely even that any injury occurs as a result of this research, the Board of Regents of the University of Wisconsin System, and the University of Wisconsin-La Crosse, their officers, agents and employees, do not automatically provide reimbursement for medical care or other compensation. My third-party payor, such as my health insurer or Medicare, or I, must provide payment for treatment of any injury. If any injury or illness occurs in the course of research, or for more information, please notify the investigator in charge. I have been informed that I am not waiving any rights that I may have for injury resulting from negligence of any person or the institution.

Concerns about any aspects of this study may be referred to either the Principal Investigator, Christy Smelker (608) 779 – 4856 or the Supervising Faculty Member, Dr. Carl Foster (608) 785 – 8687. Questions regarding the protection of human subjects may be addressed to Dr. Dan Duquette, the Chair of the UW-La Crosse Institutional Review Board for the Protection of Human Subjects, (608) 785 – 8124.

Subject (print name): \_\_\_\_\_ Date: \_\_\_\_\_

Subject Signature: \_\_\_\_\_ Date: \_\_\_\_\_

Investigator: \_\_\_\_\_ Date: \_\_\_\_\_

APPENDIX B  
REVIEW OF LITERATURE

## REVIEW OF LITERATURE

### Hypertension

Blood pressure is a measurement of the force of blood against the walls of arteries and is determined by the amount of blood the heart pumps and the amount of resistance to blood flow in the arteries (1, 2). When blood pressure stays elevated over time it is called hypertension, or high blood pressure. The Sixth Report of the Joint National Committee (JNC VI) on Prevention, Detection, Evaluation and Treatment of High Blood Pressure defined hypertension as a diagnosis of 2 or more diastolic blood pressure (DBP) measurements  $\geq 90$ mm Hg on at least 2 subsequent visits or when the average of multiple systolic blood pressure (SBP) readings on 2 or more subsequent visits is consistently  $\geq 140$  mm Hg (3). With the use of these definitions, it is estimated that fifty million people or one out of four adults, in the United States have hypertension (1, 3 – 7). According to the American Heart Association, high blood pressure alone causes no symptoms, and as a result, nearly one third of individuals with the disorder are unaware that they have it. Of all people who have hypertension, 90 – 95 percent are said to have “essential hypertension” (1, 5, 8). Essential, primary or idiopathic hypertension is defined as high blood pressure in which the cause is not known.

### Measuring Blood Pressure

Hypertension detection begins with proper blood pressure measurements. Patients should be seated for at least five minutes and have their arms bared and supported at heart level. An appropriate cuff size should be used. Measurements should be taken by auscultation with a mercury sphygmomanometer and both SBP and DBP should be recorded. The cuff is inflated above arterial blood pressure, enough to allow for closure of the brachial artery ( $\geq 200$  mm Hg) and then the pressure is released at approximately 2 mm Hg/sec to allow blood to begin to flow back through the artery. The sounds that are heard are Korotkoff sounds. The first Korotkoff sound to be heard is SBP and the disappearance of sound or the fifth Korotkoff sound is DBP. Two or more readings separated by 2 minutes should be averaged. If the readings differ by more than 5 mm Hg, additional readings should be obtained and averaged (3, 9).

### Risk Factors

High blood pressure is dangerous because it forces the heart to work harder and also can cause the walls of the arteries to become thickened. Hypertension increases the risk for heart disease and stroke, which are the first and third leading causes of death, respectively, in the United States. Hypertension is the primary risk factor for stroke and it is a risk factor for coronary heart disease (6). According to the American Heart Association, patients with hypertension are seven times more likely to have a stroke, three times more likely to develop coronary artery disease, and six times more likely to develop congestive heart failure than normotensive individuals. Hypertension also

contributes to the development of other problems, such as kidney disease, blindness, and other debilitating conditions (5).

### Uncontrollable Risk Factors

Non modifiable or uncontrollable risk factors are heredity, age, gender, and ethnicity. Blood pressure clearly has a genetic basis with a tendency to run in families, with heritability estimates ranging from 25 to 65 percent (10).

