Institute of Physiology and Anatomy
German Sport University Cologne
Univ.-Prof. Dr. med. Otmar Bock

Acute and chronic effect of aerobic and resistance exercises on ambulatory blood pressure in hypertensive patients

Approved thesis submitted for the degree
Ph.D. Exercise Sciences
by
Andrea Solera Herrera
from San Jose, Costa Rica

Cologne
2011

First referee: Prof. Dr. rer. nat. Klaus Baum

Second referee: Univ.-Prof. Dr. med. Hans Georg Predel

Chair of the doctorate committee: Frau Univ.-Prof. Dr. I. Hartmann-Tews

Thesis defended on: 15th November 2011

Affidavits following § 7 Abs. 5 of the doctorate rules from the German Sport University Cologne, from February 02nd 2009.

Herby I declare: The work presented in this thesis is the original work of the author except where acknowledged in the text. This material has not been submitted and evaluated at any other time by any other person or institution. Those parts or single sentences, which have been taken verbatim from other sources, are identified as citations.

Cologne, January 25th, 2011.

ACKNOWLEDGEMENTS

This doctoral dissertation was realized with the great support of my mentors Prof. Dr. rer. nat. Klaus Baum of the Anatomy and Physiology Institute and Univ. Prof Dr. med. Hans Georg Predel of the Sport Medicine Institute at the German Sport University. I would like to extend them my sincere thanks and gratitude for their support, confidence, patience, understanding and scientific advice.

I am very grateful to Dr. Dr. med. Sandra Rojas for her support at the beginning of my academic career. Her advice and teachings were, are and will be very useful for me.

I also want to express here my great appreciation to Dr. med. Georgina Montiel for her inestimable contribution. Her medical support was an incalculable help.

Additionally, I would like to thank, all Members of the Sport Medicine Institute at the German Sport University and all Members of the Prof. Baum Training Institute, who gave me the possibility to conduct my research in their facilities. Sincere thanks not only for the technical support, but also for the offered friendship. Especially thanks to Anna Bickenbach for her assistance and help.

I want to warmly thank all the patients who have been involved in my research, for being so cooperative, disciplined and dedicated. It was a great pleasure for me to instruct them during the exercise.

Finally and most importantly, I would like to thank my whole family, for his love, encouragement and faith in me.

A Sebastián, mi más grande tesoro,

Tu inocencia me dio sabiduría
Tu sonrisa inspiración
Tu fragilidad fortaleza
Tu ternura ganas de seguir luchando
Tu compañía seguridad de que todo valió la pena

Que Dios te bendiga hoy y siempre

TABLE OF CONTENTS

LIST OF ABBREVIATIONS AND SYMBOLS

ABP	ambulatory blood pressure
ABPM	ambulatory blood pressure monitor
ACSM	American College of Sport Medicine
AC	aerobic training condition
AE	after exercise
AFI	automatic function imaging
AG	aerobic group
ANOVA	analysis of variance
ARC	aerobic and resistance training condition
ARG	aerobic and resistance group
BE	before exercise
BMI	body mass index
BP	blood pressure
°C	grade Celsius
CC	control condition
CVD	cardiovascular diseases
CG	control group
cm	centimeter
CO	cardiac output
dABPM	diastolic ambulatory blood pressure monitoring
dBP	diastolic blood pressure
dOBP	diastolic office blood pressure
dL	deciliter
DSHS	German Sport University
ECG	electrocardiogram
ECHO	echocardiogramm
g	gram
hr	hour
HbA1c	glycated hemoglobin
HDL	high-density lipoprotein
HOMA	homeostasis model assessment
HTN	hypertension
HR	heart rate

hsCRP	high sensitivity C reactive protein
kg	kilogram
L	liter
LDL	low-density lipoprotein
m	meter
mg	milligram
min	minute
mmHg	millimeter of mercury
mM	millimol
Nm	Newton meter
NYHA	New York Heart Association
OBP	office blood pressure
p	significance level
PEH	Post-exercise hypotension
PNE	plasma norepinephrine
PRA	plasma renin activity
PW	pulswave
PWC	physical work capacity
R	randomization
RC	resistance training condition
rep	repetition
RER	expiratory exchange ratio
RG	resistance group
RM	repetition maximum
RPE	rating of perceived exertion
rpm	revolutions per minute
s	second
sABPM	systolic ambulatory blood pressure monitoring
sBP	systolic blood pressure
SBPM	self blood pressure measurement
SD	standard deviation
sOBP	systolic office blood pressure
SD	standard deviation
SPSS	statistical program for social science
SVR	systemic vascular resistance

TPR	Total peripheral resistance
VE	ventilation
VCO_2	carbon dioxide production
VO_2	oxygen uptake
VO_2 max	maximal oxygen consumption
VO_2 peak	peak oxygen uptake
W	Watt
WHO	World Health Organization
wk	week
↓	decrease
↑	increase
♀	female
♂	male
Δ	change

LIST OF TABLES

LIST OF FIGURES

1. GENERAL INTRODUCTION

1.1. HYPERTENSION: DEFINITION AND EPIDEMIOLOGY

Hypertension (HTN), for adults usually defined as persistent systolic blood pressure above 140 and/or diastolic blood pressure above 90 mmHg, is one of the most important public health problems around the world, affects about a quarter of the adult population in many countries (Fagard et al., 2005; Mohrman and Heller, 2006; Pescatello et al., 2004; Kjeldsen et al., 2009) and is a strong risk factor for the development of cardiovascular diseases (CVD) and renal failure (Kaplan, 1990; Predel, 2007; Messerli, 2007).

The relationship of blood pressure (BP) and risk of cardiovascular disease events is continuous, consistent and independent of other risk factors. It has been established that the higher the BP the greater the incidence of coronary artery disease, myocardial infarction, heart failure, stroke, peripheral arterial disease and renal insufficiency (Chobanian et al., 2003; Pescatello et al., 2004; Kjeldsen et al., 2009). In contrast, it has been clearly demonstrated that the risk of serious cardiovascular incidents is reduced by preventive measures and appropriate treatment of HTN (Morhman and Heller, 2006; Kjeldsen et al., 2009).

Systolic blood pressure rises throughout the adult range whereas diastolic pressure peaks at about age 60 years in men and 70 years in women and decreases gradually thereafter (Kjeldsen et al., 2009). Recent findings found that at ages 40-69 years each increment of 20 mmHg in systolic BP and 10 mmHg in diastolic BP is associated with doubles the risk of CVD across the entire BP range from 115/75 to 185/115 mmHg (Lewington et al., 2002). For this reason, a new category designated *prehypertension* (Table 1) has been added to the classification of HTN to stress to the public health importance of reducing BP and preventing this chronic disease via healthy lifestyle interventions in the general population (Chobanian et al., 2003; Pescatello et al., 2004; Mohrman and Heller, 2006; Kjeldsen et al., 2009).

1.2. TYPES OF HYPERTENSION

Hypertension is commonly classified into two groups: essential hypertension and secondary hypertension (Mohrman and Heller, 2006).

1.2.1. Essential hypertension

According to Mohrman and Heller (2006), essential hypertension affects approximately 90% of cases but the primary abnormality that produces high blood pressure is unknown. Some few universally accepted facts about essential hypertension are:

a. Family history of HTN contributes to the development of this disorder.
b. An increase in total peripheral resistance occurs during the established phase of HTN and it may be due to:
    - Decrease of density of microvessels.
    - The pronounced structural adaptations that occur in the peripheral vascular bed.
    - A continuously increased activity of the vascular smooth muscle cells.
    - An increased sensitivity or reactivity of the vascular smooth muscle cells to external vasoconstrictor stimuli.
    - Diminished production and/or effect of endogenous vasodilator substances (e.g. nitric oxide).
c. Blood pressure-regulating reflexes could have an adaption to regulate blood pressure at a higher than normal level.
d. Environmental factors, such as high salt diets and psychological stress can contribute to the development of hypertension.

1.2.2. Secondary hypertension

According to Mohrmann and Heller (2006), secondary hypertension is presented in the remaining 10% of hypertensive patients. These causes can be traced to a variety of sources, including:

a. Epinephrine- and aldosterone-producing tumors.
b. Certain forms of renal disease (e.g. renal artery stenosis, glomerular nephritis, pre-eclampsia during the pregnancy).

c. Certain neurological disturbances (e.g. brain tumors which increase intracranial pressure).
d. Certain thyroid and parathyroid disorders (e.g. Cushing´s syndrome).
e. Lead poisoning.
f. Drugs side effects (e.g. hormonal contraceptives, hormonal replacement therapy, abuse of cortisol).

Testing the specific cause of BP elevation is indicated, if possible before initiation of antihypertensive therapy, because secondary hypertension responding poorly to these medicaments (Kjeldsen et al., 2009).

1.3. CLASSIFICATION OF HYPERTENSION

According to the traditional definition of high normal blood pressure, Table 1 provides the World Health Organization (WHO) blood pressure classification scheme for adults aged 18 years or older based on the mean of 2 or more properly measured seated readings on each of 2 or more office visits (Pescatello et al., 2004; Chobanian et al., 2007; Kjeldsen et al., 2009).

However it is important to consider that the blood pressure thresholds differ according with the procedure of measurement (see following sections).

BP Classification	**Systolic (mmHg)**		**Diastolic (mmHg)**
Optimal	<120	and	<80
Normal	120-129	and/or	80-84
High normal or pre-hypertension	130-139	and/or	85-89
Grade 1 hypertension	140-159	and/or	90-99
Grade 2 hypertension	160-179	and/or	100-109
Grade 3 hypertension	≥180	and/or	≥110
Isolated systolic hypertension	≥140	and	<90

Table 1 Classification of blood pressure levels for adults aged 18 years or older according to the World Health Organization Guidelines (Chobanian et al., 2003).

BLOOD PRESSURE MEASUREMENTS

Blood pressure is characterized by large inherent variability both within 24 hours and between days (Pickering et al., 2006; Kjeldsen et al., 2009). For this reason, decisions based on single measurements will result in erroneous diagnosis and inappropriate management. Reliability of measurements is improved if repeated measures are obtained. The different procedures of measurement are summarized in the following sections (O'Brien et al., 2003).

1.4.1. Office or clinic blood pressure measurement (OBP)

Blood pressure can be measured in the office or in the clinic by a mercury sphygmomanometer or other non-invasive semi-automatic devices which should be properly calibrated and validated. (Chobanian et al., 2003; Kjeldsen et al., 2009). Aspects to consider during the OBP measurement are:

a. Patient should be seated quietly for several minutes in a quiet room before beginning BP measurement, with feet on the floor and arm supported at heart level.
b. An appropriate-sized cuff (cuff bladder encircling at least 80% of the arm) should be used to ensure accuracy.
c. At least 2 measurements spaced by 1-2 min should be made. Additional measurement should be obtained, if the first two are quite different.
d. Systolic BP is the point at which the first of 2 or more sounds is heard and diastolic BP is the point before the disappearance of sounds.
e. Measurement of BP in the standing position is indicated frequently, especially in those at risk for postural hypotension (e.g. diabetic and elderly patients).

1.4.2. Ambulatory blood pressure monitoring (ABPM)

The currently available ambulatory monitors are fully automatic and can record BP for 24-hr periods or longer while patients go about their normal daily activities. This provides a profile of BP away from the medical environment thereby allowing identification for individuals with a white-coat hypertension defined as a high clinic BP in the absence of target-organ injury and evidence of normal blood pressure outside the

clinic (Thibonnier, 1992; O´Brien et al., 2000; Chobanian et al., 2003; O'Brien et al., 2003; Marchiando and Elston, 2003; Pickering et al., 2006; Kjeldsen et al., 2009).

The monitors are typically programmed to take readings every 15 to 30 minutes throughout the day and night. At the end of the recording period the readings are downloaded onto a computer (Marchiando and Elston, 2003; Pickering et al., 2006) and allow information on 24-hr average BP as well as average blood pressure values on more restricted portions of the 24 hours, such as during the day, the night and the morning (Kjeldsen, 2009).

Cross-sectional and longitudinal studies have shown that ABPM predicts cardiovascular events better than OBP (Verdecchia et al., 1994; Staessen et al., 1999; Staessen et al., 2000; Dolan et al., 2005; Sega et al., 2005) and correlates closely with the subclinical organ damage of hypertension (Appel and Stason, 1993; Verdecchia, 2000).

Aspects to consider during the ABPM measurement are (O'Brien et al., 2000; O'Brien, et al., 2003; Kjeldsen et al., 2009):

a. The devices should be validated and calibrated by international standardized protocols.
b. An appropriate-sized cuff should be used to ensure accuracy.
c. The readings should be set at no more than 30-min intervals to obtain an adequate number of values.
d. The patient should be engaged in normal activities but avoiding strenuous exercise.
e. At the time of cuff inflations, the arm should be extended and reposeful.
f. The patient should provide information in a diary on unusual events, on medicaments intake and on duration and quality of night sleep.
g. Another ABPM should be obtained if the first examination has <70% of the expected values because of a high number of artifacts.
h. The hypertension threshold by this method should be considered, because ABPM is usually several mmHg lower than OBP (see Table 2).

1.4.3. Self blood pressure measurement (SBPM)

Although self-measurements of BP at home cannot provide the extensive information of the ABPM, they can give values on different days in a setting close to daily life conditions, evaluate white coat hypertension (Appel and Stason, 1993; Celis et al., 2005) and improve the patient's adherence to treatment regimens (Friedman et al., 1996).

In addition, SBPM may offer some advantage over OPB in predicting cardiovascular outcomes in hypertension, but the data is insufficient and the results of current trials must awaited (Appel and Stason, 1993).

Aspects to consider during the SBPM measurement are (O'Brien, et al., 2003; Kjeldsen et al., 2009):

a. Home devices should be validated by standardized protocols and calibrated regularly for accuracy.
b. There should be a 5-min period of rest before measurement.
c. The cuff of the measuring device must be at the level of the heart on the arm with the highest blood pressure.
d. During diagnosis and initiation of treatment, duplicate SBPM should be made in the morning and evening for 1-wk.
e. The hypertension threshold by SBPM method should be considered, because it is usually several mmHg lower than OBP (see Table 2).

1.4.4. Exercise blood pressure

Systolic blood pressure increases with rising dynamic work as a result of increasing cardiac output whereas diastolic pressure usually remains about the same or moderately lowers (Heck et al., 1984; O'Brien et al., 2003). However, there is evidence that an exaggerated blood pressure response with exercise testing may be predictive of future hypertension (Singh et al., 1999; Mathews et al., 1998), hypertension-related target organ damage (Manolio et al., 1994) and mortality from myocardial infarction (Mundal et al., 1996).

While systolic blood pressure may be recorded reasonably well during bicycle exercise, diastolic values may be grossly overestimated or underestimated. Nevertheless, an exaggerated diastolic blood pressure response to exercise should not be ignored (Franz, 2003; O'Brien et al., 2003).

Although, a fixed workload was used to determine the exercise BP threshold it should be considered that a fixed workload does not represent the same percentage of maximal exercise capacity in all patients. In this context an exercise BP value greater than 200/100 mmHg at a moderate workload (100-W) for adults aged 20-50 years should be regarded as abnormal (see Table 2); for adults aged 51-60 and 61-70 years are valid the upper limits of 210/105 and 220/110 respectively (Franz, 2003).

Finally, an elevated recovery systolic BP after exercise may be predictive of future hypertension (Singh et al., 1999). In this instance, the recovery BP following a 100-W workload should be <140/90 mmHg after 5-min (Franz, 2003).

Method	Systolic BP (mmHg)	Diastolic BP (mmHg)
Office blood pressure	140	90
24-hr average ambulatory blood pressure monitoring	125-130	80
Day interval ambulatory blood pressure monitoring	130-135	85
Night interval ambulatory blood pressure monitoring	120	70
Self blood pressure measurement	130-135	85
Exercise blood pressure at 100-W	200	100
5-min recovery blood pressure after 100-W workload	140	90

Table 2 BP thresholds according with the type of measurement. Values for adults aged 20-50 years.

1.4. HIGH BLOOD PRESSURE PREVENTION AND NO-PHARMACOLOGICAL TREATMENT

The primary public health goal of antihypertensive therapy is to achieve the maximum reduction of cardiovascular and renal morbidity and mortality. This requires treatment of all the reversible risk factors identified, including smoking, dislipidaemia or obesity

as well as the reduction of the raised blood pressure below 140 mmHg systolic and 90 mmHg diastolic (Chobanian et al., 2003; Murphy et al., 2007).

Besides pharmacological treatments, adoption of healthy lifestyles by all individuals is essential for the prevention of high BP and an indispensable part of the management of those who have already developed the disease (Whelton et al., 2002a); because of this power to decrease BP enhances antihypertensive drug efficacy and decrease cardiovascular risk (Chobanian et al., 2003; Kjeldsen et al., 2009).

Major lifestyle modifications have the potential to lower BP which include: reducing weight in those who are overweight (He et al., 2000), adopting an eating plan to stop HTN, reducing sodium intake (Vollmer et al., 2001), moderating alcohol consumption (Xin et al., 2001; Dickinson et al., 2006), as well as making physical activity (Kelley and Kelley, 2000; Whelton et al., 2002b; Fagard, 2005a).

Although smoking cessation doesn´t appear to lower the BP (Kjeldsen et al., 2009), it is another recommended lifestyle modification because its role in the prevention of many cardiovascular diseases and future hypertension (Al-Safi, 2005). Table 3 summarizes effects of lifestyles changes on systolic BP reduction (Chobanian et al., 2003).

Modification	**Recommendation**	**Approximate Systolic BP Reduction**
Weight reduction	Maintain normal body mass index (18.5-24.9 kg/m^2)	5-20 mmHg/10 kg weight loss
Adopt eating plan to stop hypertension	Consume a diet rich in fruits, vegetables and low-fat dairy products with a reduced content of saturated and total fat.	8-14mmHg
Dietary sodium reduction	Reduce dietary sodium intake to no more than 2.4-g sodium or 6-g sodium chloride.	2-8 mmHg
Physical activity	Engage in regular aerobic physical activity, at least 30-min per day, most days of the week.	4-9 mmHg
Moderation of alcohol consumption	Limit consumption to no more than 2 drinks per day in most men and no more than 1 drink per day in women and lighter-weight persons.	2-4 mmHg

Table 3 Lifestyle modifications to manage hypertension. The effects of implementing these modifications are dose and time dependent and could be higher for some individuals.

1.5. JUSTIFICATION AND PURPOSE

Even though increasing ones physical activity is a common recommendation for the prevention and treatment of high BP (Whelton et al., 2002a; Chobanian et al., 2003; Pescatello et al., 2004), there exists lacking consensus about the optimal training frequency, intensity, duration and mode of exercise required to lower HTN. In addition, confounding variables such as the methods and the equipment used to measure arterial blood pressure could give confused results which should be clearly researched (Pescatello et al., 2004).

Because the need of data concerning the influence of different exercise modes on BP (especially resistance training), the present thesis has the purpose to clarify, in two randomized controlled trials, the acute and chronic effects of aerobic training vs. resistance training vs. the combination of both on the ambulatory blood pressure monitoring in patients with hypertension grade I. These two studies will be presented in the following two chapters, each with its respective introduction, methodology, results, discussion and conclusion.

The results of both studies can give valuable information not only for the medical and physiological fields, but also for the trainers who work prescribing exercise to this population and whose objective is to reduce the incidence of this chronic disease.

2. "CHRONIC EFFECT OF AEROBIC AND RESISTANCE EXERCISES ON AMBULATORY BLOOD PRESSURE"

2. 1. INTRODUCTION CONCERNING CHRONIC EFFECT OF EXERCISE ON BLOOD PRESSURE

2.1.1. Background

Currently there is a great amount of evidence suggesting that BP, the incidence of cardiovascular disease and mortality is lower in subjects who are more fit or physically-active (Fagard, 2002; Fagard, 2005b; Fagard, 2006). Despite the strength of this association the ideal physical activity for gaining health benefits remains unclear (Kelley, 1995; Kelly, 1999; Kelley et al., 2001; Whelton et al., 2002b; Cardoso et al., 2010).

The wide spectrum of variables that are playing a role under the term "physical activity" such as type or mode of exercise, length of training, frequency, intensity and duration of training (Pollock et al., 1998), make it difficult to quantify the benefits of exercise on BP and to provide general recommendations about the optimal characteristics of the training program (Fagard, 2001).

If we focus only on the variable type of exercise we find that exercise can be classified in two broad categories (Fagard and Cornelissen, 2007; Cardoso et al., 2010):

a. Aerobic training: designed to improve the function of the cardiovascular system and executed using cycle exercises and carried out with large muscle groups contracting at mild to moderate intensities for a long period of time.
b. Resistance training, designed to increase muscular strength, power as well as the aerobic capacity of skeletal muscle cells and executed by muscles from a specific body segment which are contracted against a force that opposes the movement. This kind of training can be subdivided into: isometric –static-, without movements of the limbs and isotonic –dynamic- with movements of the limbs (Williams et al., 2007). Furthermore, the predominantly dynamic training programs were divided into a:

- "Circuit program": it consists if lifting on set of weights with shorter rest periods between exercises and this circuit may be repeated after one complete tour, or
- "Conventional program of isolated exercises": it consists of lifting two or more sets of heavier weights in an isolated exercise with longer rest periods before going to the next exercise.

Given the wide popularity of the dynamic resistance training, it is important to mention, that this thesis deals only on this type of resistance training, unless otherwise noted.

Although both aerobic and resistance exercise appear to produce positive benefits on health and fitness variables, the physiological mechanisms for improvement may be different (Pollock et al., 2000; Williams et al., 2007). During the aerobic exercise increases the oxygen consumption, heart rate, cardiac output, stroke volume and systolic blood pressure, with maintenance of diastolic blood pressure resulting in a concomitant widening of pulse pressure and modest increase in mean pressure, with a decrease in peripheral vascular resistance. In contrast, during the resistance training, although there is an increase of heart rate, cardiac output and systolic blood pressure, the diastolic blood pressure rises too and the oxygen consumption show only a little augment. Despite the increase of cardiac output, as a result of muscle contraction, the intramuscular pressure exceeds the intravascular pressure reducing the blood flow and causing muscle ischemia and hypoxia.

Nevertheless, the results of studies about the chronic effect of both types of exercise on blood pressure remain still controversial.

For example, with respect to aerobic exercise, Westhoff et al. (2007) found that 12-wk of aerobic exercise significantly decreased systolic and diastolic ABP of 8.5/5.1 mmHg respectively in patients older than 60 years, with isolated systolic hypertension and taking antihypertensive medication and attributed the blood pressure reduction to an improvement of the endothelial function. Nevertheless, Cornelissen et al. (2009) detected that 10-wk of aerobic training performed at 33% and 66% intensity reduces the systolic office and exercise blood pressure but does not alter the ABP. Moreover, Ishikawa-Takata et al. (2003) studied the duration and frequency/week of aerobic exercise and found that training between 30 and 120 min/wk can achieved a significant

systolic and diastolic office blood pressure decrease. However, they did not measure the ABP.

Concerning to resistance training, Van Hoff et al. (1996) detected in healthy but sedentary persons that resting, ambulatory and exercise blood pressure were not affected by a 16-wk program of strength training performed 3 times/wk at 70% 1RM. In contrast, Harris and Holly (1987) using circuit weight training at 10 stations, with 3 sets for 20 to 25 repetitions of the exercises and with an initial workload of 40% of 1RM, noted in male borderline hypertensive subjects a 5% lower diastolic blood pressure at the end of the study. More recently, Strasser et al. (2008) found a significant ABP reduction after 4-months resistance training executed 3 times/wk, nevertheless in this trial lacked a control group.

Moreover, some studies compared the effect of aerobic exercise vs resistance exercise on blood pressure. Cononie et al. (1991) detected that 6 months resistance training does not adversely affect or reduce blood pressure, while endurance training produces a significant diastolic reduction of 5 mmHg in 70-79 years old individuals with somewhat elevated blood pressure. Parallel cardiac output, peripheral vascular resistance and plasma levels of angiotensin I and II and epi- and norepinephrine did not change in any of the groups. More recently, Collier et al. (2008) compared the effect of 4-wk aerobic exercise with the resistance training on hemodynamic characteristics and found that arterial stiffness increased following the resistance training but decreased after aerobic exercise; vasodilatory capacity improved following both trainings but increased more after resistance exercise and systolic and diastolic blood pressure had a significant reduction of about 4 mmHg following both exercise types. However both studies had not control group.

Using the meta-analytic technique, which is statistical method to integrate individual study findings addressing a common problem (Glass, 1981) it is possible to clarify the controversial points of the matter, to detect where a lack of information exists about the topic to identify areas of weakness and to provide directions for future research. For these reasons, several authors in the last years have used this procedure to quantify the chronic effects of aerobic and resistance training on blood pressure. Table 4 and 5 show the main results of recent meta-analysis respecting this classification.

Reference	N	Main findings	Aspects to consider
Kelley and McClellan, 1994	9	Lower extremities aerobic exercise produces: - Resting BP ↓ 7/6 mmHg ($p<0.05$)	- BP was measured in resting. - Subjects had hypertension.
Kelley, 1995	9	Aerobic exercise produces: - Resting BP ↓ 3/3 mmHg (↓ 2/4%, $p<0.05$) - Positive relationship between training duration per day and BP reduction ($p<0.05$).	- Subjects were adults with normal BP. - BP was measured in resting.
Kelley, 1996	7	Aerobic training produces: - ABP ↓ 1.7/2.6 mmHg ($p>0.05$ and $p<0.05$) - BMI ↓1%, resting HR ↓ 9% and VO_2max ↑ 9%.	- 6 studies had hypertensive subjects. - Not all reported medication status of the subjects. - 4 studies were randomized.
Kelley et al., 2001	16	Walking produces: - Resting BP ↓ 3/2 mmHg (↓ 2/3%, $p<0.05$) - VO_2max ↑ 12%, resting HR ↓ 5.5 lat/min. BP changes were not different between normotensive and hypertensive subjects.	- BP was measured in resting. - Only 6 studies reported subjects without medication. - 11 studies were randomized.
Fagard, 2001	44	Dynamic aerobic training produces: - BP ↓ 3.4/2.4 mmHg (↓ 2/4 %, $p<0.001$) BP lowering effect is more pronounced in hypertensive than in normotensive.	- Only 2 studies measured ABP.
Whelton et al., 2002b	54	Aerobic exercise produces: -BP ↓ 3.8/2.6 mmHg ($p<0.001$). The BP reduction did not have relationship with lost weight.	- Only 15 studies reported hypertension status. - In 4 trials was administrated antihypertensive medication.
Fagard, 2005a	44	Aerobic training produces: -BP ↓ 3.4/2.4 mmHg ($p<0.001$). -Exercise systolic BP decrease 7 mmHg and HR 6 beats/min..	-Participants were not always advised to keep diet or lifestyle constant throughout the study.
Cornelissen and Fagard, 2005a	72	Chronic aerobic training produces: -Resting BP ↓ 3.0/2.4 mmHg ($p<0.001$) and daytime ABP ↓ 3.4/3.5 mmHg ($p<0.01$; n=12). By hypertensive, the resting BP ↓ was 6.9/4.9 mmHg ($p<0.001$; n=30). -SVR ↓ 7.1%, PNE ↓ 28.7%, PRA ↓ 19.8%, weight ↓ 1.2 kg, waist circumference ↓ 2.8 cm, percent body fat ↓ 1.4%, HOMA index ↓ 0.31U and HDL ↑ 0.032 mM/L ($p<0.05$).	- 31 studies gave no information on antihypertensive medication. - Time between the last training session and the BP measurements is usually no reported. - Hemodynamic changes were reported in only 18 studies. - PNE and PRA changes were reported in only 16 studies.
Dickinson et al., 2006	21	Aerobic exercise produces: -BP ↓4.6/2.4 ($p<0.05$).	-1/4 of trials included patients on antihypertensive medication.
Murphy et al., 2007	9	Walking produced only significant effect on resting diastolic BP (↓ 1.54 mmHg, $p<0.05$), but not on systolic BP.	-Subjects had normal BP (baseline BP was 127/78 mmHg).

Table 4 Recent meta-analyses about the effect of aerobic exercise on blood pressure. Abbreviations and symbols are as follows: N= number of computed studies, ↓=decrease, BP=blood pressure, ABP= ambulatory blood pressure, BMI=body mass index, HR=heart rate, VO_2max=maximal oxygen consumption, SVR=systemic vascular resistance, PNE=plasma norepinephrine, PRA=plasma renin activity, HOMA index=homeostasis model assessment index of insulin resistance, HDL=high density lipoprotein cholesterol.

As seen in the Table 4, a variety of studies have found not only a positive chronic effect of aerobic training on resting blood pressure but also in glucose homeostasis, blood lipids and body fat among others (Kelley, 1995; Kelley 1996; Whelton et al., 2002b; Cornelissen and Fagard, 2005a). This parallel positive effect of aerobic exercise on other cardiovascular risk factors makes it difficult to explain if the blood pressure reduction comes from the influence of physical activity itself or from its effects on other cardiovascular risk factors or from the combined effects of all of these.

Reference	N	Main findings	Aspects to consider
Kelley, 1997	9	Dynamic resistance exercise produces: - Resting BP ↓ 4.5/3.8 mmHg ($p<0.05$). - No significant differences were found for either systolic or diastolic pressure between hypertensive and normotensive subjects.	- BP was measured in resting. - Only 1 study reported the intensity at which subjects trained. - Only 3 studies measured hypertensive individuals.
Kelley and Kelley, 2000	11	Progressive resistance exercise produces: - Resting BP ↓ 3/3 mmHg ($p>0.05$). - Percent body fat ↓ 2% and lean body mass ↑ 4 kg ($p<0.05$). - No significant changes for body weight, BMI, VO_2max and resting HR. In addition, BP response was no different between studies that used a circuit training protocol vs a conventional training protocol.	- BP was measured in resting. - 2 studies reported subjects without medication. - 3 studies measured hypertensive individuals. - 5 studies used circuit training (moderate intensity with little rest periods) and 4 conventional training protocol (high intensity with longer rest periods).
Cornelissen and Fagard, 2005b	9	Resistance training produces: - Resting BP ↓ 3.2/3.5 mmHg (significant only the change on diastolic BP, $p<0.05$). - VO_2max ↑ 10.5% Reduction in BP did not differ among studies with different intensities. BP response was no different between trials that used circuit training protocol or conventional training protocol.	- BP was measured in resting. - Only 1 study reported subjects without medication. - Only 3 studies measured hypertensive individuals. - 5 studies used circuit training and 4 conventional trainings protocol.

Table 5 Recent meta-analyses about the effect of resistance exercise on blood pressure. Abbreviations and symbols are as follows: N=number of computed studies, BP=blood pressure, BMI= body mass index, HR=heart rate, VO_2max=maximal oxygen consumption, ↑=increase, ↓=decrease.

On the other hand, the few meta-analyses about the influence of resistance training on blood pressure have led to inconsistent outcomes (see Table 5). While Kelley (1997) observed a significant resting BP reduction following resistance exercise, Kelley and Kelley (2000) found that the BP remained constant and Cornelissen and Fagard (2005b) detected only a significant reduction of the diastolic but not systolic resting BP.

Nevertheless, conclusions about the chronic effect of resistance training must be cautious because this topic has been less well studied (Fagard, 2006; Predel, 2007). Comparing two meta-analyses made by Cornelissen and Fagard (2005a and 2005b) in the same year and under the same selection criteria, they found that only 12% of the publications about the chronic effect of exercise on BP tested the effectiveness of resistance training as treatment against hypertension (Cornelissen and Fagard, 2005a and 2005b). Furthermore, a current review about the chronic effect of aerobic and resistance training on ambulatory blood pressure (24-hr) shows the same trend, while 30 studies were developed using the aerobic exercise as antihypertensive therapy, only 2 were made using the resistance training (Cardoso et al., 2010).

Dangerous increases of 320/250 mmHg shown in peak systolic and diastolic BP respectively during one repetition maximum lift -1RM- (Mac Dougall et al., 1985) might be influencing the lack of research using this mode of exercise. Nevertheless, it must be considered that these results were achieved during a maximal test performed by athletes who did not avoid the Valsalva maneuver during the exercise execution. This maneuver increases intrathoracic pressure which in turn increases the blood pressure as a consequence of holding the breath during the lifting efforts (Mac Dougall et al., 1992). Maintaining a normal breathing pattern can evade the athlete this dramatic acute increase in both systolic and diastolic blood pressure (Pollock et al., 1998). In fact, Baum et al. (2003) measured continuously blood pressure during the leg press exercise executed at 80% intensity and avoiding the Valsalva maneuver and found a maximal blood pressure responses of only 185/120 mmHg.

Although resistance training has not yet been recommended as a sole intervention to reduce the risk of hypertension and to decrease BP in mildly hypertensive subjects because its beneficial effects for the musculoskeletal system and quality of life and because it does not appear to raise resting and ambulatory blood pressure, current reviews and professional association position statements recommend its prescription at moderate intensity and preferably in combination with aerobic endurance training (Pollock et al., 2000; Pescatello et al., 2004; Bjarnason-Wehrens et al., 2004; Cornelissen and Fagard, 2005b; Williams et al., 2007, Cardoso et al., 2010).

Beside these cautious recommendations, the meta-analyses authors and the position statements of the professional associations emphasize the need for further research in this area and agree about the following aspects to consider in future researches:

a. Including scientific criteria such as control group or control phase (Kelley, 1995; Fagard, 2001).
b. Random allocating of subjects to the active or control group, or random order of the training and non-training phases to avoid selection bias (Kelley, 1995; Kelley et al., 2001; Fagard, 2005a).
c. Advising to keep diet or lifestyle constant throughout the study periods (Fagard, 2001; Fagard, 2005a; Dickinson et al., 2006).
d. Frequent monitoring of the training protocols (Fagard, 2005a).
e. Separating the last exercise session and the blood pressure measurement at least 24-hr to avoid an acute hypotensive effect of exercise on blood pressure (Kelley, 1997; Thompson et al., 2001; Cornelissen and Fagard, 2005a).
f. Enrolling hypertensive subjects, because the blood pressure reduction after training appears to be more pronounced in hypertensive than in normotensive persons (Kelley, 1996; Kelley, 1997; Kelley and Kelley, 2000; Whelton et al., 2002b; Fagard, 2001; Cornelissen and Fagard, 2005b; Murphy et al., 2007).
g. Complete reporting of the training and measurement protocols (Kelley, 1997; Cornelissen and Fagard, 2005b).
h. Including ambulatory blood pressure monitoring to avoid experimental error and the "white coat syndrome" (Thibonnier, 1992; Kelley, 1996; Pickering et al., 1988; Wendelin-Saarenhovi et al., 2001).
i. Enrolling participants without antihypertensive medication to avoid a possible interaction between exercise and drugs (Kelley and Kelley, 2000).
j. Reporting the number and reasons for withdrawal of subjects in each group (Kelley, 1999).

Given (1) the few controlled trials examining the chronic effect of aerobic training, resistance training or the combination of both on ambulatory blood pressure, (2) the discrepant findings about the antihypertensive effect of resistance training and (3) the conflicting results about the effect of both trainings modes on different patho-physiological variables which are related to arterial hypertension; it is critical to develop a quantitative approach using the previous recommendations to compare the chronic

influence of aerobic training, resistance training and their combination on systolic and diastolic ambulatory blood pressure as a non-pharmacological intervention in adults with hypertension grade I.

2.1.2. Research questions

a) Which mode of exercise (aerobic training, resistance training or the combination of both) has the strongest antihypertensive effect on 24-hr ambulatory BP, office BP and exercise BP?
b) Which experimental treatment has the best effect on anthropometric characteristics, blood parameters, exercise capacity and others hemodynamic values?
c) Is there a relationship between hemodynamic values, anthropometric characteristics, blood parameters and exercise capacity?

2.1.3. Hypothesis

Experimental groups that include aerobic exercise intervention will have a greater reduction on the blood pressure compared to groups that do not perform this type of exercise, because its parallel positive effects on pathophysiological disorders.

2.2. METHODS

2.2.1. Participants

The study was approved by the Ethic Committee of the German Sports University (DSHS). Participants were recruited from the general population by advertisements printed in local newspapers.

Interested volunteers initially participated in an orientation session to receive information on the aims, inclusion and exclusion criteria, procedures and risks of the study. A written informed consent was then obtained from all the interested volunteers.

2.2.1.1. Inclusion criteria:

Volunteers were considered for this study if they had a current 24-hr ambulatory blood pressure between 130/80 and 150/90 mmHg. These values are considered as Stage I Essential Hypertension according to the European Society of Hypertension- (O'Brien et al., 2003).

2.2.1.2. Exclusion criteria:

Volunteers who did not meet the inclusion criteria and/or one of the following characteristics were not allowed to participate in the study.

a. Volunteers currently taking antihypertensive treatments in the past 12-wk prior to the study.
b. Subjects engagement in regular physical exercise training in the past 12-wk prior to inclusion in the study.
c. Participants showing cardiac problems by like: symptomatic peripheral arterial occlusive disease, aortic insufficiency grad > III, stenosis grad > 1, hypertrophic obstructive cardiomyopathy, congestive heart failure (NYHA grad > II), uncontrolled cardiac arrhythmia with hemodynamic relevance and signs of acute ischemia in exercise electrocardiogram.
d. Persons with chronic obstructive pulmonary disease grad ≥2
e. Volunteers with physical limitations prohibiting exercise.

According to these criteria, 62 patients (48 Male, 14 Female) were enrolled and finished the experimental treatments. The recruitment, screening and drop out of participants are showed in Figure 1.

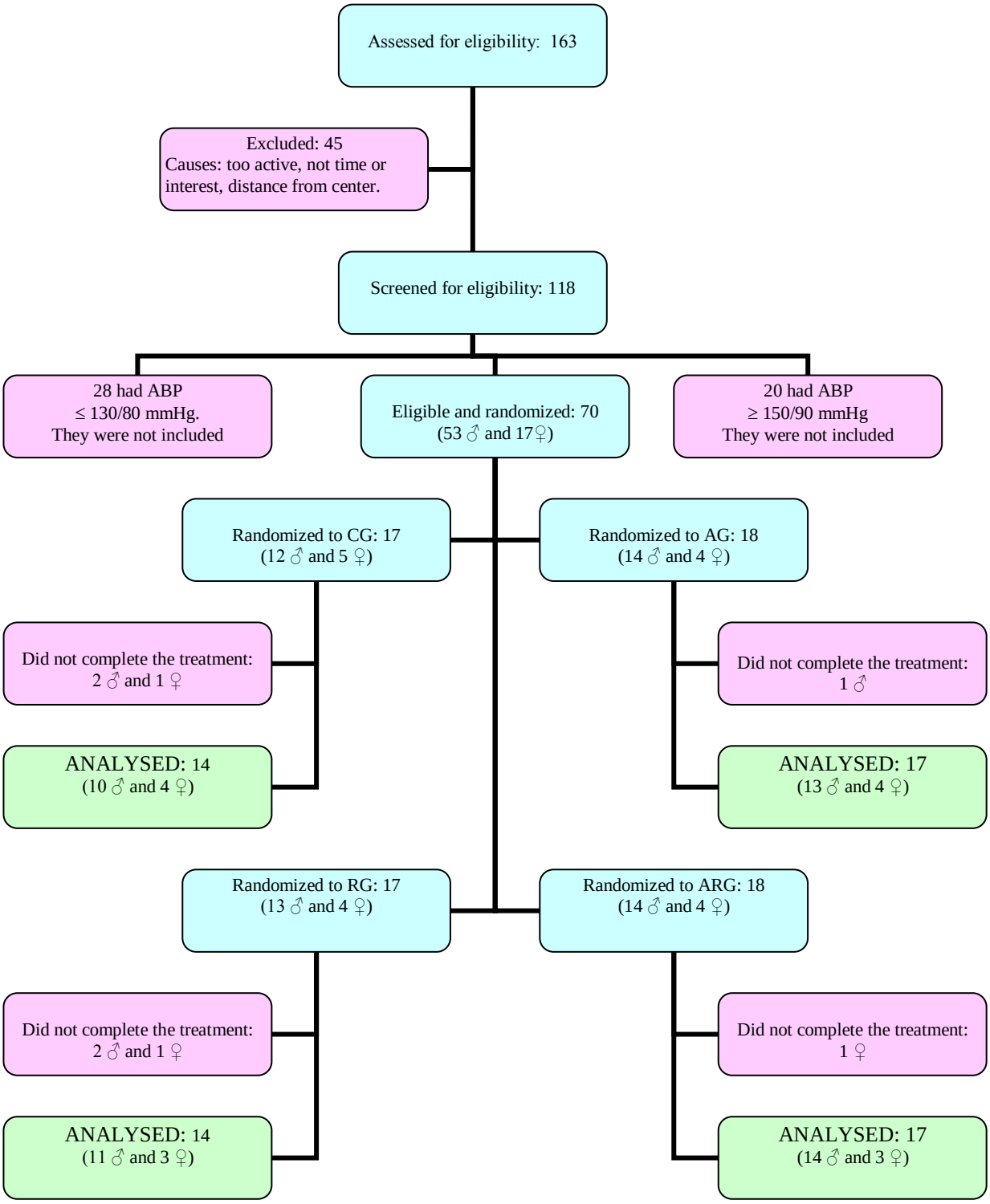

Figure 1 Diagram flow of participants throughout the study. Abbreviations and symbols are as follows: ABP=Ambulatory Blood Pressure; AG=aerobic training group; RG=resistance training group; ARG=aerobic and resistance training group; CG=control group; ♂= male and ♀= female.