As the population ages, hypertension becomes more prevalent and increasingly more difficult to treat. The American Heart Association reports that hypertension occurs most often in people over the age of thirty-five (5). Although findings on the influence of age on the magnitude of blood pressure reduction after exercise are conflicting, it is clear that hypertensive patients of advanced age can tolerate exercise well and benefit from a properly designed exercise program (11). Hypertension is less prevalent in premenopausal women than in men of comparable age, but women develop hypertension at a greater rate after menopause. By the age of seventy-five, nearly three in four American women develop hypertension (5). Some studies have indicated, though, that women obtain more benefits than men with exercise training (10)

The prevalence of hypertension differs among ethnic groups compared with the general population (3). African-Americans develop high blood pressure more often than whites and have the highest prevalence of hypertension in the world (3). The American Heart Association, reports that one in three African-Americans have hypertension and that it also tends to occur earlier and to be more severe than in whites (5). Minimal information indicates that with exercise African-Americans blood pressures are also

reduced (10). Ethnicity affects the blood pressure-lowering effects of exercise training, as Asian/Pacific Island patients can reduce their blood pressure more and also more consistently than Caucasian patients (10).

### Controllable Risk Factors

The major modifiable or controllable risk factors for hypertension are smoking, eating too much salt, heavy and regular use of alcohol, inactivity, obesity, and stress. For those who have salt sensitive essential hypertension, high salt intake significantly exacerbates their hypertension (1). Heavy and regular use of alcohol elevates blood pressure both acutely and chronically. The mechanism by which alcohol raises blood pressure is unknown (12). There is a greater tendency of obesity (a body mass index of  $\geq 30$ ) in inactive individuals and there is a higher prevalence of hypertension in the obese and inactive. Stress is another risk factor for hypertension. It fluctuates widely from person to person and is difficult to measure (1).

### Interventions

There are four main therapies for treating hypertension, including medication, losing weight, exercise training, and reduction in dietary sodium intake. There is clear evidence that changes in these factors, hence changes in lifestyle, can reduce or normalize BP in many patients. Although sustained modifications in diet and lifestyle are difficult to achieve they may lower blood pressure and obviate or lower the need for drug therapy. Unfortunately, most patients with established hypertension do not make sufficient lifestyle changes, do not take medication, or do not take enough medication to achieve control (1).

### Medication Therapy

Patients with severe hypertension have a tendency to require more medication. With the increased use of pharmacological agents there is also an increased incidence of side effects. Medication therapy works in approximately 60 to 70 percent of people and so the fact that people are taking a drug does not necessarily mean it is working (12). There also is a higher rate of non-compliance among patients with severe hypertension along with a decreased quality of life (7). Essential hypertension can be treated by two types of drugs, drugs that increase renal blood flow and drugs that decrease tubular reabsorption of salt and water (1). Common antihypertensive medications are diuretics, beta-blockers, calcium channel blockers, and ACE inhibitors. However, even those who are achieving optimal benefits from medication are still at higher risk of morbidity and mortality than normotensive people with similar levels of blood pressure (13). In addition, hypertensives are burdened with the possibility of adverse effects from their treatment regimens, such as diarrhea, sleep disturbance, depression, dizziness, impotence, and fatigue (10). The implementation of regular exercise as an adjunct to medical therapy can improve blood pressure control at lower doses and reduces adverse effects (7).

### Weight Loss

Weight loss is an important factor in the management and prevention of hypertension as obesity is a major cause of hypertension. The most consistent and significant reductions in blood pressure have been seen with weight loss (15). An appreciable reduction in blood pressure can be seen with weight loss of as little as 10 lb in overweight people with hypertension (3). A recent review found that a weight

reduction of 4 to 8 percent corresponds to a blood pressure decrease of approximately 3 mm Hg systolic and diastolic (16). A recent study directly compared the blood pressure-lowering effect of weight loss and endurance training programs. (10, 17). They compared the effects of twelve weeks of exercise with and without weight loss and weight loss by dietary means, which included restricted dietary sodium intake. All three interventions had the desired overall effects as the exercise training group increased their maximal oxygen uptake by ten percent, decreased body weight by an average of 7 kg and reduced systolic and diastolic blood pressure by 12.5 and 7.9 mm Hg, respectively (17). The effects of exercise training and dietary-induced weight loss do not appear to be additive (7, 10, 11).

### Dietary Modification

The JNC VI report recommends that salt intake be limited to the FDA Daily Value Level (no more than 2400 mg/d) and that moderate sodium reductions be used, along with other lifestyle modifications or as an adjunct to drug therapy, for prevention and treatment of hypertension (3). Although experts continue to disagree on whether decreased salt intake should be recommended for prevention of hypertension, few dispute that sodium reduction independently and significantly lowers blood pressure in hypertensive people while posing minimal risk (18). The established data strongly suggest that sodium reduction remains a valuable component of nondrug therapies.

### Physical Activity

While antihypertensives are clearly the first line of treatment for severe hypertension, physical activity is a nonpharmacological treatment that is often featured

prominently in the treatment of mild to moderate hypertension (6). Physical activity and physical fitness appear to diminish the risk of developing hypertension. The Treatment of Mild Hypertension Study (TOMHS) has shown that lifestyle modifications, including weight loss and increased physical activity, contribute significantly to blood pressure control (19). In addition, the trial reported physical activity improved quality of life and had positive effects on the general well being of the subjects.

Endurance exercise training generally has beneficial effects on other coronary heart disease (CHD) risk factors and results in, on average, a 5 mg/dl increase in HDL cholesterol and an average reduction in total cholesterol levels of 10 mg/dl (6). Also, exercise training beneficially affects other CHD risk factors such as glucose tolerance, insulin resistance, left ventricular hypertrophy and cardiovascular fitness (6). The Framingham study estimates the benefits of exercise training to have a 20% reduction in the risk of CHD, which is substantially greater than the benefit obtained from diuretic or beta-blocker therapy (20).

### Exercise Prescription

The main reason for the underutilization of exercise in patients with hypertension is poor understanding of exercise and the interaction of frequency, intensity and duration required to achieve blood pressure reductions (7). The American College of Sports Medicine (21) offers the following exercise guidelines for the hypertensive patient. The duration of exercise needs to be continuous to produce benefits. A daily accumulation of 30 to 60 minutes of exercise is preferred. For those unable to sustain longer exercise periods, intermittent bouts (10-minute minimum) can also be implemented at different

times throughout the day. Longer exercise durations offer added benefits. Hypertensive patients should exercise at least 3 times a week but it is more beneficial if exercise is undertaken most days of the week. The intensity of exercise should be low to moderate and correspond to an exercise heart rate of approximately 50 to 80% of predicted maximal heart rate (7, 21).

### Post Exercise Hypotension

Recent studies in humans have shown that immediately after a single bout of exercise, there are profound changes in the mechanisms that regulate and determine arterial pressure. These changes result in a post-exercise hypotension (PEH) that lasts nearly two hours in healthy individuals and may last beyond twelve hours in hypertensive patients (13, 22). PEH is defined as a reduction in systolic and/or diastolic arterial blood pressure below control levels after a single bout of exercise (13). Numerous studies have confirmed the existence of PEH in normotensive and hypertensive individuals (7, 24 – 41). PEH is characterized by a persistent drop in systemic vascular resistance that is not completely offset by increases in cardiac output (22). Immediately post-exercise, cardiac output declines from high exercising values more rapidly than systemic vascular resistance recovers. This imbalance in the two determinants of arterial pressure results in a hypotension that is maintained for hours (18).

### Effects of Exercise Intensity on PEH

In examining the effects of exercise intensity on PEH, the majority of studies have utilized submaximal cycle ergometry protocols at intensities which have ranged between

40 – 100% of maximal exercise, as indicated by measurements of  $\text{VO}_2$ , heart rate reserve or predicted maximal heart rate (19, 23 – 25, 31, 33 – 40).

Pescatello et al. found no difference in the magnitude of PEH observed following 30-min bouts of cycle ergometry at these intensities (30). MacDonald et al. using the Finapres method (intra-arterial blood pressure monitor), found that mild to moderate (50 – 70%  $\text{VO}_{2\text{peak}}$ ) intensity exercise, elicited similar magnitudes of PEH in a normotensive population (24). This study confirmed the existence of PEH in a normotensive population, although the decrement found in the study of ~ 8/5 mm Hg are of lesser magnitude than those seen with a hypertensive population (24). This may suggest that hypertensive individuals may receive greater benefit from an acute bout of exercise than normotensive individuals. A study by Marceau et al. found that 10 weeks of closely supervised exercise training at 50% and 70% of maximal oxygen uptake induced comparable decreases in average 24-hour blood pressure and in average 24-hour blood pressure load (26). These results indicate that low to moderate intensity exercise is just as, if no more, efficacious as higher intensity training for reducing blood pressure in hypertensive individuals (10).

However, in a study by Hagberg et al. it was found that higher exercise intensity, 70% compared to 50%  $\text{VO}_{2\text{max}}$ , resulted in a greater decrease in systolic blood pressure, 13 mm Hg compared to 8 mm Hg respectively, for a longer period of time, three hours compared to one hour (33). This may suggest that a higher intensity may have a more dramatic and longer lasting effect.