2.2.2. Study design

After completing and signing the written informed consent, participants visited the Institute of Cardiology and Sport Medicine at the German Sport University (DSHS) during two consecutive days before and after 12-wk experimental interventions. During both visits the participants received a complete medical examination including blood samples, exercise capacity and hemodynamic values.

After evaluating and confirming the inclusion and exclusion criteria during the first two visits, the participants were randomly allocated to four experimental groups: (a) AG: aerobic training group, (b) RG: resistance training group, (c) ARG: aerobic + resistance training group and (d) CG: control group.

The training programs were carried out 3 times per week during 12-wk at the Fitness Center "Trainingsinstitut Prof. Dr. Baum", while the control group refrained for performing any exercise during the same period of time. During the experimental trials, subjects were repeatedly instructed to keep dietary, drinking and smoking habits as constant as possible. Following the 12-wk of intervention, all subjects were measured between 24- and 72-hr after the completion of the last exercise session. To avoid diurnal variation, all measurements were taken at the same time of day and in the same order as pre-measurements. The general study schedule is shown in Figure 2.

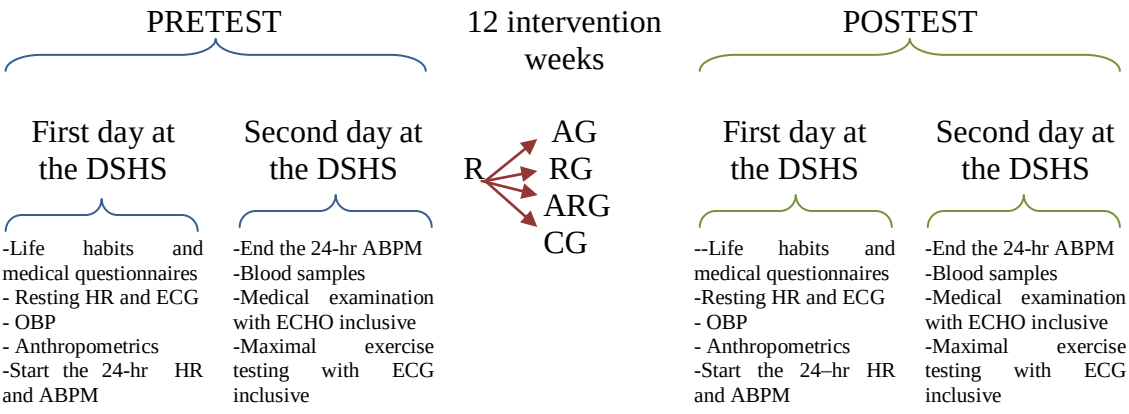

Figure 2 Chronic stage schedule. Abbreviations are as follows: HR=heart rate, ECG=electrocardiogram; OBP=office blood pressure; ABPM=ambulatory blood pressure monitoring; ECHO=echocardiogram; R= randomization; AG=aerobic group; RG= resistance group; ARG=aerobic + resistance group; CG=control group.

2.2.3. Protocol

2.2.3.1. Pretest and Posttest measurements

The following variables were measurement before and after the 12-wk intervention:

a. Health history and physical activity: the subjects were asked by questionnaires about their health history, drug consumption and physical activity habits.

b. Resting heart rate: was measured by a 12-lead electrocardiogram system (Mac 1200, GE Medical Systems, Freiburg, Germany) while the participants were resting quietly in a supine position for 5 min.

c. Anthropometrics: Volunteers were weighted (kg) on a balance scale (Seca, model 862, Hamburg, Germany). Height was measured to the nearest 0.5 cm using a stadiometer and body mass index (BMI) was calculated as weight (kg) per height^2 (m^2). Waist circumference (cm) was measured at the level midway between the lower rib margin and the iliac crest.

d. Office blood pressure (OBP): after a minimum of 5 min seated rest, the OBP was determined twice (M 5-1, Omron Healthcare, Japan), once in each arm. When the difference was greater than 10 mmHg, it was repeated in the arm, where the highest value was determined. The measurements were averaged for further analysis.

e. Ambulatory blood pressure and heart rate monitoring: during the first visit to the DSHS an ambulatory blood pressure monitor (Spacelabs, model 90217, Washington, USA) was attached to each person. It was programmed to take the ABP and heart rate measurements each 15 min from 6:00 hr until 22:00 hr and then each 30 min from 22:00 hr until 6:00 hr. Volunteers left the Institute with instructions to keep their arm extended and still when the monitor was recording, to proceed with their typical activities, not to engage in formal exercise, to record the activities performed during the day and to return the monitor the following day. If the awake averaged ABP was < 130/80 mmHg other >150/90 mmHg subjects were excluded from further participation.

f. Blood sampling: a 12-hr fasting venous blood sample was obtained for determination of glycated hemoglobin (HbA1c), glucose, total cholesterol, high-density lipoprotein (HDL) low-density lipoprotein (LDL), triglycerides, uric acid, creatinine and high sensitivity C reactive protein (hsCRP). Samples were drawn into EDTA vacutainer tubes, centrifuged for 10 min at 3400 rpm and 20°C (Rotanda 460R, Hettich, Tuttlingen, Germany) and sent to a biochemical laboratory for their analysis.

g. Medical examination: prior to engaging in the exercise program and with the aim to evaluate the exclusion criteria, all the participants received a complete medical examination. The cardiac function was examined by resting (Mac 1200, GE Medical Systems, Freiburg, Germany) and exercising 12-leads electrocardiogram (nSpire Health, model Zan 680, Oberthulba, Germany) and a two-dimensional echocardiogram (GE Medical Systems, Freiburg, Germany) with M-Mode, PW, CW, tissue Doppler, strain, strain rate and AFI including.

h. Maximal exercise testing:

Cardiopulmonary exercise test: A maximal oxygen uptake test with a simultaneously 12 leads electrocardiogram (nSpire Health, model Zan 680, Oberthulba, Germany) was performed by each participant. On a cycle-ergometer (Ergoline, Ergoscript 2012 EL, Bitz, Germany), subjects were asked to choose a pedaling rate between 60- and 65-rpm and to maintain that rate throughout the test. They began cycling at 25-W with an increase of 25-W every 2-min until exhaustion was reached. Rating of perceived exertion (RPE) on 15-point Borg scale, heart rate (HR), blood pressure and lactate concentration in capillary blood (Ebioplus, Eppendorf, Hamburg, Germany) were determined during the last 30-s of each workload. During the exercise blood pressure measurements, participants were instructed to keep their arm extended and relaxed on the handlebar to reduce measurement errors.

The oxygen and carbon dioxide sensors were calibrated using gases with known oxygen and carbon dioxide concentrations before each test. The flow sensors were also calibrated before each test using a 1-L syringe. Breath-by-breath gas exchange measurement allowed on line determination of ventilation (VE),

oxygen uptake (VO_2), carbon dioxide production (VCO_2) and respiratory exchange ratio (RER). Peak oxygen consumption (VO_2 peak) was expressed as the highest attained VO_2, during the final 30-s of exercise and physical work capacity (PWC) was described as maximal workload per weight (W/kg). Maximal effort was achieved when subjects met two of the following criteria: (a) a final RPE score of 17 or greater on the Borg Scale (rankings from 6 to 20), (b) an RER greater than 1.10 and/or (c) no change in HR following a change in workload.

Maximal torque of quadriceps femoris and biceps brachii: was isometrically tested with participants in an upright sitting position and the hip and knee joint other elbow joint flexed to 90°. A force transducer (Digimax, Mechatronic Ltd, Hamm, Germany) was used and the lever arm was calculated as the distance between knee or elbow joint space and contact point of force transduction. The best of three trials of each leg and each arm were taken for further computation.

2.2.3.2. Exercise training regime

After pretests, the training programs were carried out 3 times per week over a period of 12-wk. The exercise intervention was structured and supervised by an experienced exercise physiologist at the Fitness Center "Trainingsinstitut Prof. Dr. Baum".

While the CG did not exercise during the 12-wk, the participants in the AG, RG and ARG groups warmed- up by pedaling 5-min at 40% of their $HR_{reserve}$ (Karvonen et al., 1957) prior to engaging in the training session (see formula below).

$$HR_{exercise} = [(HR_{max} - HR_{rest}) \times \% \text{ Intensity}] + HR_{rest}$$

$HR_{exercise}$ formula: HR_{max} indicates the maximal HR during the cardiopulmonary exercise test and HR_{rest} the lowest Heart Rate during the resting ECG.

Following the warm- up, participants performed specific work, according to the group to which they were randomly assigned. Intensity and duration of the exercise program were progressively increased throughout the 12-wk program. Initially, the participants exercised at 50% intensity; followed by increments of 5% every 2-wk (see Table 6).

In order to individualize the exercise protocols for each person, the aerobic training intensity was calculated with the $HR_{exercise}$ formula (see formula above), while the resistance training intensity was determined during the first week of training with a test of one repetition maximum of each resistance exercise.

Aerobic training: was performed on a cycloergometer (Motion Cycle 400, Emotion Fitness, Hochspeyer, Germany) and the intensity was controlled by telemetry by heart rate monitors (Polar, model FS1, Kempele, Finland).

Resistance training: weight machines (Conex multiform, La Roque d'Anthéron, France) were used for strengthening muscle groups of the upper and lower body. In each session, the participants completed 2 circuits of 13 resistance exercises: leg extension, seated leg flexion, seated calf (heel) raise, seated erector spinal, seated abdominal, leg press, row, butterfly, lat pulldown, overheat shoulder press, seated chest press, biceps dumbbell curl and triceps dumbbell extension. Each exercise was repeated 10 times. Pauses between types of each exercise lasted about 30s. The strength training component lasted about 30 min. Participants were instructed to avoid the Valsalva maneuver during the exercises.

Aerobic and resistance training: participants performed both previously described treatments. About 1-hr was needed to complete a training session during the last two weeks.

Training Weeks	Aerobic training group:	Resistance training group:	Aerobic and resistance training group:
1-2	20 min, 50% $HR_{reserve}$	2 circuits, 10 rep, 50% 1RM	Both treatments
3-4	20 min, 55% $HR_{reserve}$	2 circuits, 10 rep, 55% 1RM	Both treatments
5-6	25 min, 60% $HR_{reserve}$	2 circuits, 12 rep, 60% 1RM	Both treatments
7-8	25 min, 65% $HR_{reserve}$	2 circuits, 12 rep, 65% 1RM	Both treatments
9-10	30 min, 70% $HR_{reserve}$	2 circuits, 15 rep, 70% 1RM	Both treatments
11-12	30 min, 75% $HR_{reserve}$	2 circuits, 15 rep, 75% 1RM	Both treatments

Table 6 Exercise protocols. Abbreviations are as follows: $HR_{reserve}$= reserve heart rate; 1RM= 1 repetition maximum; rep= repetitions.

2.2.4. Statistical Analysis

Descriptive statistics were obtained on all study parameters and the results are presented as mean ± standard deviation (SD), unless otherwise indicated. All analyses were computed with the Statistical Package for Social Sciences, version 15.0 (SPSS Inc., Chicago, Illinois, USA) and significance level was set at $p<0.05$.

One-way analyses of variance (ANOVA) were used to assess differences between initial values of the four groups on all the variables measured. In addition, the effect of the experimental treatments on the anthropometric characteristics, blood parameters, exercise capacity and hemodynamic values was examined by using two-way analysis of variance (ANOVA) (4 treatments x 2 measurement times) with repeated measures in one factor. Following a significant F ratio (Geisser-Greenhouse correction for the assumption of sphericity), significant interactions were analyzed using simple effects post-hoc analyses between time points within-groups.

In addition, variables were classified into four groups: hemodynamic values, anthropometric characteristics, blood parameters and exercise capacity. Pearson product-moment correlations were performed to determine whether changes in hemodynamic variables were related to changes in variables from other group. Figure 3 displays the variables analyzed in each group.

Finally, two Pearson product-moment correlations were computed to determine whether baseline systolic and diastolic blood pressures were related to changes on blood pressures following the 12-wk intervention program. Data from 14 participants of the control group were excluded for these correlations, yielding a total of 48 participants.

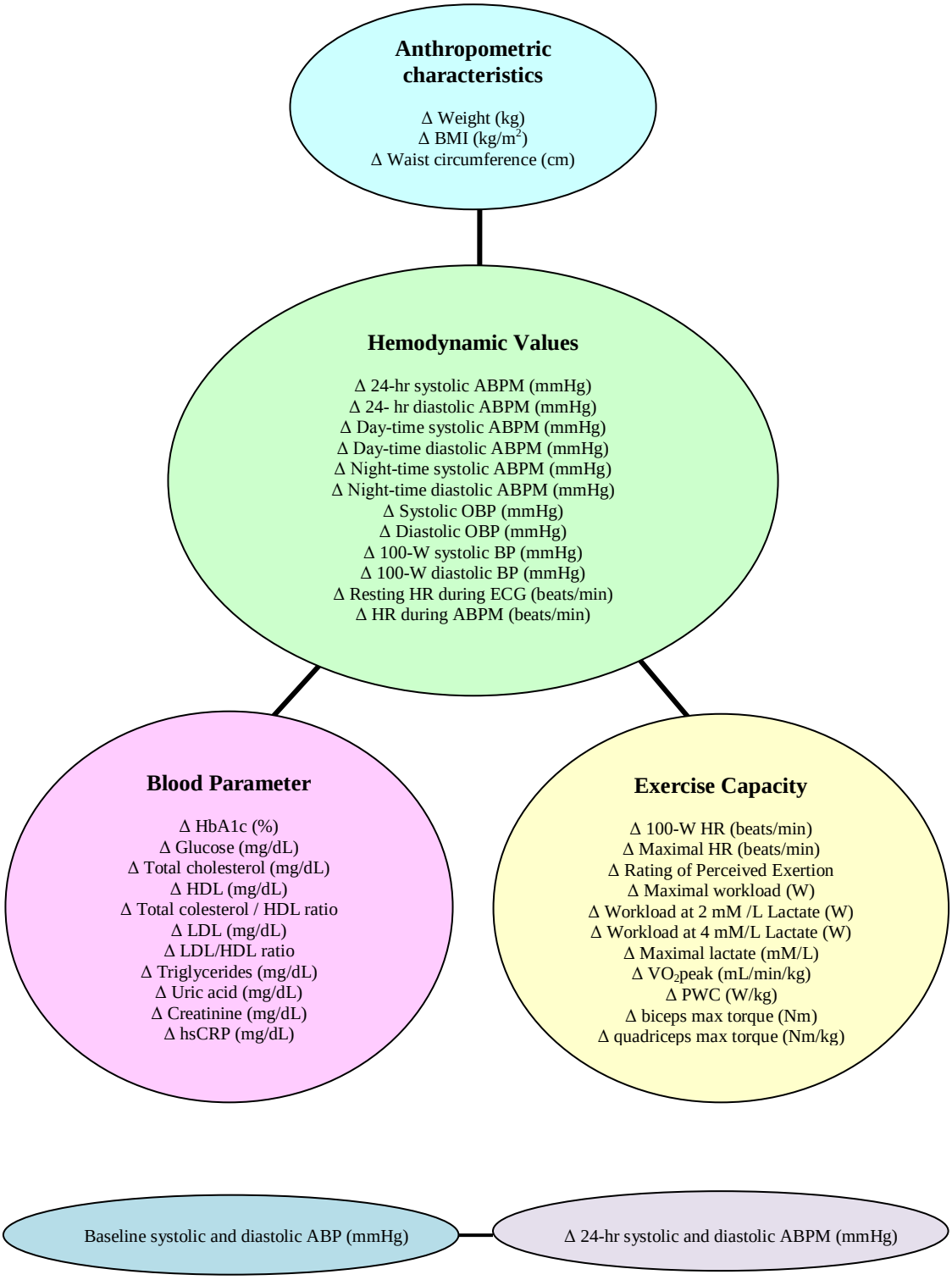

Figure 3 Variables analyzed with Pearson Correlations. Abbreviations and symbols are as follows: BMI=Body Mass Index; ABPM=Ambulatory Blood Pressure Monitoring; OBP= office blood pressure; BP=blood pressure; HR=heart rate; ECG=electrocardiogram; HbA1c=glycated hemoglobin; HDL=high density lipoprotein; LDL=low density lipoprotein; hsCRP=high sensitivity C reactive protein; VO_2=oxygen uptake; PWC=Physical work capacity; Δ=change (value obtained during the posttest minus value obtained during the pretest).

2.3. RESULTS

Between the months of April 2008 and September 2009, 62 randomized participants (48 males, 14 females) completed the treatments and were included in the final analyses. The mean group age was 54.5 ± 11.4 years, while the mean height was 178.6 ± 7.6 cm and 165.4 ± 7.1 for males and females, respectively.

Based on the 24-hr ABPM baseline measurements, 52 patients showed grade I hypertension (i.e., 24-hr ABPM > 130/80 mmHg) and 10 grade I isolated systolic hypertension (i.e. systolic pressure > 130 mmHg and diastolic pressure < 80 mmHg).

The health history questionnaire showed that six participants were current smokers (6.2%) and more than half (63%) reported a family history of cardiovascular diseases. Only one participant reported being diabetic and another ingested anti-hyperlipidemic medications. In both cases, the medication regimen was unaltered during the course of the 12-wk intervention study.

The one-way ANOVA detected no significant differences between the initial values of the four groups on all the variables measured.

2.3.1. Anthropometric characteristics

Significant interactions were found for BMI ($p = 0.01$) and waist circumference ($p = 0.02$) following the ANOVA procedure. Post-hoc analysis indicated a significant BMI and waist circumference reduction following AG and ARG interventions (Figure 4 and 5).

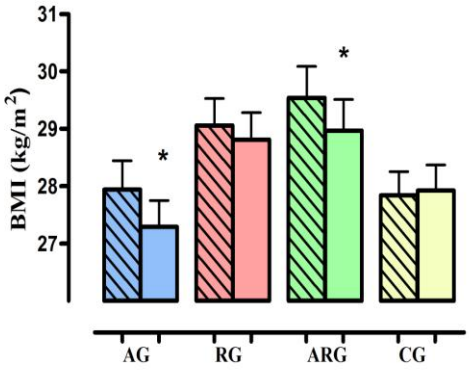

Figure 4 Participant's BMI before (shaded bars) **and after** (filled bars) **experimental interventions. ***p<0.05 from Pre-test.

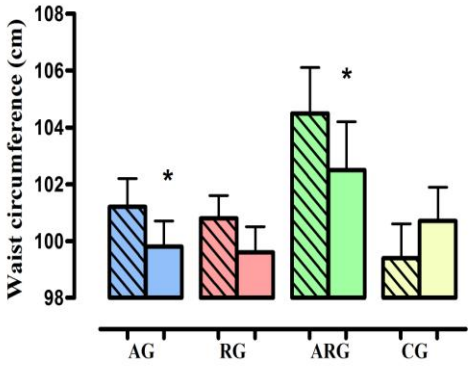

Figure 5 Participant's waist circumference before (shaded bars) **and after** (filled bars) **experimental interventions. ***p<0.05 from Pre-test

Summary

Descriptive and inferential statistic of anthropometric variable is shown in Table 7.

Anthropometric characteristics	**Test**	**AG (n = 17)**	**RG (n = 14)**	**ARG (n = 17)**	**CG (n = 14)**
Weight (kg)	Pre-test	87.0 ± 10.8	88.3 ± 11.3	92.7 ± 20.4	86.2 ± 14.3
	Post-test	85.4 ± 10.7	87.9 ± 12.2	91.2 ± 20.2	86.3 ± 14.9
Interaction p =0.06	Δ	- 1.6 ± 1.5	-0.4 ± 2.2	-1.5 ± 2.3	+0.1 ± 1.5
BMI (kg/m^2)	Pre-test	27.9 ± 4.0	29.1 ± 3.7	29.5 ± 4.3	27.8 ± 3.2
	Post-test	27.3 ± 3.6	28.8 ± 3.7	29.0 ± 4.3	27.9 ± 3.5
Interaction p =0.01	Δ	**-0.7 ± 0.7***	-0.3 ±0.7	**-0.5 ± 0.7***	+0.1 ± 0.5
Waist circumference (cm)	Pre-test	101.2 ± 7.9	100.8 ± 6.6	104.5 ± 13.0	99.4 ± 9.2
	Post-test	99.8 ± 7.0	99.6 ± 7.0	102.5 ± 13.5	100.7 ± 9.8
Interaction p =0.02	Δ	**-1.4 ± 2.8***	-1.2 ± 5.2	**-2.0 ± 2.6***	-1.3 ± 2.7

Table 7 Participant's anthropometric characteristics. Data is presented as means ± SD. Abbreviations are as follows: AG=aerobic training group, RG=resistance training group, ARG=aerobic and resistance training group; CG=control group; Δ=change. *****p < 0.05 from Pre-test

2.3.2. Blood Pressure

2.3.2.1. Ambulatory blood pressure

Systolic ambulatory blood pressure (sABPM) reductions were -3.3 ± 4.8, -4.9 ± 7.7 and -5.8 ± 7.2 mmHg following aerobic training alone, resistance training alone and the combination of aerobic and resistance exercise training, respectively (Table 8). A significant interaction was found on the 24-hr sABPM ($p = 0.03$). Post-hoc analyses showed that the significant reductions were following AG, RG and ARG interventions, whereas the sABPM of the control group remained unchanged (Figure 6).

A trend towards reduction on the 24-hr diastolic ABPM was found after three exercise interventions compared to control group (Table 8); however, this change did not reach statistical significance (Figure 7).

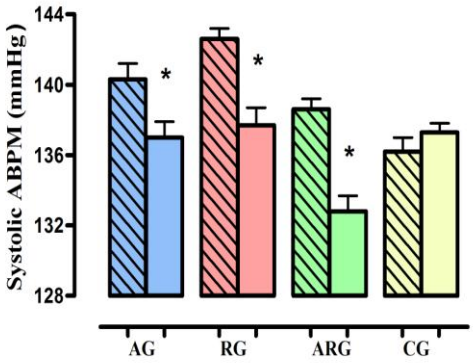

Figure 6 Comparisons of 24-hr systolic ABPM before (shaded bars) **and after** (filled bars) **interventions. ***$p<0.05$ from Pre-test.

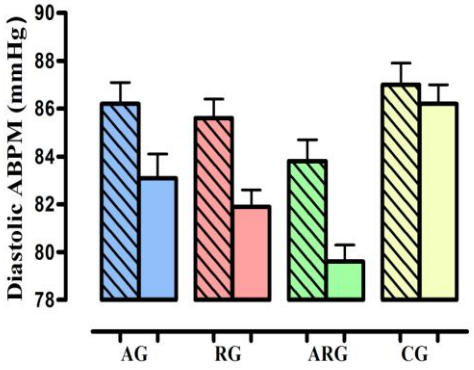

Figure 7 Comparisons of 24-hr diastolic ABPM before (shaded bars) **and after** (filled bars) **interventions.**

Figures 8 and 9 show systolic and diastolic day-time ABPM results. Although there was no significant interactions found ($p = 0.07$ and $p = 0.23$ respectively), the systolic and diastolic day-time ABPM reduction following the three exercise interventions reached between 4.0 and 6.4 mmHg (Table 8), whereas the values of the control group remained unchanged.

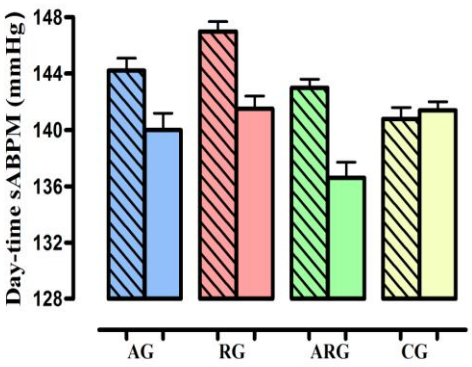

Figure 8 Comparisons of day-time systolic ABPM before (shaded bars) **and after** (filled bars) **interventions.**

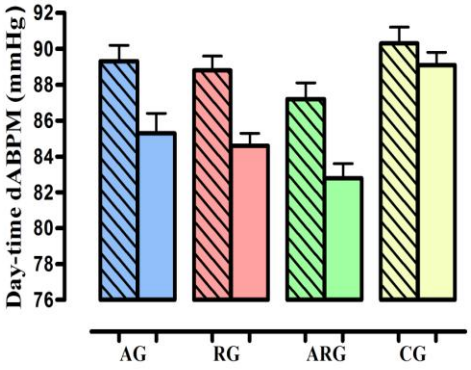

Figure 9 Comparisons of day-time diastolic ABPM before (shaded bars) **and after** (filled bars) **interventions.**

During the night-time sABPM was found a significant interaction (p = 0.04). Post hoc analyses found only a significant blood pressure decrease of 3.9 mmHg following the ARG treatment, while the BP behavior following the others interventions tended to: decrease after the resistance training, to stay the same after aerobic training and to increase after the control condition (Figure 10). Although there was no significant interaction during the night-time dABPM (p = 0.15) following the RG and ARG interventions the dABPM tended to decrease, whereas the BP after AG and CG remained unchanged (Figure 11).

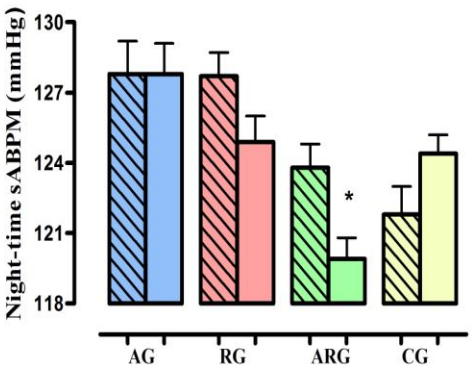

Figure 10 Comparisons of night-time systolic ABPM before (shaded bars) **and after** (filled bars) **interventions. ***p<0.05 from Pre-test.

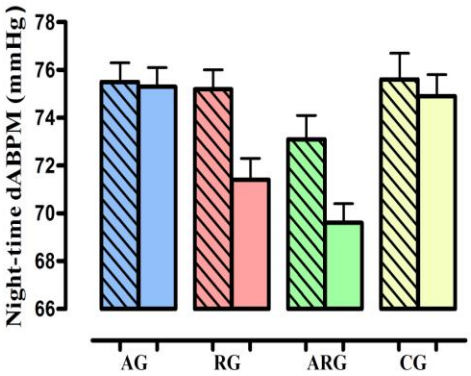

Figure 11 Comparisons of night-time diastolic ABPM before (shaded bars) **and after** (filled bars) **interventions.**

2.3.2.2. Office blood pressure

A trend towards reduction in both, systolic and diastolic OBP (sOBP and dOBP respectively) measured in the arm were found following AG, RG and ARG interventions (Table 8 and Figures 12 and 13); however, ANOVA interactions did not reach statistical significance (p = 0.07).

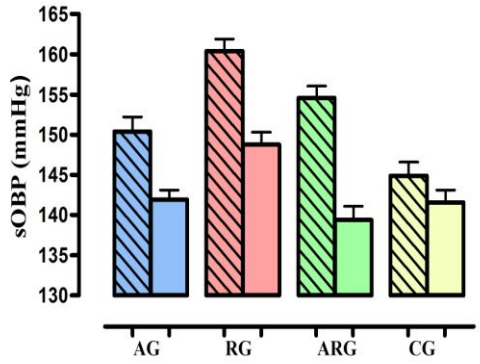

Figure 12 Comparisons of systolic OBP before (shaded bars) **and after** (filled bars) **interventions.**

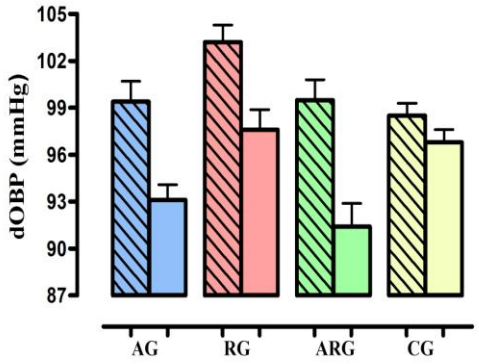

Figure 13 Comparisons of diastolic OBP before (shaded bars) **and after** (filled bars) **interventions.**

2.3.2.3. 100-W Blood pressure

During the maximal exercise test, BP was measured at 100-W. Greater reductions in systolic and diastolic BP were detected following the exercise interventions (Table 8). Although no significant interaction was found on the systolic blood pressure during the exercise test (p = 0.08), diastolic blood pressure values were significantly reduced (p = 0.01) following the RG and ARG treatments (Figures 14 and 15).

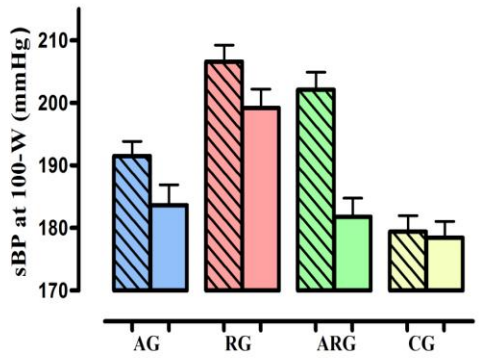

Figure 14 Comparisons of systolic BP at 100-W before (shaded bars) **and after** (filled bars) **interventions.**

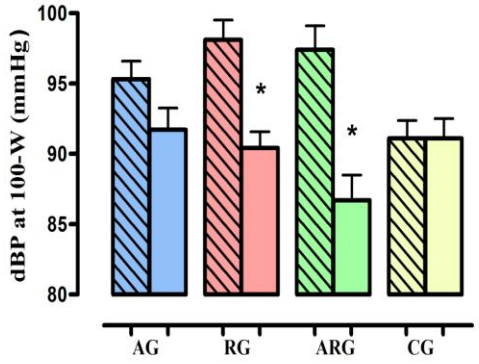

Figure 15 Comparisons of diastolic BP at 100-W before (shaded bars) **and after** (filled bars) **interventions. ***p<0.05 from Pre-test.

Summary

Descriptive and inferential statistics for the 24-hr ambulatory blood pressure monitoring, office blood pressure and 100-W blood pressure are presented in Table 8.

Blood pressure	**Test**	**AG (n = 17)**	**RG (n = 14)**	**ARG (n = 17)**	**CG (n = 14)**
24-hr systolic ABPM (mmHg). *Interaction p = 0.03*	Pre-test	140.3 ± 7.02	142.6 ± 5.1	138.6 ± 4.6	136.2 ± 6.7
	Post-test	137.0 ± 8.8	137.7 ± 7.7	132.8 ± 7.3	137.3 ± 4.3
	Δ	**-3.3 ± 4.8***	**-4.9 ± 7.7***	**-5.8 ± 7.2***	+1.1 ± 7.1
24- hr diastolic ABPM (mmHg) *Interaction p =0.16*	Pre-test	86.2 ± 6.8	85.6 ± 6.2	83.8 ± 6.7	87.0 ± 7.1
	Post-test	83.1 ± 7.7	81.9 ± 5.9	79.6 ± 5.9	86.2 ± 6.2
	Δ	-3.1 ± 3.4	-3.7 ± 4.4	-4.2 ± 4.9	-0.8 ± 4.9
Day-time systolic ABPM (mmHg). *Interaction p =0.07*	Pre-test	144.2 ± 6.8	147.0 ± 5.4	143.0 ± 4.8	140.8 ± 6.6
	Post-test	139.7 ± 9.2	141.5 ± 7.4	136.6 ± 8.6	141.4 ± 4.7
	Δ	-4.5 ± 6.1	-5.5 ± 7.9	-6.4 ± 8.7	+0.6 ± 7.7
Day-time diastolic ABPM (mmHg) *Interaction p =0.23*	Pre-test	89.3 ± 7.5	88.8 ± 6.5	87.2 ± 6.9	90.3 ± 7.2
	Post-test	85.3 ± 8.3	84.6 ± 5.7	82.8 ± 6.6	89.1 ± 5.5
	Δ	-4 ± 4.5	-4.2 ± 5.2	-4.4 ± 4.8	-1.2 ± 4.5
Night-time systolic ABPM (mmHg). *Interaction p =0.04*	Pre-test	127.8 ± 11.0	127.7 ± 7.6	123.8 ± 7.6	121.8 ± 9.4
	Post-test	127.8 ± 9.9	124.9 ± 8.3	119.9 ± 6.9	124.4 ± 6.0
	Δ	0 ± 4.8	-2.8 ±7.5	**-3.9 ± 7.4***	+2.6 ± 6.9
Night-time diastolic ABPM (mmHg) *Interaction p =0.15*	Pre-test	75.5 ± 6.2	75.2 ± 6.2	73.1 ± 8.0	75.6 ± 8.9
	Post-test	75.3 ± 6.3	71.4 ± 6.9	69.6 ± 6.2	74.9 ± 7.4
	Δ	-0.2 ± 3.2	-3.8 ± 4.5	-3.5 ± 7.8	-0.7 ± 5.4
Systolic OBP (mmHg) *Interaction p =0.07*	Pre-test	150.4 ± 14.4	160.4 ± 11.9	154.6 ± 11.45	144.9 ± 13.5
	Post-test	141.4 ± 9.1	148.8 ± 11.8	139.4 ± 13.3	141.6 ± 11.6
	Δ	-9 ± 9.8	-11.6 ± 14.2	-15.2 ± 13.6	-3.3 ± 11.8
Diastolic OBP (mmHg) *Interaction p =0.07*	Pre-test	99.4 ± 9.9	103.2 ± 8.5	99.5 ± 10.3	98.5 ± 7.6
	Post-test	93.1 ± 7.7	97.6 ± 10.0	91.4 ± 11.4	96.8 ± 5.9
	Δ	-6.3 ± 4.9	-5.6 ± 7.8	-8.1 ± 7.6	-1.7 ± 6.1
100-W systolic BP (mmHg) *Interaction p =0.08*	Pre-test	191.5 ± 18.7	206.6 ± 20.7	202.1 ± 22.1	179.4 ± 20.2
	Post-test	183.6 ± 26.1	199.2 ± 23.8	181.8 ± 23.3	178.4 ± 21.0
	Δ	-7.9 ± 17.4	-7.4 ± 19.5	-20.3 ± 27.9	-1 ± 14.7
100-W diastolic BP (mmHg) *Interaction p =0.01*	Pre-test	95.3 ± 10.1	98.1 ± 11.1	97.4 ± 13.4	91.1 ± 9.9
	Post-test	91.7 ± 12.2	90.4 ± 9.3	86.7 ± 14.1	91.1 ± 11.0
	Δ	-3.6 ± 8.8	**-7.7 ± 12.9***	**-10.7 ± 7.11***	0 ± 7.7

Table 8 Blood pressure values. Data is presented as means ± SD. Abbreviations are as follows: AG=aerobic training group, RG=resistance training group, ARG=aerobic and resistance training group, CG=control group, ABPM= ambulatory blood pressure monitoring, OBP=office blood pressure, BP= blood pressure; Δ=change. *****$p < 0.05$ from Pre-test

2.3.3. Resting and ambulatory heart rate

No significant interactions were found between the intervention treatments and measurement times on the resting heart rate (p = 0.62). Nevertheless, the 24-hr heart rate showed a significant interaction (p = 0.04) and the post-hoc analysis determined significant heart rate decreases following AG and ARG treatments (Figures 16 and 17).

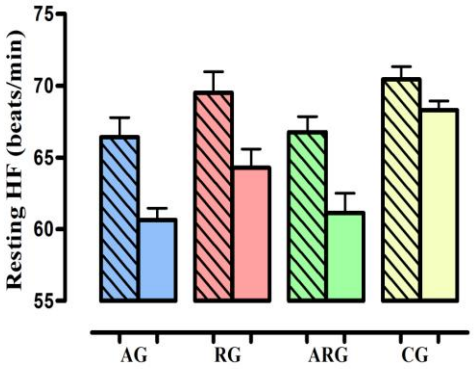

Figure 16 Comparisons of resting HR before (shaded bars) **and after** (filled bars) **interventions.**

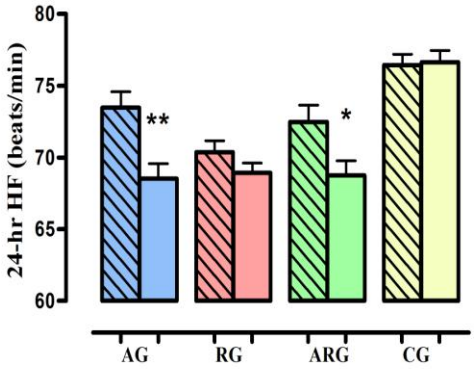

Figure 17 Comparisons of 24-hr HR before (shaded bars) **and after** (filled bars) **interventions.** ***p<0.05 from Pre-test.

Summary

Descriptive and inferential statistics for the resting heart rate during ECG and heart rate during ABPM are presented in Table 9.

Variable	**Test**	**AG (n = 17)**	**RG (n = 14)**	**ARG (n = 17)**	**CG (n = 14)**
Resting heart rate during ECG (beats/min)	Pre-test	66.4 ± 10.9	69.5 ± 11.6	66.8 ± 8.6	70.4 ± 7.0
	Post-test	60.7 ± 6.6	64.3 ± 10.3	61.1 ± 10.9	68.3 ± 5.1
Interaction p =0.62	Δ	-5.7 ± 8.9	-5.2 ± 6.6	-5.7 ± 9.7	-2.1 ± 8.2
Heart rate during ABPM (beats/min)	Pre-test	73.5 ± 8.8	70.4 ± 6.5	72.5 ± 9.3	76.4 ± 6.1
	Post-test	68.5 ± 8.2	68.9 ± 5.4	68.8 ± 7.9	76.6 ± 6.4
Interaction p =0.04	Δ	**-5.0 ± 6.1***	-1.5 ±5.3	**-3.7 ± 5.3***	+0.2 ± 4.2

Table 9 Resting and ambulatory heart rate. Data is presented as means ± SD. Abbreviations are as follows: ECG= electrocardiogram, ABPM=ambulatory blood pressure monitoring, AG=aerobic training group, RG=resistance training group, ARG=aerobic and resistance training group; CG=control group; Δ=change. *p < 0.05 from Pre-test.

2.3.4. Blood parameters

ANOVA results found a significant interaction for HDL (p = 0.01). Post- hoc analysis indicated a significant HDL reduction only after the RG intervention.

Summary

Descriptive and inferential statistics for the blood parameter results are summarized in Table 10.

Blood parameters	**Test**	**AG (n = 17)**	**RG (n = 14)**	**ARG (n = 17)**	**CG (n = 14)**
HbA1c (%)	Pre-test	5.3 ± 0.4	5.6 ± 0.4	5.8 ± 0.8	5.4 ± 0.4
	Post-test	5.4 ± 0.3	5.7 ± 0.3	5.7 ± 0.6	5.5 ± 0.3
Interaction p =0.24	Δ	+0.1 ± 0.2	+0.1 ± 2	-0.1 ± 0.3	+0.1 ± 0.2
Glucose (mg/dL)	Pre-test	86.4 ± 12.8	91.2 ± 16.4	97.1 ± 30.8	86.1 ± 10.1
	Post-test	96.9 ± 10.9	94.1 ± 13.7	96.7 ± 31.2	89.4 ± 14.3
Interaction p =0.20	Δ	+10.5 ± 12.8	+2.9 ± 14.1	-0.4± 14.5	+3.3 ±17.1
Total cholesterol (mg/dL)	Pre-test	224.3 ± 46.7	242.6 ± 32.9	228.6 ± 29.9	237.4 ± 47.7
	Post-test	229.6 ± 43.3	227.3 ± 24.1	230.5 ± 40.2	229.0 ± 34.7
Interaction p =0.10	Δ	+5.3 ± 23.5	-15.4 ± 20.4	+1.9 ± 29.3	-8.4 ± 24.4
HDL(mg/dL)	Pre-test	49.7 ± 13.67	59.2 ± 10.3	52.6 ± 11.5	52.7 ± 14.2
	Post-test	50.7 ± 11.7	55.4 ± 8.9	54.1 ± 11.0	50.8 ± 12.8
Interaction p =0.01	Δ	+1 ± 4.6	**-3.78 ± 4.7**	+1.52 ± 6.1	-1.91 ± 3.6
Total cholesterol / HDL ratio	Pre-test	4.7 ± 1.2	4.3 ± 1.1	4.5 ± 0.9	4.7 ± 1.2
	Post-test	4.7 ± 1.0	4.2 ± 0.9	4.4 ± 0.9	4.7 ± 1.1
Interaction p =0.88	Δ	0 ± 0.7	-0.1 ± 0.3	-0.1 ± 0.3	0 ± 0.5
LDL (mg/dL)	Pre-test	143.6 ± 37.1	153.3 ± 29.2	146.0 ± 22.2	149.5 ± 31.5
	Post-test	143.7 ± 30.7	140.3 ± 25.5	143.1 ± 28.5	143.9 ± 25.4
Interaction p =0.26	Δ	+0.1 ± 4.6	-13.0 ± 4.7	-2.9 ± 6.1	-5.6 ± 3.6
LDL/HDL ratio	Pre-test	3.0 ± 0.9	2.7 ± 0.9	2.9 ± 0.6	2.9 ± 0.8
	Post-test	2.9 ± 0.8	2.6 ± 0.8	2.7 ± 0.7	3.0 ± 0.8
Interaction p =0.84	Δ	-0.1 ± 0.5	-0.1 ± 0.2	-0.2 ± 0.3	+0.1 ±0.4
Triglycerides (mg/dL)	Pre-test	183.6 ± 100.9	160.2 ± 58.8	132.3 ± 46.6	161.2 ± 85.6
	Post-test	147.4 ± 46.2	143.7 ± 52.2	134.2 ± 58.4	176.8 ± 101.8
Interaction p =0.84	Δ	-36.2 ± 101.4	-16.5 ± 46.3	+1.9 ± 28.9	+15.6 ± 62.5
Uric acid (mg /dL)	Pre-test	5.7 ± 1.2	5.6 ± 0.9	6.2 ± 1.1	5.3 ± 1.2
	Post-test	5.8 ± 1.3	6.0 ± 1.0	6.3 ± 1.2	5.5 ± 1.4
Interaction p =0.78	Δ	+0.1 ± 0.7	+0.4 ± 0.4	+0.1 ± 0.7	+0.2 ± 0.7

Blood parameters	Test	AG (n = 17)	RG (n = 14)	ARG (n = 17)	CG (n = 14)
Creatinine (mg/dL) *Interaction p =0.66*	Pre-test	1.0 ± 0.1	1.0 ± 0.2	1.0 ± 0.1	1.0± 0.1
	Post-test	1.0 ± 0.1	1.1 ± 0.2	1.0 ± 0.1	1.0 ± 0.1
	Δ	0 ± 0.1	+0.1 ± 0.1	0 ± 0.1	0 ± 0.1
hsCRP (mg/dL) *Interaction p =0.33*	Pre-test	0.1 ± 0.1	0.3 ± 0.3	0.2 ± 0.1	0.1 ± 0.1
	Post-test	0.2 ± 0.4	0.3 ± 0.2	0.2 ± 0.1	0.3 ± 0.2
	Δ	+0.1 ± 0.4	0 ± 0.4	0 ± 0.1	+0.1 ± 0.2

Table 10 Participant's blood parameters. Data is presented as means ± SD. Abbreviations are as follows: AG=aerobic training group; RG=resistance training group; ARG=aerobic and resistance training group; CG=control group; Δ=change; HbA1c=glycated hemoglobin; HDL=high-density lipoprotein; LDL=low-density lipoprotein; and hsCRP= high-sensitivity C reactive protein. *****$p<0.05$ from Pre-test.