### Effects of Exercise Duration on PEH

Two studies by MacDonald et al. documented that the duration of exercise does not play a significant role in determining the occurrence or magnitude of PEH. The initial study indicated that each of 15, 30 and 45 min of cycle ergometry at 70%  $VO_{2peak}$  elicited a similar hypotensive response in healthy young men. The second study found that 10 min of exercise was also sufficient to cause PEH (28). This is contradictory to earlier work by Bennet et al., who suggested that as the duration of exercise increased so did the magnitude of the hypotension (31). MacDonald et al. observed in both of their studies that blood pressure was more stable or still declining at the end of the 1 hour measurement period following the longer duration, whereas it was increasing after the shorter exercise bouts (28).

Thus, it may be possible that the duration of exercise may affect the duration of the hypotension. These findings support numerous studies that found PEH to occur following a variety of exercise durations and suggest that moderate intensity exercise of relatively short duration is sufficient to evoke a hypotension response (28).

### Duration of PEH

The duration of the hypotensive period following an acute exercise session has not been consistently quantified (39). Pescatello et al. concluded that post-exercise blood pressures were lower than pre-exercising levels for 7 to 12 hours after exercise (30, 39). These findings are consistent with earlier reports of post-exercise hypotension in hypertensive patients for up to 4 hours after exercise (33, 40, 42). Rueckert et al. confirmed the findings of others that a single bout of dynamic exercise results in a

decreased post-exercise resting blood pressure in individuals with established

hypertension for approximately 2 hours post-exercise (38). The major new finding was

that the hemodynamic pattern of the post-exercise hypotension was biphasic (38).

Initially, there was a drop in both total and regional vascular resistance. later, there was a

drop in cardiac output with total peripheral resistance at or above pre-exercise levels (38).

Contrary to Pescatello et al. they found that the decline in blood pressure was not

sustained once the study participants left the test site and went about their normal

activities. This difference could be that during testing, Pescatello et al. measured

auscultatory blood pressures and then compared those to ambulatory pressure recorded

later, whereas Rueckert et al. compared ambulatory blood pressure readings to other

control ambulatory readings (38, 39).

#### Effect of Exercising Muscle Mass on PEH

It has been estimated that the mass of the upper limbs account for approximately

7.6% of the body's mass, whereas the lower limbs account for approximately 32%. Thus

at the same relative exercise intensity, the total active muscle mass and absolute

metabolic rate would be greater for leg ergometry than for arm ergometry (25).

MacDonald et al. hypothesize that if PEH is mediated by some peripheral factor, then leg

ergometry endurance exercise would result in a greater decline in blood pressure.

However, they found that the mass of the working muscle does not directly affect the

magnitude of PEH but may influence the duration of the response. These results suggest

that a central mechanism or decreased vascular responsiveness is responsible for PEH

(25).

### Effect of Recovery Position on PEH

One of the possible mechanisms underlying PEH is some alteration in peripheral vascular regulation. Raine et al. hypothesized that PEH may be the result of an impaired vasoconstrictor response. To study this they examined the central and peripheral hemodynamic responses during supine and seated recovery after maximal upright exercise. They found that there was a lower peripheral resistance in the supine compared with the seated recovery position, suggesting that there is potential for greater vasoconstriction although this is not evoked to increase blood pressure. This further suggests that the arterial baroreceptor reflex is reset to a lower operating pressure after exercise (29).

### Effects of Resistance Training on PEH

The American College of Sports Medicine reports that resistance training has not consistently been shown to significantly lower blood pressure in hypertensive patients and is not recommended as the only form of exercise for these patients (21). The information available on the effects of strength training on resting blood pressure is limited and conflicting (10). In contrast to ACSM, MacDonald et al. concluded that post-exercise hypotension occurs following acute bouts of either resistance or submaximal dynamic exercise (34). A recommended circuit weight-training program should consist of 10 – 15 repetitions per set with weights of 30 – 50 percent of max. Rest intervals of 15 to 30 seconds should be included. Anaerobic heavy lifting should be avoided by patients with any signs of CVD (21, 34).

### Mechanisms

The underlying mechanisms mediating the hypotensive response elicited by exercise training remain elusive and controversial. Current opinion prevails that exercise training must act upon several pathophysiological mechanisms. Ultimately, the effects of exercise result in the reduction of total peripheral resistance, cardiac output or both.

Exercise may lower blood pressure through a variety of other mechanisms, including effects on the sympathetic nervous system, insulin sensitivity, electrolyte balance, neural and baroreceptor reflex mechanism, and vascular structure. Improved endothelial function is another possible mediator of the hypotensive response observed with exercise training (7).