2.3.5. Exercise capacity

2.3.5.1. Cardiopulmonary exercise test

No significant interactions were found for maximal heart rate ($p = 0.17$), maximal lactate ($p = 0.07$) and rating of perceived exertion ($p = 0.98$; Figure 18). However, ANOVA indicated significant interactions in maximal workload ($p \leq 0.001$), workload achieved at 2 ($p = 0.003$) and 4 mM/L lactate ($p \leq 0.001$), peak oxygen consumption ($p = 0.004$) and physical work capacity ($p \leq 0.001$).

Post-hoc analysis determined a significant increase in maximal workload after exercise interventions. This increase was particularly stronger after AG and ARG, whereas the values of the untrained group did not differ significantly (Figure 19).

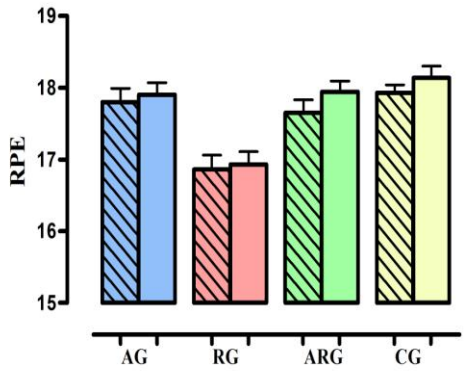

Figure 18 Comparisons of rating of perceived exertion before (shaded bars) **and after** (filled bars) **interventions.**

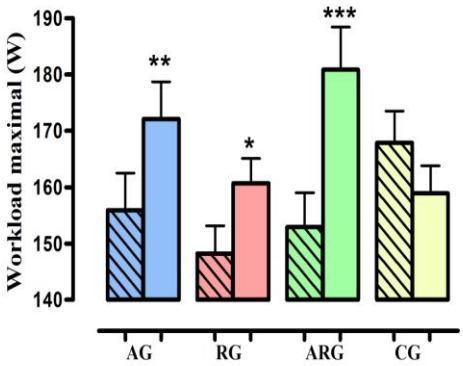

Figure 19 Comparisons of maximal workload before (shaded bars) **and after** (filled bars) **interventions.** ***p<0.05, ****p<0.01 and *****p<0.001 from Pre-test.

Participants in the exercise groups achieved the 2 mM/L lactate concentration during the post-test at a higher workload compared to their pre-test values. Participants in the control group did not have a significant improvement (Figure 20). Similar results were obtained on the workload at 4 mM/L lactate concentration; however, the increase of 7-W following resistance exercises did not reach statistical significance (Figure 21).

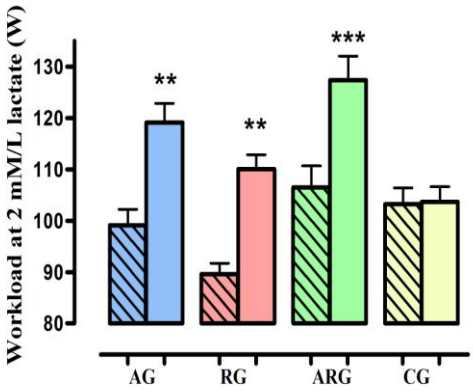

Figure 20 Comparisons workload at 2 mM/L lactate concentration before (shaded bars) **and after** (filled bars) **interventions. ****p<0.01 and *******p<0.001 from Pre-test.

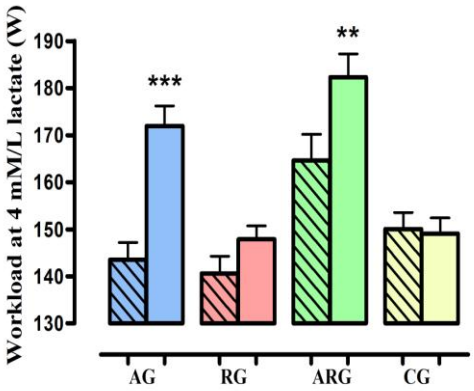

Figure 21 Comparisons workload at 4 mM/L lactate concentration before (shaded bars) **and after** (filled bars) **interventions. ****p<0.01 and *******p<0.001 from Pre-test.

A significant increase on peak oxygen consumption was found after the three exercise interventions as opposed to the control group (Table 11; Figure 22).

Finally the physical work capacity showed a significant increase following AG, RG and ARG, while the values of the CG had a significant reduction after the 12-wk interventions (Figure 23).

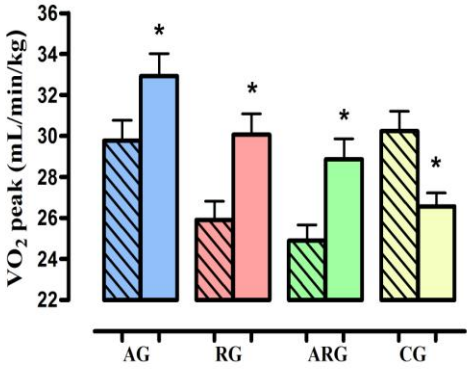

Figure 22 Peak oxygen consumption before (shaded bars) **and after** (filled bars) **interventions. ***p<0.05 from Pre-test**.**

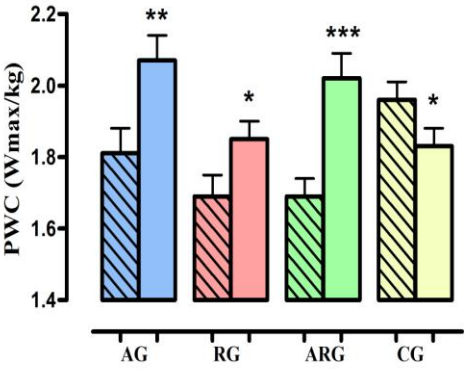

Figure 23 Physical work capacity before (shaded bars) **and after** (filled bars) **interventions. ***p<0.05, ******p<0.01 and *******p<0.001 from Pre-test.

Summary

Maximal oxygen uptake test results with a simultaneously 12-leads electrocardiogram are showed in Table 11.

Variables	Test	AG (n = 17)	RG (n = 14)	ARG (n = 17)	CG (n = 14)
100-W HR (beats/min) *Interaction p =0.08*	Pre-test	130.8 ± 15.0	133.2 ± 19.3	122.4 ± 13.5	126.7 ± 15.9
	Post-test	122.5 ± 16.0	124.5 ± 13.7	118.0 ± 13.7	125.9 ± 16.3
	Δ	-8.3 ± 7.3	-8.7 ± 12.9	-4.4 ± 8.4	-0.8 ± 7.7
Maximal HR (beats/min) *Interaction p =0.17*	Pre-test	159.6 ± 19.9	152.1 ± 16.7	155.2 ± 18.7	161.4 ± 17.0
	Post-test	155.0 ± 17.7	151.4 ± 13.5	158.2 ± 20.9	156.9 ± 15.4
	Δ	-4.6 ± 5.7	-0.7 ± 5.5	+3 ± 7.8	-4.5 ± 8.1
RPE *Interaction p =0.98*	Pre-test	17.8 ± 1.5	16.9 ± 1.6	17.6 ± 1.4	17.9 ± 0.9
	Post-test	17.9 ± 1.4	16.9 ± 1.1	17.9 ± 1.2	18.1 ± 1.2
	Δ	+0.1 ± 1.6	0 ± 1.9	+0.3 ± 1.8	+0.2 ± 0.9
Maximal workload (W) *Interaction p ≤0.001*	Pre-test	155.9 ± 51.9	148.2 ± 38.6	152.9 ± 48.3	167.9 ± 44.3
	Post-test	172.1 ± 52.2	160.7 ± 35.0	180.9 ± 59.0	158.9 ± 38.7
	Δ	**+16.2 ± 17****	**+12.5 ± 16***	**+28 ± 20****	-9 ± 19
Workload at 2 mM /L Lactate (W) *Interaction p =0.003*	Pre-test	99.1 ± 24.6	89.6 ± 16.5	106.5 ± 33.4	103.2 ± 25.4
	Post-test	119.1 ± 29.7	110.0 ± 22.6	127.4 ± 36.7	103.6 ± 23.7
	Δ	**+20.1 ± 19****	**+20.4 ± 18****	**+20.9 ± 15****	-0.4 ±14
Workload at 4 mM/L Lactate (W) *Interaction p ≤0.001*	Pre-test	143.5 ± 29.0	140.6 ± 28.7	164.6 ± 43.6	150.0 ± 28.0
	Post-test	171.9 ± 33.8	147.8 ± 22.9	182.3 ± 38.6	149.1 ± 26.0
	Δ	**+28.4 ±19*****	+7.2 ± 18	**+17.9 ± 16****	-0.9 ± 12
Maximal lactate (mM/L) *Interaction p =0.07*	Pre-test	5.7 ± 2.2	5.1 ± 1.4	4.8 ± 1.4	5.6 ± 1.6
	Post-test	5.5 ± 2.0	5.1 ± 1.4	5.6 ± 1.6	5.0 ± 1.6
	Δ	-0.2 ± 1.5	0 ± 1.5	+0.8 ± 1.4	-0.6 ± 1.3
VO_2peak (mL/min/kg) *Interaction p =0.001*	Pre-test	29.8 ± 7.9	25.9 ± 7.1	24.9 ± 6.0	30.2 ± 7.6
	Post-test	32.9 ± 8.7	30.1 ± 8.0	28.9 ± 8.0	26.6 ± 5.2
	Δ	**+3.1 ± 7.9***	**+4.2 ± 5.3***	**+4.0 ± 4.4***	**-3.7 ± 6.4***
PWC (W/kg) *Interaction p ≤ 0.001*	Pre-test	1.8 ± 0.5	1.7 ± 0.4	1.7 ± 0.4	2.0 ± 0.4
	Post-test	2.1 ± 0.6	1.9 ± 0.4	2.0 ± 0.6	1.8 ± 0.4
	Δ	**+0.3 ± 0.3****	**+0.2 ± 0.2***	**+0.3 ± 0.2*****	**-0.2 ± 0.2***

Table 11 Cardiopulmonar exercise test results. Data is presented as means ± SD. Abbreviations are as follows: AG=aerobic training group; RG=resistance training group, ARG=aerobic and resistance training group; CG=control group; Δ=change; HR=heart rate; RPE=rating of perceive exertion; VO_2peak=highest attained oxygen consumption; and PWC=physical work capacity. *****p<0.05, **p<0.01 and ***p<0.001 from Pre-test.

2.3.5.2 Maximal isometric torque

Although non-statistically significant interaction was found, ANOVA results indicate a trend to higher biceps muscle strength following RG and ARG interventions (Figures 24-27).

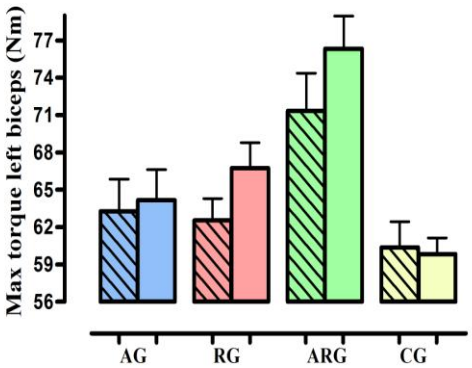

Figure 24 Comparison of maximal isometric torque of the left biceps before (shaded bars) **and after** (filled bars) **interventions.**

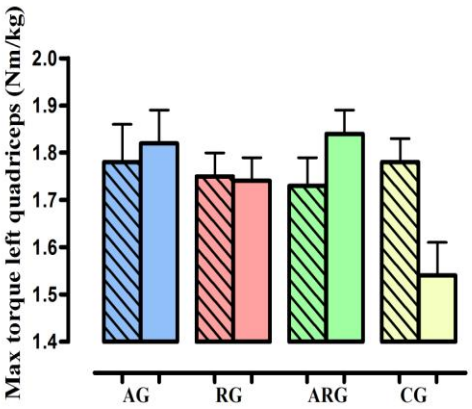

Figure 25 Comparison of relative maximal isometric torque of the left quadriceps before (shaded bars) **and after** (filled bars) **interventions.**

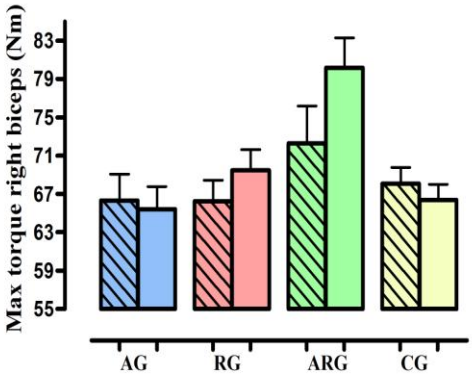

Figure 26 Comparison of maximal isometric torque of the right biceps before (shaded bars) **and after** (filled bars) **interventions.**

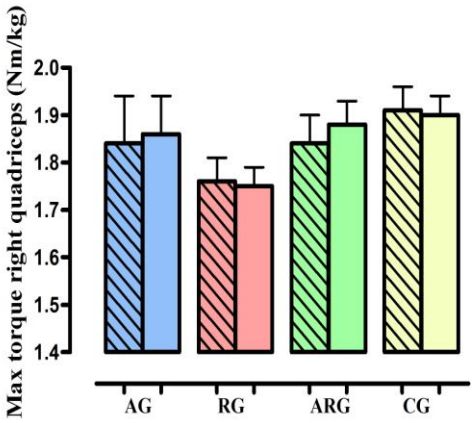

Figure 27 Comparison of relative maximal isometric torque of the right quadriceps before (shaded bars) **and after** (filled bars) **interventions.**

Summary

Maximal biceps and quadriceps muscle torque before and after the 12-wk intervention is summarized in Table 12.

Muscle	Test	AG (n = 17)	RG (n = 14)	ARG (n = 17)	CG (n = 14)
Left biceps max torque (Nm)	Pre-test	63.3 ± 20.4	62.5 ± 14.0	71.4 ± 23.8	60.4 ± 16.1
	Post-test	64.2 ± 19.3	66.7 ± 16.0	76.3 ± 20.7	59.9 ± 10.1
Interaction p =0.21	Δ	+0.9 ± 8.2	+4.2 ± 5.7	+4.9 ± 8.5	-0.5 ± 7.7
Left quadriceps max torque (Nm/kg)	Pre-test	1.8 ±0.7	1.7 ± 0.4	1.7 ± 0.5	1.8 ± 0.4
	Post-test	1.8 ± 0.6	1.7 ± 0.4	1.8 ± 0.4	1.5 ± 0.6
Interaction p =0.09	Δ	0 ± 0.2	0 ± 0.2	+0.1 ± 0.2	-0.2 ± 0.7
Right biceps max torque (Nm)	Pre-test	66.3 ± 21.8	66.3 ± 17.1	72.3 ± 30.7	68.1 ± 13.5
	Post-test	65.4 ± 18.4	69.5 ± 17.0	80.2 ± 24.7	66.4 ± 12.5
Interaction p =0.06	Δ	-0.9 ± 9.4	+3.2 ± 8.6	+7.9 ± 14.0	-1.7 ± 2.2
Right quadriceps max torque (Nm/kg)	Pre-test	1.8 ± 0.8	1.8 ± 0.4	1.8 ± 0.5	1.9 ± 0.4
	Post-test	1.9 ± 0.7	1.8 ± 0.4	1.9 ± 0.4	1.9 ± 0.3
Interaction p =0.84	Δ	+0.1 ± 0.2	0 ± 0.2	+0.1 ± 0.2	0 ± 0.2

Table 12 Maximal torque for biceps and quadriceps muscles. Data is presented as means ± SD. Abbreviations are as follows: AG=aerobic training group; RG=resistance training group; ARG=aerobic and resistance training group; CG=control group; Δ=change.

2.3.6. Correlation results

Table 8 shows the significant Pearson product-moment correlations detected in this study. In general, we found: (1) positive correlations between anthropometric characteristics (weight and BMI) and BP; (2) positive correlations between blood parameters (HbA1c, LDL/HDL ratio and triglycerides) and BP and (3) negative correlations between exercise capacity (maximal workload, lactate, VO_2peak and PWC) and BP.

Groups	Variable A	Variable B	r	P
Anthropometric characteristics and hemodynamic values	↓ Δ weight	↓ Δ systolic 24-hr ABP	0.34	0.008
		↓ Δ systolic day ABP	0.38	0.002
		↓ Δ diastolic OBP	0.41	0.001
		↓ Δ 100-W diastolic BP	0.41	0.001
	↓ Δ BMI	↓ Δ systolic 24-hr ABP	0.35	0.005
		↓ Δ diastolic 24-hr ABP	0.26	0.049
		↓ Δ systolic day time ABP	0.33	0.010
		↓ Δ diastolic OBP	0.33	0.008
		↓ Δ 100-W diastolic BP	0.33	0.008
Blood parameters and hemodynamic values	↓ Δ HbA1c	↓ Δ systolic OBP	0.36	0.004
		↓ Δ diastolic OBP	0.27	0.034
		↓ Δ 100-W systolic BP	0.37	0.004
		↓ Δ 100-W diastolic BP	0.27	0.034
	↓ Δ LDL / HDL ratio	↓ Δ diastolic OBP	0.29	0.023
		↓ Δ 100-W diastolic BP	0.29	0.023
	↓ Δ Triglycerides	↓ Δ diastolic OBP	0.38	0.003
		↓ Δ 100-W diastolic BP	0.37	0.003
Exercise capacity and hemodynamic values	↑ Δ maximal workload	↓ Δ systolic 24-hr ABP	-0.38	0.003
		↓ Δ systolic day ABP	-0.34	0.006
		↓ Δ systolic night ABP	-0.39	0.002
		↓ Δ diastolic OBP	-0.30	0.016
		↓ Δ 24-hr HR	-0.27	0.034
	↑ Δ maximal lactate	↓ Δ systolic 24-hr ABP	-0.34	0.009
		↓ Δ systolic day ABP	-0.30	0.019
		↓ Δ systolic night ABP	-0.38	0.008
		↓ Δ diastolic OBP	-0.30	0.022
		↓ Δ 100-W diastolic BP	-0.30	0.022
	↑ Δ VO_2 peak	↓ Δ systolic 24-hr ABP	-0.30	0.026
		↓ Δ diastolic 24-hr ABP	-0.29	0.037
		↓ Δ systolic day ABP	-0.27	0.048
		↓ Δ diastolic day ABP	-0.28	0.047
		↓ Δ systolic night ABP	-0.38	0.008
		↓ Δ systolic OBP	-0.35	0.010
	↑ Δ PWC	↓ Δ systolic 24-hr ABP	-0.33	0.008
		↓ Δ systolic day ABP	-0.33	0.009
		↓ Δ diastolic OBP	-0.34	0.006
		↓ Δ 100-W diastolic BP	-0.34	0.006
		↓ Δ 24-hr HR	-0.30	0.019
Hemodynamic values	24-hr sABPM baseline	24-hr sABPM change	-0.29	0.042
	24-hr dABPM baseline	24-hr dABPM change	-0.28	0.047

Table 13 Correlation results. Symbols and Abbreviations are as follows: ↓=decrease; ↑=increase; Δ=change, ABP=ambulatory blood pressure; OBP=office blood pressure; BP=blood pressure; BMI=body mass index; HR=heart rate; HbA1c= glycated hemoglobin; LDL=low density lipoprotein HDL=high density lipoprotein; VO_2=oxygen uptake; PWC=physical work capacity.

In addition, significant inverse associations were found between the 24-hr baseline systolic ABPM and the systolic ABPM change (p=0.042) (Figure 28), and the 24-hr baseline diastolic ABPM and the diastolic ABPM change (p=0.047) (Figure 29).

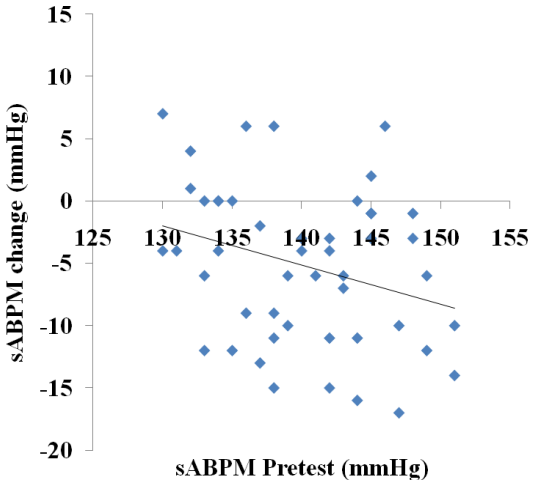

Figure 28 Correlation between sABPM baseline and sABPM change (p = 0.042)

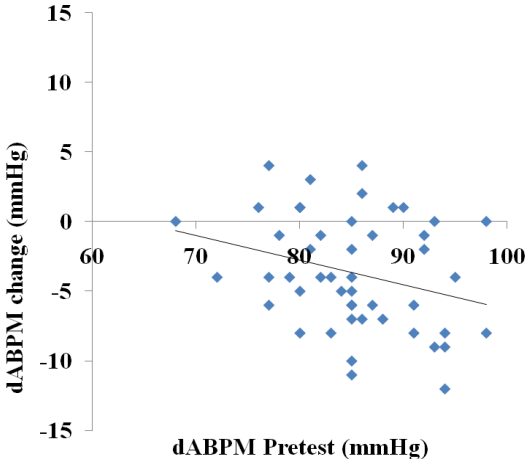

Figure 29 Correlation between dABPM baseline and dABPM change (p = 0.047)

2.4. DISCUSSION

The central objective of this study was to determine the chronic effect of aerobic training, resistance training and the combination of both on ambulatory blood pressure in persons with hypertension grade I. Alongside this examination of the influence of these training forms on other hemodynamic values, anthropometric characteristics, blood parameters and exercise capacity, were carried out as an attempt to determine if the blood pressure reduction results were from the influence of physical activity itself, from its effects on other cardiovascular risk factors or from the combined effects of all of these.

2.4.1. Anthropometric characteristics:

The baseline values of BMI and waist circumference, practical assessment methods to calculate abdominal fat distribution, demonstrated that the subjects were overweight with a BMI of 28.4 ± 3.65 kg/m^2 and waist circumference of 101.15 ± 9.7 cm (WHO, 1995).

Despite the fact that in the present study weight loss was not a program goal and subjects were repeatedly instructed to keep dietary, drinking and smoking habits as constant as possible, the results show a statistic significant BMI and waist circumference reduction of -0.65 ± 0.7 kg/m^2 and -1.47 ± 2.8 cm respectively following the aerobic exercise intervention. These outcomes demonstrate the role that an aerobic exercise program plays in the maintenance or increase of lean body mass (Murphy et al., 2007).

The lack of weight, BMI and waist circumference reduction following 12-wk resistance training must be carefully interpreted because an increase of lean body mass could mask the reduction of the percent body fat (Pollock et al., 2000; Bjarnason-Wehrens et al., 2004; Williams et al., 2007), which is known to be particularly important in terms of cardiovascular risk (Murphy et al., 2007). In addition, an increase in muscle mass contributes to the increases in basal metabolic rate (Bjarnason-Wehrens et al., 2004;

Williams et al., 2007), so resistance training can also induce a percent body fat diminution (Trombetta et al., 2003).

Significant detected changes in anthropometric variables following the combined training could be influenced not only by the greater volume of training but also by its aerobic and resistance training components. For example, comparing the change after the isolated aerobic or resistance training to the combined exercise training, it was found that participants of the combined group had a greater waist circumference reduction firstly because the aerobic component is considered a significant calorie burner and secondly because the resistance training assists the body in expending calories via an increase in lean body mass and basal metabolism (Pollock et al., 2000; William et al., 2007). In addition, the smaller BMI reduction achieved following the combined training compared to the isolated aerobic intervention could explain that the lean body mass gained by the resistance training component compensates the lost body fat.

Although the BMI and waist circumference reductions following the three interventions are modest, loss of adipose tissue is important to consider as numerous studies demonstrate that obesity-related hypertension seems to be associated with elevations in muscle sympathetic nerve activity, abnormalities in arterial baroreflex function, activation of the renin-angiotensin-aldosteron system and elevations in circulating concentrations of insulin and leptin (Grassi et al. 2000; Fisher and Fadel, 2010); while body fat reduction following caloric restriction or in combination with caloric expenditure has been associated with reductions in muscle sympathetic nerve activity (Trombetta et al., 2003), and therefore blood pressure.

In conclusion, both training forms assist with the loss of adipose tissue (Williams et al., 2007) and supports the general view that physical activity is important not only in the management of hypertension but also for the prevention of cardiovascular diseases (He et al., 2000; Rashid et al., 2003).

2.4.2. Hemodynamic values:

As mentioned in the introduction, the hypothesis of this study stated that the experimental groups that include aerobic exercise intervention would have a greater reduction on the blood pressure compared to groups and conditions that did not perform this type of exercise because the influence of aerobic training in reducing office blood pressure in hypertensive subjects had been well documented and accepted (Pescatello et al., 2004).

However, the results from different studies show different magnitudes of change after the aerobic intervention. For example, while we found a significant systolic 24-hr ambulatory blood pressure reduction of -3.3 mmHg, Westhoff et al. (2007), using a similar aerobic treatment protocol, achieved a decrease of -8.5 mmHg testing persons older than 60 years with isolated systolic hypertension, a condition associated with ageing and arterial stiffness that was only present in 16% of our participants. In addition, Westhoff et al. (2007) attributed the systolic ambulatory blood pressure reduction to an improvement of the endothelial function. Moreover, Cornelissen et al. (2009) did not detect a significant change of the ABP after 10-wk of aerobic training performed at 33% and 66% intensity, both intensities lower that those used in our study and which are probably not sufficient stimulus to trigger a significant blood pressure reduction.

In addition, comparing our systolic 24-hr ABPM results after the aerobic training with the findings from two meta-analyses, which used the ABPM as measurement technique, it was found that our results differ with the no significant decrease of only -1.7 mmHg detected by Kelley (1996); but seems consistent with the significant day-time ABPM reduction of -3.3 mmHg reported by Cornelissen and Fagard (2005a).

Although the aerobic training did not produce any statistical significance in other blood pressure measurements, they tended however to be significant and their reductions were greater than those reported as significant by meta-analytic studies. For example, whereas the present study´s diastolic 24-hr ABPM was reduced -3.1 mmHg, Kelley (1996) reported a significant decrease of -2.6 mmHg; while our systolic and diastolic

day-time ABPM and OBP were decreased -4.5/-4.0 and -9/-6.3 mmHg, Cornelissen and Fagard (2005a) detected significant reductions of -3.3/-3.5 and -6.9/-4.9 mmHg respectively; and finally while our aerobic intervention produced a systolic exercise blood pressure decrease of -7.9 mmHg, Fagard (2005a) reported a significant reduction of -7 mmHg following endurance training. Given that meta-analysis is a quantitative approach for increasing statistical power and improving estimates of effect sizes (Glass et al., 1981; Cornelissen and Fagard, 2005a), it might conclude that the blood pressure reductions of this thesis could be clearly significant if the number of participants per group would have been slightly higher.

Unlike aerobic exercise, the position statements of the professional associations have been cautious about recommending strength training as the only form of exercise for reducing hypertension (Pescatello et al., 2004; Pollock et al., 2000; Bjarnason-Wehrens et al., 2004; Predel, 2007), because only few studies had researched the influence of long term resistance training on blood pressure.

The reason of this lack of information could be that in former times, it was thought that resistance training could cause a chronic elevation of blood pressure by inducing vascular hypertrophy and increasing vascular resistance due to large cute increases in BP elicited by the exercise (Mac Dougall et al., 1985; Cornelissen and Fagard, 2005b; Cardoso et al., 2010). However our results do not support this argument. In contrast, a significant systolic BP reduction of -4.9 mmHg and a borderline non-significant diastolic ABPM decrease of -3.7 mmHg were found following the 12-wk intervention.

Comparing to other studies, our blood pressure improvement contradicts a study, which found no effect of a 16-wk strength training performed at higher intensities (70-90% 1RM) on ambulatory blood pressure (Van Hoof et al., 1996); but confirm the findings of Harris and Holly (1987), who using circuit weight training at 40% intensity achieved a 5% diastolic blood pressure reduction. In this sense, although Cornelissen and Faggard (2005b) found that the intensity of training did not significantly influence the BP-lowering effect of resistance training, it is then assumed that the blood pressure reduction could be favored with the circuit training carried out at moderate intensity (50-70% 1RM). More studies are definitively needed to clarify the underlying

mechanism responsible for training-induced reduction in systolic blood pressure and the roll of intensity and kind of resistance training.

While Kelley (1997) in his meta-analysis detected significant resting systolic and diastolic blood pressure reduction of -4.5/3.8 mmHg respectively our office systolic and diastolic blood pressure decreases of -11.6/-5.6 mmHg were not significant; nevertheless the clear tendency to improve suggests that resistance training also might have potential benefits on resting BP.

As mentioned in the hypotheses, it was expected that the combination aerobic and resistance training had a similar effect as the aerobic training on the ABPM; nevertheless, the significant -5.8 mmHg systolic blood pressure reduction produced by the combined aerobic-resistance training was greater as the -3.3 and the -4.9 mmHg decrease found following the aerobic training and the resistance training, respectively. Similarly, the variables night-time ABPM and 100-W diastolic BP were also significantly reduced after the combined exercise and their magnitudes tended to be higher than those found after the isolated aerobic or isolated resistance training intervention. Moreover the other hemodynamic variables did not reach significance statistic (24-hr dABPM, day-time sABPM, day-time dABPM, night-time dABPM, sOBP, dOBP and 100-W sBP), but had a tendency to show a greater improvement after the combined exercise intervention.

Even though several authors warn that the diastolic blood pressure recorded during bicycle exercise could be overestimated or underestimated (Franz, 2003; O'Brien et al., 2003), we have found a high significant 100-W diastolic BP reduction of -7.7 and -10.7 mmHg following the resistance training and combined training respectively. In contrast to aerobic training, the fact that during resistance training also raises the diastolic blood pressure (Williams et al., 2007) may cause physiological adaptations that benefit the diastolic response during aerobic training. Nevertheless, further research in this area should be done using more precise methods to verify the exercise diastolic blood pressure decrease and to determine the potential physiological mechanisms that might underlie this reduction.

Although Fagard (2001) in his meta-analysis did not find a relationship between exercise volume and blood pressure decrease, because the training session of the combined group lasted 1-hr and the training sessions of the AG and RG lasted only 30-min approximately, it is then thought that the greater effect could be due to exercise duration per day and not to the combination of exercise types. However, until research is conducted to clarify the doubt, it is plausibly then thought that the combined exercise group had greater improvements because this group benefited from the physiological adaptations of both training types.

As mentioned in the introduction, the pathogenesis of hypertension and its mode of progression are complex, multifactorial and incompletely understood (Fisher and Fadel, 2010). It is well known that mean arterial blood pressure is determined by cardiac output (CO) and total peripheral resistance (TPR) and reductions in arterial pressure after chronic exercise must be mediated by decreases in one or both of these variables (Pescatello et al., 2004; Mohrman and Heller, 2006). Since reductions in resting CO do not happen after chronic exercise; TPR seems to be the responsible of the BP decrease (Cornelissen and Fagard, 2005a). As derived from Poiseuille's equation, TPR is directly proportional to blood viscosity and length of the vessel, but inversely proportional to the fourth power of the radios (Pescatello et al., 2004; Mohrman and Heller, 2006). Thus even small changes in the vessel diameter have a very large influence on vascular resistance.

The chronic TPR reduction after chronic exercise could be mediated by different physiological adaptations. For example, there is current evidence indicating that an abnormality in the autonomic modulation of blood pressure homeostasis, i.e. a sympathetic activation coupled with a parasympathetic inhibition plays a pathogenic role causing and sustaining the essential hypertension state (Grassi et al., 2010). Although the causes of this so called 'neurogenic hypothesis of hyperthension' remain unclear, sympathetic overactivity is the genesis of major complications of hypertension and is the reason that promotes vasoconstriction, produces dyslipidaemia, increases insulin resistance, impairs arterial compliance, augments coagulation, aggravates atherosclerosis, develops ventricular hypertrophy, produces diastolic dysfunction and triggers arrhythmias, tachycardia, congestive heart failure, ischaemic coronary events and sudden death (Grassi, 2009).

Even during aerobic training which increases the load on the cardiovascular system and sympathetic activity (Mohrman and Heller, 2006), many authors suggest that aerobic exercise is able to decrease chronically the sympathetic activity and increase the parasympathetic activity (Pollock et al., 2000; Williams et al., 2007; Fisher and Fadel 2010). This assumption could be supported by the results from other studies which found that aerobic training restores the baroreflex in never-treated hypertensive patients (Laterza et al., 2007), reduces the norepinephrine spillover (Brown et al., 2002; Cornelissen and Fagard, 2005a), declines the plasma rennin activity (Cornelissen and Fagard, 2005a), improves the vascular responsiveness by decrease the α-adrenergic vasoconstrictor (Cheng and Chiang, 1996) which impair the endothelial function (Kingwell, 2000; Westhoff et al., 2007) and reduces the vasoconstrictor endothelin-1 levels (Maeda et al., 2001).

A common clinical measure of whole-body sympathetic and parasympathetic neural activity can be obtained by determination of heart rate (Fisher and Fadel, 2010). In this study the heart rate was measured during the electrocardiogram and by the ambulatory blood pressure monitoring. The insignificant -5 beats/min resting HR reduction measured during the electrocardiogram following exercise interventions corresponds to a decrease of -9%, similar to the significant magnitudes reported by Kelley (1996), Kelley et al. (2001) and Cornelissen and Fagard (2005a) in their meta-analyses. However, it should be noted that the values of our control group decreased 3% indicating a possible effect of an external variable during measurement takings. In contrast, the 24-hr HR measured during the ABPM showed a significant decrease of 7% and 5% following the aerobic and combined intervention, whereas the values of the resistance and control group remained unchanged.

The lack of a change in resting and ambulatory HR following resistance training could support the hypothesis that the significant systolic blood pressure decrease is in this case not influenced by neural control. In the same way, Van Hoof et al. (1996) did not detect any change in sympathetic tone, assessed by heart rate variability in response to resistance training and also Cononie et al. (1991) found no change in plasma levels of angiotensin I and II, epinephrine and norepinephrine following the 12-wk intervention.

Possible physiological explanations could be that resistance training changes the muscular composition, increases diameter of already existing arteries and veins, promotes angiogenesis (i.e., new vessel growth) (Mc Guigan et al., 2001; Pescatello et al., 2004) and/or improves the vasodilatory capacity (Collier et al., 2008). As a consequence it could reduce the peripheral resistance at rest (Kelley and Kelley, 2000) and lead to a reduction not only of the blood pressure response to a given resistance load (Mc Cartney et al., 1993), but also during a given aerobic load.

In conclusion, the significant decline in sABPM and HR during the 24-hr ABPM after aerobic and combined interventions suggest that aerobic training lowers the blood pressure via restoring neural control, whereas the significant sABPM and 100-W diastolic blood pressure decrease and the lack of a significant ambulatory HR change suggest that the resistance training reduces the blood pressure probably via remodeling the vascular structure. Nevertheless, future research should be conducted to verify these assumptions.

2.4.3. Blood Parameters

Different studies show that the aerobic and resistance trainings have a small positive effect on HDL and LDL cholesterol and a moderate positive effect on insulin response to glucose challenge, insulin sensitivity and triglycerides (Pollock et al., 2000; Williams et al., 2007), all values compatible with an overall improvement of cardiovascular risk. Nevertheless there was no finding that aerobic and combined exercise made an influence on any measured parameter.

Moreover, as happened with the aerobic and combined interventions and with the exception of the HDL-cholesterol, the blood parameters remained also unchanged following resistance training. While some authors report only a small positive effect of this kind of exercise on HDL-cholesterol (Pollock et al., 2000; Bjarnason-Wehrens et al., 2004; Cornelissen and Fagard, 2005b, Williams et al., 2007) a significant decrease of -3.8 mg/dL was detected after resistance training. Nevertheless, the posttest value remained in the normal range and did not affect the LDL/HDL ratio.

However, it is important to consider that during the current study we restricted the selection of participants to those that were sedentary, had 24-hr ABPM between 130-150/80-90 mmHg and were without antihypertensive medication. We did not consider their blood parameters as inclusion or exclusion criteria. The fact that with the exception of Total-Cholesterol, participants had normal blood values at the beginning of data collection, making difficult to produce a significant improvement through exercise.

2.4.4. Exercise capacity

The American College of Sport Medicine (ACSM) makes a difference between the quantity and quality of exercise needed to attain health-related benefits and fitness (Pollock et al., 1998). Even though our participants had hypertension grad I, the exercise intensities prescribed in this study falls within ACSM recommendation for developing and maintaining the cardiorespiratory and muscular fitness in healthy adults.

The aim of this study was to test the aerobic and/or resistance training effect on blood pressure using 50-75% intensities with hypertensive persons but under strict supervision. Using these intensities there was expected improvement in functional capacity with a greater ease of performance of everyday physical activities, better quality of life and a lower chance of developing cardiovascular diseases (Blair and Connelly, 1996).

Maximal oxygen uptake is generally considered the best indicator of cardio respiratory endurance and aerobic fitness (Murphy et al., 2007). According with Wilmore and Costill (2005), men and women, non athletes, aged between 50 and 59 years should have values between 34-41 and 24-33 ml/kg respectively. Considering that the participants of this study had a baseline below average physical fitness for men and women of their age (30.06 ± 7.1 and 20.16 ± 5.2 ml/kg respectively) clearly confirmed that they were sedentary at the beginning of training.

The lack of significant interactions in the following variables maximal heart rate, maximal lactate and rating of perceive exertion verifies that all the participants realized the posttest with a similar effort, as with the effort achieved during the pretest.

As expected, improvements found in maximal workload, workload at 2 and 4 mM/L lactate, peak oxygen consumption and physical work capacity variables indicates that in contrast to the control group, the aerobic and combined exercise programs produced physiological adaptations and enhanced the cardiopulmonary capacity of the participants. Moreover, according to Pollock et al. (1998), the increase in VO_2max ranges from 10% to 30%, depends upon the quantity and quality of training and is

directly related to frequency, intensity and duration of exercise. In case of the aerobic and combined exercises, the participants achieved a maximal oxygen uptake enhancement of 11% and 16% respectively, demonstrating that our 12-wk exercise programs produced the estimated improvement.

Both increases are compatible with the findings of Cornelissen and Fagard (2005a) who in their meta-analysis reported a peak oxygen consumption increase of 13% following dynamic endurance exercises performed for an average of 40 minutes per session, 3 times per week, at an intensity of 65% and lasting 16-wk.

Regarding to resistance group, significant improvements observed in the variables maximal workload, workload at 2mM/L lactate, peak oxygen consumption and physical work capacity indicate that despite that this group did not perform aerobic exercise the resistance training produced physiological adaptations that improved also the cardiopulmonary capacity. It other words, the dynamic resistance training can contain an aerobic component to some extent (Cornelissen and Fagard, 2005b), but with some differences with respect to aerobic exercise. For example, during resistance training, as a result of muscle contraction, the intramuscular pressure exceeds the intravascular pressure reducing the blood flow and this pressure load increases the perfusion to the contracting skeletal muscle (Williams et al., 2007). In addition, a lower blood pressure-heart rate product (systolic BP x HR) and higher diastolic blood pressures probably lead to better oxygenation myocardium when compared to endurance training (Bjarnason-Wehrens et al., 2004) and it could increase the aerobic exercise tolerance.