### Cardiac Output

Changes in post-exercise heart rate and cardiac output have been inconsistent and may be influenced by exercise intensity or initial hemodynamic state of the subjects (38). Hagberg et al. found systolic blood pressure was significantly lower after exercising older hypertensives at either 50 or 70% of maximum oxygen consumption. They established that this was primarily due to a reduction in cardiac output, since total peripheral resistance was increased throughout both recovery periods (33). Cardiac output was reduced in recovery because of the reduction in stroke volume. Heart rate was above or no different, from that at rest before exercise. Changes in plasma volume could not entirely account for the reduction in stroke volume. Therefore, other mechanisms altering venous return and/or myocardial contractility appeared to be responsible for the reduction in systolic blood pressure evident after a single bout of submaximal exercise in

individuals with essential hypertension (33). However, other studies have found increased cardiac output (43) and heart rate (42).

### Systemic Vascular Resistance

In comparison with rest, post-exercise hypotension is characterized by a persistent drop in systemic vascular resistance that is not completely offset by increases in cardiac output (13, 22). At the end of exercise, cardiac output declines from high exercising values more rapidly than systemic vascular resistance recovers. This imbalance in the two determinants of arterial pressure results in a hypotension response that is maintained for several hours (22). Halliwill also noted that vasodilatation appears not to be limited to only the active skeletal muscle (i.e., the legs) but involves inactive regions (e.g., the arms) as well (13). Environmental factors affect systemic vascular resistance as well. Exercise in a hot environment will exacerbate PEH in two ways, a greater loss of plasma volume due to sweating and a greater drop in vascular resistance due to vasodilatation of the cutaneous vessels (22).

### Endothelial Function

The endothelial cells lining the arterioles and small arteries synthesize several substances that, when released, can affect the degree of relaxation or contraction of the arterial wall. The most important of these vasodilator substances is called endothelial-derived relaxing factor (EDRF), or nitric oxide (NO) (1). The healthy vascular endothelium maintains normal vasomotor tone, promotes clot lysis, and inhibits vasoconstriction, platelet aggregation, inflammation and intimal growth (16). The NO system plays a central role in the regulation of endothelial vascular tone, with failures of

NO synthesis partially accounting for the increased vascular resistance that is observed in hypertension (9). During acute exercise, there is an increase of blood flow, sheer stress on the endothelial cells and catecholamines, which all stimulate the release of NO and may even increase NO production after an acute bout of exercise (22).

Impairment of the essential functions of the endothelium is associated with coronary risk factors and fully developed atherosclerosis (16). Hypertension may also be part of the cause of injury to the endothelium and can also cause blood vessel abnormalities. These abnormalities of endothelial dysfunction may be related to the complications of hypertension and the determination of blood pressure itself (44).

Simple strategies, such as nutritional supplementation with L-arginine, may have an effect of blood pressure reduction by acting on the common pathway of NO-mediated vasodilatation. Schuster-Decker et al. concluded that supplementation of L-arginine, vitamin E, and vitamin C produced short-term reductions in blood pressure that are significantly greater than no treatment or only exercising (9).

#### Sympathetic Nerve Activation

It is well established that exercise results in increased secretion of catecholamines (45). The results of several studies on sympathetic nerve activity indicate that inhibition of basal sympathetic nerve discharge may contribute to PEH in hypertensive patients (13). The studies have used plasma levels of norepinephrine as an indirect measure of sympathetic nerve activity and tend to be inconsistent and it is clear additional data are needed. Circulating hormones, local metabolic factors, or both may play a role in

mediating PEH. Unfortunately, the studies to date have not provided information that clarifies the influence of these mechanisms on PEH (13).

### Somatic Afferents

Contracting skeletal muscle stimulates both mechanoreceptor and metaboreceptor sensory afferents, but this contribution to PEH has not been established. It is known that dynamic exercise and electrical stimulation of somatic afferents produce a number of similar responses, including sustained increases in arterial pressure and heart rate and post contraction/stimulation reductions in arterial pressure. However, it is not clear whether this effect is mediated by a sustained activation during the post-exercise period or by a decrease in afferent activity on cessation of exercise (32).

### Baroreceptor Reflex

The rise in arterial blood pressure above resting levels during short-term exercise is thought to be mediated by an upward resetting of the baroreceptor reflex operating set point (13). It is unclear whether the downward setting of the baroreceptor reflex at the cessation of exercise could be involved in the mediation of PEH (23).

Somers et al. reported that baroreflex impairment during exercise appeared to persist for 10 min after exercise is terminated. The reasons for this are unclear but many factors such as body temperature, skin vasodilatation, circulating catecholamines and other hormones may be involved (23). They found after 20 min the baroreflexes appeared to recover with a subsequent increase in sensitivity. Although the baroreflex sensitivity recovered slowly after exercise, at 40 and 60 min it was higher than the control value and they inferred that it could therefore contribute to the sustained reduction in

blood pressure after a period of heavy exercise. Blood pressures remained consistently low for the entire hour post-exercise. They felt that the immediate post-exercise reduction may be induced by local metabolite vasodilatation and that the resetting of the baroreflex sensitivity maintained the reduction in post-exercise blood pressure (23). Further investigation is warranted to explain the resetting mechanism.

### Thermoreflexes

Exercise increases metabolic heat production and internal body temperature. Activation of these mechanisms increase cutaneous vascular conductance, which in turn is thought to decrease systemic vascular resistance, and thus reduce arterial blood pressure. These thermally-induced physiological adjustments may contribute to PEH (13).

In general, an important role for various humoral, local metabolic and thermal factors in PEH has not been well established and it is clear that additional data is needed to provide explanation of the mechanisms involved (13).

### Exercise as a Predictor of Hypertension

Exercise may be a useful tool in the early detection of hypertension, it has been suggested that a hyperdynamic blood pressure response to exercise is useful in predicting the development of essential hypertension (45, 46) Hypertensive individuals have a significantly greater rise in systolic blood pressure following exercise than do normal individuals (45, 47). Davidoff et al. found exercise testing useful in determining potential hypertension. Out of 721 subjects, 236 subjects had an exaggerated systolic blood pressure reading of 200 mm Hg or more during an exercise test and they found that

seventeen percent of these subjects developed hypertension over a period of fifteen years (45).

Dlin et al. followed 75 individuals who were normotensive at rest but showed an exaggerated blood pressure response to exercise, defined as systolic blood pressure  $\geq 200$  mm Hg and or a rise of diastolic pressure of 10 mm Hg or more if this value then exceeded 90 mm Hg (47). A follow-up was performed approximately 6 years later and 10.6% of the subjects were found to be hypertensive (47).

Iskandrain and Heo. defined the hemodynamic profiles of the older and younger hypertensive patient. The hemodynamic profile of an older hypertension patient reveals a normal cardiac index and an increase in total vascular resistance (46). The hemodynamic response of a younger hypertensive reveals a hyperdynamic circulatory response characterized by a rapid heart rate and increase cardiac output (46). Normotensive individuals who exhibit an exaggerated systolic blood pressure response to exercise do not have a clearly defined hemodynamic response. Iskandrian and Heo. questioned whether the exaggerated blood pressure response is due to a high cardiac output or to an excessive increase in total vascular resistance (46). They found that the exaggerated systolic blood pressure in normotensive subjects was due to higher cardiac output and their total vascular resistance was similar to that of healthy subjects. More research is needed to answer whether the subjects with exaggerated blood pressure response are supernormals or if they represent the hyperdynamic phase of essential hypertension (44).

### Summary

Several studies indicate that PEH is common after low to high intensity exercise in normotensive and hypertensive individuals. Minimal duration, as little as 10 min, can cause PEH whereas longer durations, such as 45 min, may have a longer lasting effect on PEH. PEH has been documented to last from 2 – 12 hours after exercise. The hemodynamic response of exercise has been found to be greater in hypertensive individuals. Studies have also found that the normotensive individual who displays a hyperdynamic blood pressure response to exercise may have a greater chance of developing hypertension later. The mechanisms responsible for the hypotensive response post-exercise are largely controversial but most likely result from a reduction in systemic vascular resistance. This seems to be mediated by the autonomic nervous system and vasodilator substances, such as EDRF. The most important factor relating to hypertension is that PEH may be linked to long-term adaptations to exercise training.

## REFERENCES

1. Guyton AC. (1991) *Textbook of Medical Physiology*. (Eighth Edition). Philadelphia PA; Saunders.
2. Hennekens CH (1998). Increasing the burden of cardiovascular disease, current knowledge and future directions for research on risk factors. *Circulation*. 97:1095-1102.
3. Joint National Committee on Detection, Evaluation, and Treatment of Blood Pressure (1997). The Sixth Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. *Arch. Intern. Med.* 157:2413-2446.
4. National High Blood Pressure Education Program (1993). Working group report on primary prevention of hypertension. National Institutes of Health. U.S. Department of Health and Human Services. NIH Publication No. 93-2669.
5. American Heart Association (1998). Heart and stroke statistical updates. Dallas, TX: American Heart Association.
6. Hagberg JM (1997). Physical activity, physical fitness, and blood pressure. *Human Kinetics*. 112-119.
7. Kokkinos PF, Narayan P, Papademetriou V (2001). Exercise as hypertension therapy. *Cardiol. Clinics*. 19:507-516.
8. Carretero OA, Oparil S (2000). Essential Hypertension Part I: Definition and Etiology. *Circulation*. 101:329-335.
9. Schuster-Decker R, Foster C, Porcori JP, Maher MA (2002). Acute effects of dynamic exercise and nutritional supplementation on blood pressure in mildly hypertensive patients. *Clin. Exerc. Physiol.* 4:17-21.
10. Hagberg JM, Park J, Brown MD (2000). The role of exercise training in the treatment of hypertension. *Med. Sci. Sports Exerc.* 30:193-204.
11. Fagard RH (1999). Physical activity in the prevention and treatment of hypertension in the obese. *Med. Sci. Sports Exerc.* 31:S624-S630.