Although several authors reported only a small effect of resistance training on VO_2max (Pollock et al., 2000; Bjarnason-Wehrens et al., 2004; Williams et al., 2007), in this study the significant VO_2peak improvement of 4.2 mL/min/kg corresponding to a 16% augment following the resistance training is greater as the VO_2max increase of 11% reported by Cornelissen and Fagard (2005b) in their meta-analysis. However, it is important to mention that about half of the studies computed in the meta-analysis used the circuit training while the other half used the conventional method. The shorter rest periods of the circuit program could have a greater aerobic component as the conventional training promoting a strongly improvement of the maximal oxygen consumption. In addition, a possible muscle volume increase achieved during the 12-wk

resistance training intervention could improve the oxidative capacity of the participants (Conley et al., 2000).

Finally, maximal biceps and quadriceps muscle torque did not show any significant change following the 12-wk aerobic intervention; nevertheless, this result was predictable since the exercise performed on the cycle ergometer was not designed to elicit a significant improvement on these variables.

In contrast, it was expected that the resistance and combined training improved strength measured by the maximal biceps and quadriceps muscle torque test. Yet, there was only a trend towards improvement, particularly in the arm extremities. The lack of a positive effect in strength could be partially explained by the fact that early strength gains following training reflect neural and muscular adaptations (Sale, 1988). The strength gain resulting from neural adaptations (e.g., improvement in motor unit recruitment) could be specific to the type of resistance training performed. Since we measured strength gain by an isometric test this could have hidden a possible strength improvement produced by the dynamic training. Moreover, strength gains resulting from muscular adaptations occur through muscle hypertrophy, which is achieved when training is performed at high intensities; therefore, we believe that since our participants trained only 2-wk at 75% the stimuli reached was not high enough to demonstrate a strength gain during the isometric test.

In conclusion, although there was only a trend towards strength gain, the significant improvement of the cardiopulmonary exercise test indicates that all exercise programs produced a positive effect on physical and functional performance capacity which leads to significant improvement in quality of life.

2.4.5. Correlations between variables:

A secondary objective of this study was to detect the relationship between changes of different variables, as an attempt to explain whether the blood pressure variations is due to the influence of physical activity itself, to its effects on other cardiovascular risk factors or to the combined effects of all of these.

In general, we found three groups of correlations: (1) positive correlations between anthropometric characteristics (weight and BMI) and blood pressure; (2) positive correlations between blood parameters (HbA1c, LDL/HDL ratio and triglycerides), blood pressure and (3) negative correlations between exercise capacity (maximal workload, lactate, VO_2peak and physical work capacity) and blood pressure.

The positive correlations between anthropometric characteristics (weight and BMI) and blood pressure could be explained by previous studies which show obesity and hypertension trigger a sympathetic activation and an impairment in baroreflex cardiovascular control (Grassi et al., 2000) while weight loss improves the neurovascular and muscle metaboreflex control (Trombetta et al., 2003). Although a metanalysis did not find association between weight loss and blood pressure reduction (Whelton et al., 2002), it is important to mention that the overage overall intervention-related weight change was only -0.42 kg, whereas in our study was -1.17 kg.

Despite the normal blood values of the subjects, there was a positive relationship between blood pressure and blood parameters identified (HbA1c, LDL/HDL ratio and triglycerides). These results have an association with the evidence which shows that insulin resistance and hyperinsulinemia may contribute to the pathogenesis of hypertension (He et al., 1999) and that blood pressure not only at rest (Whelton et al., 2002b) but also during exercise is strongly associated with reduction in serum concentration of total cholesterol and insulin resistance (Brett et al., 2000).

The negative correlations between exercise capacity (maximal workload, lactate, VO_2peak and physical work capacity) and blood pressure were compatible with the results from Fagard (2005b) and Cornelissen and Fagard (2005a), who found that the

blood pressure decrease was more pronounced with greater increases in VO_2max. These finding are compatible with evidence from epidemiological prospective follow-up studies which verified that physical activity and fitness are inversely related to the incidence of cardiovascular disease and mortality risk (Blair et al., 1995; Cornelissen and Fagard, 2005a).

Finally, we found an inverse correlation between baseline blood pressure and its change following the 12-wk intervention program. This finding is in agreement with previous meta-analysis where blood pressure reductions following training have been shown to be more dramatic when participant's initial blood pressure is higher (Kelley, 1996; Kelley, 1997; Kelley and Kelley, 2000; Whelton et al., 2002b; Fagard, 2001; Cornelissen and Fagard, 2005b; Murphy et al., 2007).

2.5. CONCLUSIONS

2.5.1. Main findings

The main findings of this randomized controlled trial are:

(1) The aerobic training, the resistance training and the combination of both trainings lower significantly the systolic ABPM -3.3, -4.9 and -5.8 mmHg respectively.

(2) The diastolic 100-W BP presented a significant reduction following the resistance and combined interventions possibly because the diastolic blood pressure increase during the resistance training promotes physiological adaptations that benefit the diastolic response during the cardiopulmonary exercise test.

(3) Parallel to the positive effect of the aerobic interventions on the ABPM, these trainings produced a significant decrease of the HF during the ABPM indicating a possible effect of the aerobic training reducing sympathetic overactivity.

(4) Although there was no statistical significance in the other blood pressure measurements found (e.g. diastolic ABPM, systolic and diastolic OBP and systolic 100-W BP), they tended strongly to decrease following exercise interventions.

(5) Both types of trainings, either isolated or combined, were associated with favorable effects not only on other cardiovascular risk factors such as BMI and waist circumference reductions but also in the exercise capacity.

(6) The greater effect of the combined training on the different variables could be due to the exercise duration per day because the training session of this group lasted 1-hr, while the training sessions of the AG and RG groups lasted only 30-min. Nevertheless this hypothesis must be researched.

(7) The blood pressure reduction correlates positively with weight, BMI, HbA1c, LDL/HDL ratio and triglycerides and negatively with the exercise capacity.

2.5.2. Implications

Although the reductions in sABPM achieved by exercise interventions were small – from 3.3 to 5.8 mmHg- compared to those achieved by drugs, which result in average reductions of about 9 mmHg (Law et al., 2003), it has been estimated that a 2 mmHg reduction of systolic BP results in a 6% reduction in stroke mortality and a 4% reduction in mortality attributable to coronary heart disease; the percentage reduction amount to 14% and 9% respectively for a 5-mmHg decrease of BP (Chobanien et al., 2003).

These results give valuable information for the medical field because despite the likelihood of achieving only a small reduction in blood pressure product of exercise, some patients with mild hypertension or prehypertension may wish to do sport in an effort to delay or prevent starting antihypertensive drug therapy. In people with more severe hypertension, exercise and other lifestyle changes may complement the blood pressure lowering effects of drugs and thereby reduce the number of medications needed to control blood pressure (Dickinson et al., 2006).

For trainers who work prescribing exercise to this population and whose objective is to reduce the incidence of this chronic disease, these findings supply them very useful information. For example, although current reviews and professional association position statements recommend the resistance training prescription at moderate intensity and preferably in combination with aerobic endurance training (Pollock et al. 2000; Pescatello et al., 2004; Bjarnason-Wehrens et al., 2004; Cornelissen and Fagard, 2005b; Williams et al., 2007; Cardoso, 2010), this study clearly presents evidence that the isolated resistance training, executed 3 times/wk with intensities between 50-75% 1RM, is also able to reduce the systolic blood pressure chronically. However, since the best results were obtained from the combined exercise intervention and considering that both types of trainings are important components of a well-rounded exercise program, our recommendation to them is to prescribe both types of exercise at least three times/wk.

Finally, resistance training not only in combination with aerobic training but also performed in isolation is not contraindicated and can be also suggested as a nonpharmacological therapy to prevent and combat high blood pressure in adults.

2.5.3. Future research

Some research directions that emerge from this study and could be investigated using new randomized controlled trials as an attempt to understand the chronic hypotensive effect of aerobic and resistance exercise trainings and the physiological mechanisms involved are:

1. To compare the same three interventions of this study but using the same training time per session with the aim to clear whether the greater reduction in BP after ART was due the positive addition effect of both types of exercise or because this group trained more time per day.
2. To compare the effect of circuit program versus conventional program of isolated exercises because the BP reduction found by us after resistance training may be due to the aerobic component of the circuit training for the reason that it has shorter rest periods between exercises.
3. To compare the use of different trainings intensities, for example low intensity (40%) versus moderated intensity (60%) versus vigorous intensity (80%), because a particular intensity could have a greater effect on BP than other.
4. To verify the effect of resistance training on the exercise diastolic blood pressure using other methods of measurement.
5. To correlate BP with other variables in order to determine the potential physiological mechanisms that might underlie the reduction after the different exercise programs.

2.6. SUMMARY

OBJECTIVE: The aim of the study was to determine the chronic effect of aerobic training, resistance training and their combination on blood pressure, anthropometric characteristic, blood parameters and exercise capacity. **METHODS:** sixty two sedentary patients (17 women and 45 men, aged 54.4 ± 11.4 years), with grad I hypertension but without antihypertensive medication were randomly-allocated to 4 groups: (1) AG: aerobic training group; (2) RG: resistance training group; (3) ARG: aerobic-resistance training group; (4) CG: control group. Excluding the CG, the other subjects trained 12-wk, 3 days/week at 50-75% intensity. Before and after the experimental phase were measured the following variables: 24-hr systolic and diastolic ambulatory blood pressure (sABPM and dABPM), weight, body mass index (BMI), waist circumference, lipid profile, glucose, creatinin, HbA1c, uric acid, high sensitivity C-reactive protein, resting heart rate, maximal heart rate, peak oxygen uptake (VO_2peak), systolic and diastolic blood pressure at 100-W, lactate threshold (LT), maximal workload and physical work capacity (PWC). **RESULTS:** Two-way ANOVA (2 measurements x 4 treatments) detected: a significant sABP reduction after AG, RG and ARG (-3.29, -4.93 and -5.82 mmHg respectively, p=0.03) whereas the BP of the control group remained unchanged and a significant diastolic exercise blood pressure at 100-W decrease following RG and ARG intervention (-7.7 and -10.7 mmHg respectively, p=0.01). While blood parameters remained inalterable, a significant BMI and waist circumference decrease was found (p=0.015 and p=0.02 respectively); as well as a significant maximal workload, LT, VO_{2peak} and PWC increase ($p \leq 0.001$, $p \leq 0.001$, p=0.004 and $p \leq 0.000$ respectively) after exercise interventions. In addition, there was a significant positive relationship between the change in 24-hr sABP and the change in weight and BMI (p=0.004 and p=0.003 respectively) and a significant negative relationship between sABPM and increase of maximal workload and PWC (p=0.004 and p=0.009 respectively). **CONCLUSIONS**: resistance training alone or in combination with aerobic training is able to reduce the sABPM and to increase the exercise capacity after a 12-wk exercise intervention in patients with grad I hypertension. Furthermore, from observation there was no adverse reactions or complications associated with the exercise; becoming resistance training in another non pharmacological available option to reduce this chronic disease.

3. “ACUTE EFFECT OF AEROBIC AND RESISTANCE EXERCISE ON AMBULATORY BLOOD PRESSURE”

3.1. INTRODUCTION CONCERNING ACUTE EFFECT OF EXERCISE ON BLOOD PRESSURE

3.1.1. Background

As mentioned in the previous chapter, regular physical exercise has been recommended for the prevention and treatment of hypertension (Chobanian et al., 2003). Furthermore, some researchers have shown that a single bout of exercise is able to reduce blood pressure during the recovery period (Pescatello et al., 2004). However, more is known about the chronic or long term than the acute or short-term of exercise on blood pressure (Guidry et al., 2006).

The immediate decrease in BP after a single episode of exercise has been called post-exercise hypotension (PEH) and in order to be relevant, it must have a significant magnitude and be sustained for a long period of time under ambulatory conditions (Cardoso et al., 2010).

Methodologically PEH becomes established comparing the blood pressure levels after exercise to: (1) the blood pressure levels on a control day when no exercise is performed or (2) the blood pressure measured before exercise.

Nevertheless, while some researchers have confirmed the cited well-know benefits, others did not find a positive effect of exercise on BP (Cardoso et al., 2010). Once again, the controversial results could be due to the type of population, type of blood pressure measurement, time of day which the exercise is performed and characteristics of the exercise used such as intensity, duration and type of training.

Regarding the type of population, evidence suggests that the PEH effect depends on the hypertensive level of the subjects. For example, Pescatello et al. (1999) compared

hypertensive versus normotensive premenopausal women and found that 30-min of cycle exercise performed at 60% of VO_2max produced a systolic and diastolic resting BP reductions of -9.5/-6.7 mmHg for up to 7 hours after exercise only by the hypertensive women, whereas resting blood pressure of the normotensive remain unchanged. Equally Wallace et al. (1999) and Quinn (2000) detected that the hypertensive group had a significant systolic and diastolic ABP reductions, while no BP difference were found for the normotensive group.

Another important aspect to consider is the procedure of measurement, because Pescatello and Kulikowich (2001) suggest that despite the well-know belief that the exercise lowers blood pressure, studies of the lack of the PEH effect are common when they using the ambulatory blood pressure monitoring.

Although the intensity of training is one of the variables mostly studied, the effect of it remains controversial. For example, Eicher et al. (2010) found over 9-hr a greater systolic and ambulatory blood pressure decrease following vigorous intensity (100% VO_2peak) versus the non-exercise control intervention, while Pescatello et al. (2004) detected for 9-hr after low and moderate intensity (40% and 60% VO_2max respectively) an average awake systolic ABP increase of 6.9 mmHg less and diastolic ABP decrease of 2.6 mmHg after exercise compared with the control condition.

Regarding the duration of exercise, Guidry et al. (2006) compared the effects of short (15-min), long duration (30-min) and control condition on the BP response to a session of aerobic exercise and found that the immediate BP-lowering effects of short-duration at low intensity (40%) are comparable to those of long-duration and higher intensity.

Jones et al. (2008) recently investigated the roll of circadian rhythm on the PEH and found that the blood pressure reduction following 30-min of cycling at 70% is less marked if the exercise is performed in the morning that if done in the afternoon.

Regarding the type of exercise, while many studies have used the aerobic exercise as a treatment, few have investigated whether resistance exercise is capable of triggering PEH (Cardoso et al., 2010), possibly due to unfounded fears that resistance training could increase the blood pressure following the bout of exercise.

In fact, only four randomized controlled trials were found which used hypertensive people as participants and their findings showed controversial results. For example, Hardy and Tucker (1998) examined 24 mildly hypertensive sedentary men who performed 7 exercises, 3 sets, 8-12 repetitions until fatigue and found that after resistance training the daytime systolic and diastolic blood pressure was significantly reduced -12/-7 mmHg respectively. Nevertheless, the hypotensive effect persisted only for 1-hr.

Moreover, Melo et al. (2008) measured 12 hypertensive women, who were receiving captopril and discovered that 20 repetitions of 6 exercises performed 3 sets at 40% 1RM reduced the systolic and diastolic ABP -7/-5 mmHg for 10-hr during the awake period.

Queiroz et al. (2009) had the participation of 15 normotensive men who performed 3 sets of 6 exercises executed until fatigue at 50%1RM and detected no change in the systolic and diastolic ABPM between post-exercises and control values, only in the office blood pressure.

Finally, a recent study evaluated the effect of different training volumes (one lap -20-min- versus two laps -40-min in a circuit training) of a low-intensity resistance exercises over the magnitude and extent of BP changes in treated hypertensive elderly individuals and concluded that resistive exercise sessions in a circuit with different volumes reduced BP during the first 60-min after exercise, but only the highest volume (40-min) promoted mean 24-hour and awake systolic BP reductions (Scher et al., 2010).

Given that (1) there was no found randomized controlled trial examining the acute effect of aerobic training, resistance training and the combination of both on ambulatory blood pressure, (2) the discrepant findings about the antihypertensive effect of resistance training, (3) the multiple benefits of strength training on health and functionality and (4) the popularity of combining both trainings in the fitness studios; it is decisive to develop a quantitative approach to compare the acute effect of aerobic training, resistance training and their combination on systolic and diastolic ambulatory blood pressure in trained adults with pre-hypertension and hypertension grade I.

3.1.2. Research questions

a) Which mode of exercise, aerobic training, resistance training or the combination of both, has the strongest PEH effect?
b) How many hours lasts the PEH effect after each treatment?

3.1.3. Hypothesis

Experimental conditions that include aerobic exercise intervention will have a greater and more lasting reduction on the blood pressure compared to conditions that do not perform this type of exercise.

3.2. METHODS

3.2.1. Participants

This second randomized control trial was also approved by the Ethic Committee of the German Sports University (DSHS).

Participants were recruited after their participations in the first Stage (chronic effect) of this Thesis. They received information about the aims, procedures and risks of the study and then written the informed consent.

3.2.1.1. Inclusion criteria:

Volunteers were considered for this study if they have performed immediately before at least a 12-wk aerobic and resistance training program and if they had a current 24-hr ambulatory blood pressure between 125/80 and 140/90 mmHg.

3.2.1.2. Exclusion criteria:

The exclusion criteria were the same as described in the first stage (chronic effect).

According to these criteria, 17 nonsmoker patients (15 Male, 2 Female) were enrolled and finished the experimental treatments: 15 persons came from the Aerobic Resistance Group (ARG) and 2 persons from the Resistance Group (RG). So, to describe the group of participants, the variables weight, height, body mass index, waist circumference, office BP, VO_2peak, cholesterol HDL, triglycerides and glucose were obtained from the posttest of the chronic stage.

The subjects from the ARG participated directly after finishing the 12-wk chronic training, whereas the volunteers of the RG trained 3 months both exercise types, to be on equal footing with the subjects from the ARG before participating in the acute stage.

3.2.2. Study design

With the objective to determine the acute effect of each type of exercise on the ambulatory blood pressure, after completing and signing the written informed consent and confirming inclusion and exclusion criteria, participants visited the Fitness Center "Trainingsinstitut Prof. Dr. Baum" between 12:00 and 16:00 hours and participated in four sessions more, in random order on four separate days: Aerobic Training Condition (AC), Resistance Training Condition (RC), Aerobic-Resistance Training Condition (ARC) and Control Condition (CC).

In addition, subjects were asked to refrain from formal exercise for a minimum of 24-hr before any testing.

3.2.3. Protocol

During the experimental sessions, subjects were repeatedly instructed to keep dietary and drinking habits as constant as possible. To avoid diurnal variation, all experimental conditions were taken the same day of the week at the same time of day during 4-wk.

Every day arrived the participants and after a minimum of 5-min of seated rest, the office blood pressure and resting heart rate were determined three times (5-1, Omron Healthcare, Japan). The measurements were averaged and used as baseline before exercise.

After these measurements, participants warmed-up by pedaling 5-min at 40% of their $HR_{reserve}$ (Karvonen et al., 1957) prior to engaging in experimental condition (see formula below).

$$HR_{exercise} = [(HR_{max} - HR_{rest}) \times \% \text{ Intensity}] + HR_{rest}$$

$HR_{exercise}$ formula: HR_{max} indicates the maximal HR during the cardiopulmonary exercise test and HR_{rest} the lowest Heart Rate during the resting ECG.

Following 5-min warm-up at 40% intensity on a cycloergometer (Motion Cycle 400, Emotion Fitness, Hochspeyer, Germany) participants performed a specific work, according to the randomized condition of each specific day:

Aerobic Condition: it was performed at 70% intensity on a cycloergometer (Motion Cycle 400, Emotion Fitness, Hochspeyer, Germany) during 30-min and the intensity was controlled by telemetry by heart rate monitors (Polar, model FS1, Kempele, Finland). In order to individualize the exercise protocol for each person, the aerobic training intensity was calculated with the $HR_{exercise}$ formula (see formula above), using the values obtained by the cardiopulmonary exercise test of the chronic stage.

Resistance Condition: weight machines (Conex multiform, La Roque d'Anthéron, France) were used for strengthening muscle groups of the upper and lower body. In each session, the participants completed 2 circuits of 13 resistance exercises: leg extension, seated leg flexion, seated calf (heel) raise, seated erector spinal, seated abdominal, leg press, row, butterfly, lat pulldown, overheat shoulder press, seated chest press, biceps dumbbell curl and triceps dumbbell extension. Each exercise was repeated 10 times at 70% intensity. Pauses between types of exercises lasted about 30s. The training lasted about 30 min. Participants were instructed to avoid the Valsalva maneuver during the exercises. In order to individualize the exercise protocol for each person, the resistance training intensity was determined with a test of one repetition maximum of each resistance exercise.

Aerobic and Resistance Condition: participants performed in consecutive form both previously described trainings programs. About 1-hr was needed to complete a training session.

Control Condition: volunteers rested 30-min.