12. Carretero OA, Oparil S (2000). Essential Hypertension Part II: Treatment. *Circulation*. 101:446-453.
13. Kenney MJ, Seals DR (1993). Postexercise hypotension: Key features, mechanisms, and clinical significance. *Hypertension*. 22:653-664.
14. Rowland TW (1998). The biological basis of physical activity. *Med. Sci. Sports Exerc*. 30:392-399.
15. Deblinger L, Obarzanek E, Oberman A, Sheps SG, Willett WC (2001). Nondrug approaches to hypertension. *Patient Care*. 10pp.
16. Mulrow CD, Chiquette A, Cornell J, et al (2000). Dieting to reduce body weight for controlling hypertension in adults. *Cochrane Database Syst. Rev*. CD000484.
17. Gordon N, Scott C, Levine B (1997). Comparison of single versus multiple lifestyle interventions: are the antihypertensive effects of exercise training and diet-induced weight loss additive. *Am. J. Cardiol*. 79:763-767.
18. Cutler JA, Follmann D, Allender Ps (1997). Randomized trials of sodium reduction: An overview. *Am J. Clin. Nutr*. 65:643S – 651S
19. Grimm RH, Grandits GA, Culter JA, et al (1997). Relationships of quality-of-life measures to long-term lifestyle and drug treatment in the treatment of mild hypertension study. *Arch. Intern. Med*. 157:638-664.
20. Anderson KM, Wilson PWF, Odell PM, Kannel WB (1991). An updated coronary risk profile. *Circulation*. 83:356-362.
21. American College of Sports Medicine (2000). *Guidelines for Exercise Testing and Prescription*. (Sixth Edition). Baltimore, MD: Lippincott Williams & Wilkins.
22. Halliwill JR (2001). Mechanisms and clinical implication of post-exercise hypotension in humans. *Exerc. Sports Sci. Rev*. 29:65-70.
23. Somer VK, Conway J, LeWinter M, Sleight P (1985). The role of baroreflex sensitivity in post-exercise hypotension. *J. Hypertens*. 3:S129-S130.
24. MacDonald JR, MacDougall JD, Hogben CD (1999). The effects of exercise intensity on post exercise hypotension. *J. Human Hypertens*. 13:527-531.
25. MacDonald JR, MacDougall JD, Hogben CD (1999). The effects of exercising muscle mass on post exercise hypotension. *J. Human Hypertens*. 14:317-320.

26. Marceau M, Kouame N, Lacourciere Y, Cleroux J (1993). Effects of different training intensities on 24-hour blood pressure in hypertensive subjects. *Circulation*. 88:2803-2811.
27. Motoyama M, Sunami Y, Kinoshita F, et al (1998). Blood pressure lowering effect of low intensity aerobic training in elderly hypertensive patients. *Med. Sci. Sports Exerc.* 30:818-823.
28. MacDonald JR, MacDougall JD, Hogben CD (1999). The effects of exercise duration on post exercise hypotension. *J. Human Hypertens.* 14:125-129.
29. Raine NM, Cable T, George KP, Campbell IG (2000). The influence on recovery posture on post-exercise hypotension in normotensive men. *Med. Sci. Sports Exerc.* 33:404-412.
30. Pescatello LS, Fargo AE, Leach CN, Scherzer HH (1991). Short-term effect of dynamic exercise on arterial blood pressure. *Circulation*. 470:231-241.
31. Bennet T, Wilcox RG, MacDonald IA (1984). Post-exercise reduction of blood pressure in hypertensive men is not due to acute impairment of baroreflex function. *Clin. Sci.* 67:97-103.
32. Rowell LB (1986). Human circulation regulation during physical stress. London, England. *Oxford University Press*.
33. Hagberg JM, Montain SJ, Martin WH (1987). Blood pressure and hemodynamic responses after exercise in older hypertensives. *J. Appl. Physiol.* 63:270-276.
34. MacDonald JR, MacDougall JD, Interisano SA, Smith KM, Youngai EV, Tarnopolsky MA (1998). Hypotension following mild bouts of resistance exercise and submaximal dynamic exercise. *Eur. J. Appl. Physiol.* 79:148-154.
35. MacDonald JR, Hogben CD, Tarnopolsky MA, MacDougall (2001). Post exercise hypotension is sustained during subsequent bouts of mild exercise and simulated activities of daily living. *J. Human Hypertens.* 15:567-571.
36. Kaufman FL, Hughson RL, Schaman JP (1987). Effect of exercise on recovery blood pressure in normotensive and hypertensive subjects. *Med. Sci. Sports Exerc.* 19:17-20.
37. Gilders RM, Voner C, Dudley GA (1989). Endurance training and blood pressure in normotensive and hypertensive adults. *Med. Sci. Sports Exerc.* 21:629-636.

38. Rueckert PA, Slane PR, Lillis DL, Hanson P (1996). Hemodynamic patterns and duration of post-dynamic exercise hypotension in hypertensive humans. *Med. Sci. Sports Exerc.* 28:24-32.
39. Pescatello LS, Miller B, Danias PG, Werner M, Hess M, Baker C, De Souza MJ (1999). Dynamic exercise normalizes resting blood pressure in mildly hypertensive premenopausal women. *Am. Heart J.* 138:926-921.
40. Kaufman FL, Hughson RL, Schaman JP (1987). Effect of exercise on recovery blood pressure in normotensive and hypertensive subjects. *Med. Sci. Sports Exerc.* 19:17-20
41. Perry P (2001). Pressure Tactics. *Saturday Evening Post.* 273:60-68.
42. Floras JS, Sinkey CA, Aylward PE, Seals DR, Thoren PN, Mark AL (1989). Postexercise hypotension and sympathoinhibition in borderline hypertensive men. *Hypertension.* 14:28-35.
43. Clerous J, Kouname N, Nadeau A, Coulombe D, Lacourciere Y (1992). After effects of exercise on regional and systemic hemodynamics in hypertension. *Hypertension.* 19:183-191.
44. Lip GH, Blann AD (2000). Does hypertension confer a prothrombotic state? Virchow's triad revisited. *Circulation.* 101:218-220.
45. Davidoff R, Schamroth CL, Goldman AP, Diamond TH, Cilliers AJ, Myburgh DP (1982). Postexercise blood pressure as a predictor of hypertension. *Aviat. Space Environ. Med.* 53:591-594
46. Iskandrian AS, Heo J (1987). Exaggerated systolic blood pressure response to exercise: a normal variant or a hyperdynamic phase of essential hypertension? *Int. J. Cardiol.* 18:207-217
47. Dlin RA, Hanne N, Silverberg DS, Bar-Or O (1982). Follow-up of normotensive men with exaggerated blood pressure response to exercise. *Am. Heart J.* 106:316-320.
48. Pi-Sunyer FX (1999). Comorbidities of overweight and obesity: current evidence and research issues. *Med. Sci. Sports Exerc.* 31: S602-S608.
49. Zhang YC, Bui JD, Shen L, Phillips MI (2000). Antisense inhibition of  $\beta_1$ -Adrenergic receptor mRNA in a single dose produces a profound and prolonged reduction in high blood pressure in spontaneously hypertensive rats. *Circulation.* 101:682-688.

50. Ferrier KE, Waddell TK, Gatzka CD, Cameron JD, Dart AM, Kingwell BA (2001). Aerobic exercise training does not modify large-artery compliance in isolated systolic hypertension. *Hypertension*. 38:222-226.
51. MacDougall JD, Tuxen K, Sale DG, Moroz JR, Sutton JR (1985). Arterial blood pressure response to heavy resistance exercise. *J. Appl. Physiol.* 58:785-790.
52. Grim CE, Henry JP, Myers H (1995). *Hypertension: Pathophysiology, diagnosis, and management*. (Second Edition). New York, NY: Raven Press, Ltd.
53. Simone G, Greco R, Mureddu G, Romano C, Guida R, Celetano A, Contaldo R (2000). Relation of left ventricular diastolic properties to systolic function in arterial hypertension. *Circulation*. 101:152-157.
54. Green DJ, O'Driscoll G, Blanksby BA, Taylor RR (1996). Control of skeletal muscle blood flow during dynamic exercise: contribution of endothelium-derived nitric oxide. *Sports Med.* 21:119-146.