Immediately after exercise or resting condition was measured the office blood pressure and resting heart rate three times again (5-1, Omron Healthcare, Japan). The results were averaged and used as post exercise values. Then the participants showered and changed their clothes in approximately 15-min. Thus, twenty minutes after the exercise began the 24-hr ambulatory blood pressure and heart rate monitoring (Spacelabs, model

90217, Washington, USA). Intervals between single blood pressure measurements were programmed every 30-min. Volunteers left the Institute with instructions to keep their arm extended and still when the monitor was recording, to proceed with their typical activities, not to engage in formal exercise, to record the activities performed during the day, to stop the monitor at 8:00 a.m. and to return it the following day. The general study schedule is shown in Figure 28.

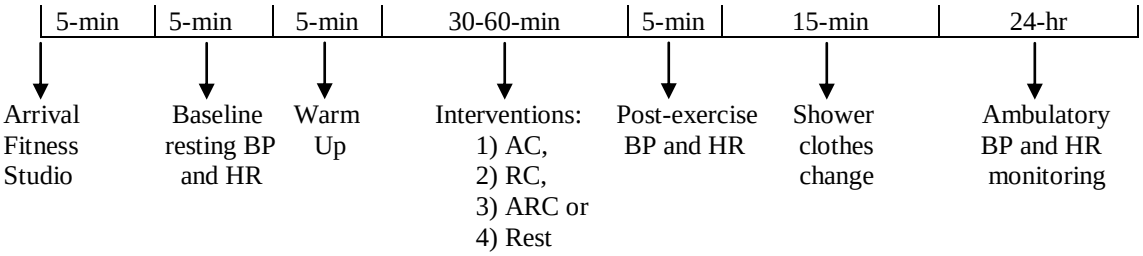

Figure 30 Acute stage schedule. Abbreviations are as follows: BP=blood pressure, HR=heart rate, AC=aerobic condition, RC=resistance condition, ARC=aerobic and resistance condition, CC=control condition.

3.2.4. Statistical Analysis

Values of $p<0.05$ were accepted as significant and analyses were performed with the Statistical Package for Social Sciences, version 15.0 (SPSS Inc., Chicago, Illinois, USA).

The data collected with the ABP device were computed in two ways for the statistical analyses:

First was processed the systolic and diastolic 24-hr ABPM. One-way ANOVAS were computed to determine possible 24-hr systolic and diastolic Ambulatory Blood Pressure differences between the 4 experimental conditions (AC, RC, ARC and CC).

Second, it was processed the change between the blood pressure prior exercise and the following 6-hr after conditions. This amount of hours was arbitrarily determined by considering, that 6 hours was the longest time period in which all participants were awake each day of measurement.

Two-ways ANOVAS with repeated measures in both factors (4 conditions x 8 time-measurements) were realized to find possible significant blood pressure change interactions. After a significant F ratio (Geisser-Greenhouse correction for the assumption of sphericity), when a significant condition-by-time interaction was seen, within-group comparisons between time points were performed using simple effects post hoc analyses.

3.3. RESULTS

As mentioned above, 17 participants (15 males, 2 females) completed the 4 conditions and were included in the final analyses. Some characteristics of the subjects are presented as mean and standard deviation in the Table 14.

Variable	Mean ± SD	Variable	Mean ± SD
Age (years)	55.0 ± 12.2	Systolic Office BP (mmHg)	139. 3 ± 12.4
Height (cm)	177.2 ± 11.1	Diastolic Office BP (mmHg)	91.6 ± 11.3
Weight (kg)	92.5 ± 19.8	Cholesterol HDL (mg/dL)	54.7 ± 11.3
Body mass index (kg/m^2)	29.15 ± 4.4	Triglycerides (mg/dL)	124.6 ± 57.6
Waist circumference (cm)	103.2 ± 13.3	Glucose (mg/dL)	96.7 ± 31.2
VO_2peak (mL/min/kg)	28.7± 7.7		

Table 14 Participants characteristics: Data are presented as means ± SD. Abbreviations are as follows: BP=blood pressure, VO2peak=highest attained oxygen consumption, HDL=high density lipoprotein.

Descriptive statistic for the 24-hr ambulatory blood pressure and heart rate monitoring are presented in Table 15. As can be seen, there was no statistically significant heart rate difference after the four conditions.

Ambulatory blood pressure and heart rate monitoring	AC	RC	ARC	CC
24-hr systolic ABPM (mmHg)	131.24 ± 5.9	130.35 ± 7.8	130.0 ± 6.7	132.0 ± 6.8
24- hr diastolic ABPM (mmHg)	78.4 ± 4.5	77.8 ± 5.8	78.1 ± 5.3	78.4 ± 5.6
Day-time systolic ABPM (mmHg)	138.1 ± 6.6	137.2 ± 8.4	137.4 ± 8.3	139.6 ± 7.4
Day-time diastolic ABPM (mmHg)	83.1 ± 5.2	83.2 ± 5.7	78.1 ± 5.3	83.9 ± 5.8
Night-time systolic ABPM (mmHg)	118.8 ± 7.2	117.2 ± 8.0	118.6 ± 7.2	118.8 ± 7.7
Night-time diastolic ABPM (mmHg)	69.4 ± 6.5	68.0 ± 6.6	68.0 ± 5.4	68.7 ± 6.7
24-hr ambulatory HR (beats/min)	71.2 ± 9.0	70.0 ± 10.8	71.2 ± 10.2	70.9 ± 12.5

Table 15 Ambulatory blood pressure values. Data are presented as means ± SD. Abbreviations are as follows: AC=aerobic condition, RC=resistance condition, ARC=aerobic and resistance condition, CC=control condition, ABPM= ambulatory blood pressure monitoring, HR=heart rate.

Figures 31 and 32 show 24-hr systolic and diastolic ambulatory blood pressure results. Although it were not found significant difference (p=0.65 and p=0.13 respectively), there is a trend towards ABPM reductions following the conditions that included resistance exercise (see Table 15).

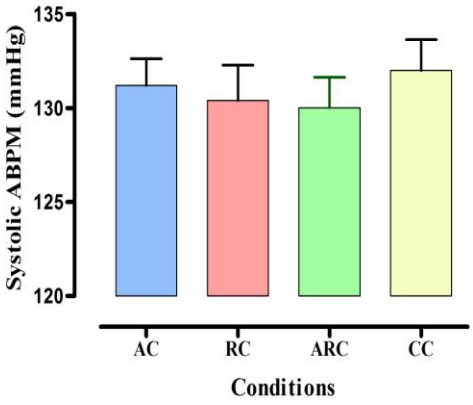

Figure 31 Comparisons of 24-hr systolic ABPM after conditions.

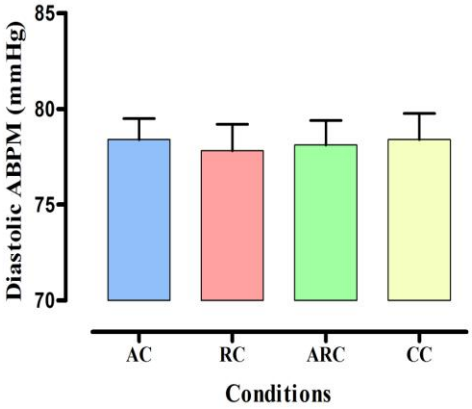

Figure 32 Comparisons of 24-hr diastolic ABPM after conditions.

Similarly day-time systolic ambulatory blood pressure also showed no significant differences (p=0.59), however the trend is to be lower after resistance exercise conditions (Figure 33 and Table 15). Moreover, day-time diastolic ambulatory blood pressure detected a high significant reduction following the combined aerobic-resistance exercise intervention comparing with the others three conditions (p<0.001) (Figure 34).

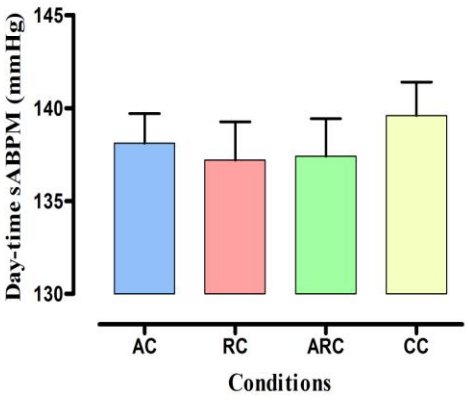

Figure 33 Comparisons of day-time systolic ABPM after conditions.

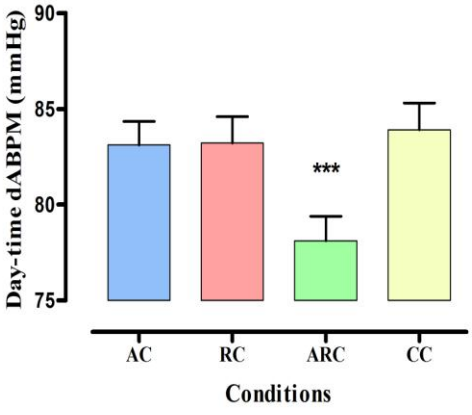

Figure 34 Comparisons of day-time diastolic ABPM after conditions. ***:p<0.001 from other.

Regarding the night-time systolic and diastolic ambulatory blood pressure, no significant difference were found following the four conditions (p=0.85 and p=0.78 respectively) however, the values tend to be lower after resistance exercise (Figures 35 and 36).

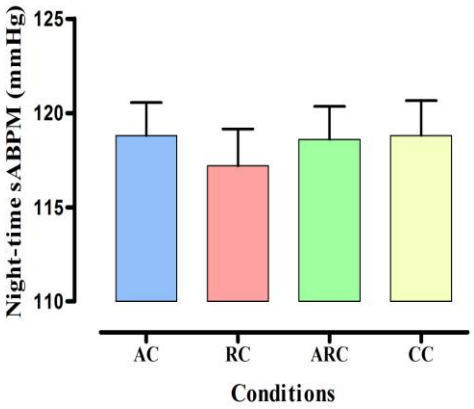

Figure 35 Comparisons of night-time systolic ABPM after conditions.

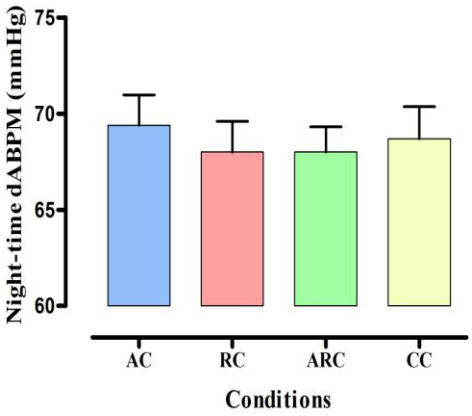

Figure 36 Comparisons of night-time diastolic ABPM after conditions.

Finally, Table 16 shows the systolic and diastolic change blood pressure in the following 6-hr after training. While there was no significant interaction between the 4 conditions and the following 6-hrs systolic ABPM (p=0.23, Figure 37), there was found a significant interaction on the diastolic 6-hr ambulatory blood pressure (p=0.02). Post-hoc analyses showed that the significant reductions lasted one hour after the resistance exercise conditions and two hours following the aerobic and the combined exercise conditions (Figure 38).

Variable	Hour	AC	RC	ARC	CC
Systolic ABP change (mmHg)	**BE**	0	0	0	0
	AE	1.12 ± 11.7	-1.41 ± 11.6	3.47 ± 15.0	4.0 ± 18.2
	1	3.6 ± 10.1	-2.6 ± 13.6	-1.2 ± 13.5	6.4 ± 18.0
	2	-4.2 ± 20.1	2.8 ± 17.7	3.0 ± 18.4	6.2 ± 18.7
	3	1.5 ± 14.8	4.5 ± 20.0	2.82 ± 21.8	0.23 ± 16.1
	4	3.2 ± 21.3	2.9 ± 20.0	4.8 ± 16.6	4.11 ± 15.2
	5	3.17 ± 17.4	6.1 ± 20.0	3.9 ± 14.4	2.6 ± 16.0
	6	4.47 ± 15.3	8.7 ± 16.7	5.0 ± 13.9	1.9 ± 15.5
Diastolic ABP change (mmHg)	**BE**	0	0	0	0
	AE	1.94 ± 8.7	1.6 ± 8.8	1.6 ± 10.6	5.4 ± 14.9
	1	**-3.5 ± 10.4***	**-6.1 ± 10.2***	**-5.4 ± 15.2***	5.9 ± 17.2
	2	**-3.2 ± 12.7***	0.2 ± 10.0	**-4.8 ± 13.9***	2.9 ± 20.0
	3	2.1 ± 11.4	3.2 ± 17.3	-0.9 ± 17.4	-2.6 ± 25.1
	4	3.0 ± 13.4	-1.5 ± 16.5	0.7 ± 14.5	-0.9 ± 16.9
	5	4.8 ± 13.3	0.00 ± 16.7	1.1 ± 15.6	0.6 ± 20.6
	6	8.1 ± 11.8	5.2 ± 15.9	2.8 ± 11.9	3.0 ± 20.8

Table 15 6-hr Ambulatory Blood Pressure Change. Data are presented as means ± SD. Abbreviations are as follows: BE= before exercise, AE=after exercise, AC=aerobic condition, RC=resistance condition, ARC=aerobic and resistance condition, CC=control condition, ABP= ambulatory blood pressure. *:p<0.05 from BE.

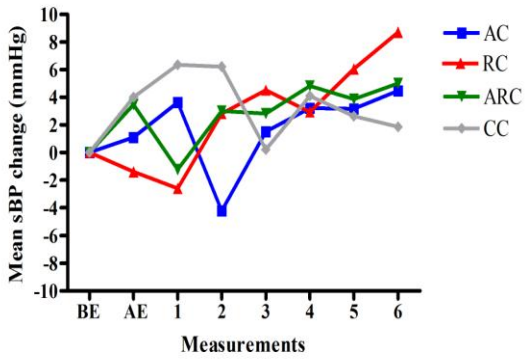

Figure 37 Comparisons of 6-hr systolic ABPM change after conditions.

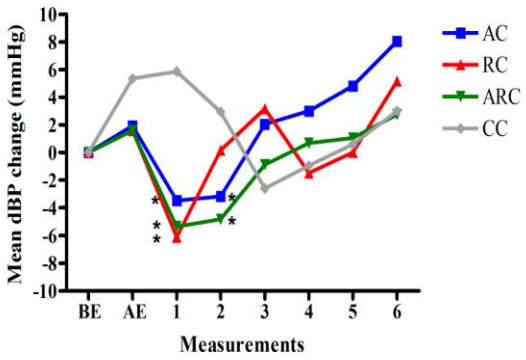

Figure 38 Comparisons of 6-hr diastolic ABPM after conditions. *:p<0.05 from BE.

3.4. DISCUSSION

The objectives of this study were: (1) to determine the acute effect of aerobic exercise, resistance training and the combination of both on ABP in trained persons with pre-hypertension and HTN grade I and (2) to measure how many hours lasts the possible PEH effect after each treatment. For this reason the discussion is presented in three sections: (1) The 24-hr ABPM after each exercise condition compared with the control condition; (2) The BP-change after each intervention compared to the blood pressure baseline; and (3) the possible underlying physiological mechanisms.

3.4.1. The 24-hr ABP response after each intervention

Considering that the BP measurements were all taken on the same day of the week, the similar heart rates averages after exercise and control conditions provide evidence indicating that the activity levels during the four days were similar.

Although several studies have detected a positive effect of aerobic exercise on the 24-hr ABPM comparing with the control condition, on the contrary this was not confirm in the present study. Considering that the intensity of the prescribed aerobic exercise, the time of day and the duration of each session was defined by us taking into account previous randomized controlled trials, which found a PEH effect (Eicher et al., 2010; Pescatello et al., 2004; Jones et al., 2008; Guidry et al., 2006), our lack of a significant BP reduction could be attributed to the following factors:

Firstly, as seen in the previous chapter and supported by several researchers (Pescatello et al., 1999; Wallace et al., 1999; Quinn, 2000; Pescatello and Kulikowich, 2001), the fact that several participants had only pre-hypertension could reduce the aerobic exercise ability to trigger a significant PEH because the magnitude of change after exercise depends of the initial BP values such that persons with the highest baseline BP experience the greatest ABP reductions.

Secondly, as shown in Table 14, many subjects tend to suffer from metabolic syndrome and according to Pescatello et al. (2008), persons with this disorder respond less

favorably to the antihypertensive effects of aerobic exercise than men without the metabolic syndrome.

Thirdly and finally, although most of the studies used sedentary people as participants, we decided to measure trained persons with the aim to detect, if the blood pressure was lower during the training days than the resting days. This methodological difference could indicate that inactive hypertensive people are more sensitive to the hypotensive effect of aerobic exercise than trained people. Nevertheless this hypothesis should be tested.

Regarding the resistance condition, our findings are similar to the results found by Queiroz et al. (2009), who after resistance exercise performed at 50% intensity until the fatigue detected did not alter the systolic and diastolic ABP; but differ from Melo et al., (2008); who found a significant systolic and diastolic blood pressure decrease following resistance training at 40%.intensity. The discrepancy with our outcomes could be attributed either the low exercise intensity, or the fact that participants in this study were receiving captopril, which could produce a possible interaction between exercise and antihypertensive drugs (Cardoso et al., 2010).

Only the combined exercise was able to produce a significant change on day-time diastolic ABP comparing with the other conditions. Once again appears the question, if the diastolic blood pressure decreased due to the combination of both exercises, or because the volume of exercise in the ARC was higher than in the others exercise conditions. However, the lack of previous data addressing this issue makes any conclusion premature.

3.4.2. The duration of BP change after each intervention

Although the PEH following aerobic exercise is well documented in hypertensive people (Cardoso et al., 2010), the duration of this phenomenon varies considerably amongst studies. While MacDonald et al. (2000) found that 10-min of cycling at 70% VO_2peak was as effective as a 30-min bout in acutely lowering BP for 60 min after exercise in young adults with borderline hypertension; others randomized controlled trials found that PEH following an aerobic exercise bout could last between 4-16 hrs

(Pescatello et al., 1999; Wallace et al., 1999; Guidry et al., 2006; Pescatello et al., 2008, Cardoso et al., 2010).

In the present study, a significant diastolic blood pressure decrease following aerobic exercise and the combined exercise was detected. However, unlike other studies, the PEH lasted only two hours. So factors such as subjects and exercise characteristics might influence the findings. For example, comparing the results of the present study with studies, which discovered longer-lasting BP reductions than us (Pescatello et al., 1991; Pescatello et al., 1999; Wallace et al., 1999; Guidry et al., 2006; Pescatello et al., 2008) it was found, that these randomized controlled trials used lower exercise intensity (between 40% and 60%) than us (70%), so these results suggest that moderate aerobic exercise intensity produces longer-lasting effects than vigorous intensity.

On the other hand, the PEH following resistance training lasted in this presents study for only one hour. In this case, this particular finding is consistent with the results from previous studies. For example, the study from Hardy and Tucker (1998), who found a systolic and diastolic ambulatory blood pressure reduction of -12/-7 respectively, which persisted only for 1-hr following resistance training by hypertensive sedentary men. In the same way Scher et al. (2010) concluded that resistive exercise sessions in a circuit with different volumes decrease ABP during the first hour after exercise. Finally, Hill et al. (1989) examined the BP response after four resistive exercises performed at 70% of 1-RM for as many repetitions as possible in six young normotensive men and found a significant decrease in diastolic BP for up to 1-hr after resistive exercise but no significant change in systolic BP from baseline.

3.4.3. Possible underlying physiological mechanisms.

Although the present thesis was not designed to examine possible mechanisms by which aerobic or resistance training acutely lower BP, it is thought that PEH is due to reductions in peripheral vascular resistance, rather than cardiac output (Guidry et al., 2006) and could be product upon interactions between many factors including: genetic predispositions, exercise characteristics and the depressor and pressor actions of BP regulatory hormones such as catecholamines, rennin and insulin (Pescatello et al., 2008).

At the genetic level, a recent study concluded that the hypertensive persons with a homozygous deletion of the angiotension converting enzyme gene (ACE DD) hat ambulatory PEH, while patients with the other polymorphic variants (ACE II/ID) did not (Blanchard et al., 2006). In addition, the same authors concluded that hypertensive subjects with three or more polymorphisms associated with the rennin-angiotensin-aldosterone system showed greater reductions in post-exercise ambulatory blood pressure.

At a systemic level, it is known that the decrease in peripheral resistance could be produced by a sympathetic inhibition and altered vascular responsiveness after exercise. Regarding to the sympathetic outflow reduction, PEH appears to be associated with a resetting of the operating point of the arterial baroreflex to a lower BP (Guidry et al., 2006, Halliwill et al., 1996); while the vascular resistance reduction could be related with decreased transduction of sympathetic out flow to vascular resistance, the release of local vasodilator substances induced by muscle contraction (e.g. prostaglandins, adenosine and ATP) and the augmented muscle blood flow, which produces a shear stress on endothelial cells and increases the nitric oxide released (Pescatello et al., 2004; Mairona, et al., 2003). These local released substances (nitric oxide, prostaglandins, adenosine and ATP) are augmented during exercise and would also facilitate peripheral vasodilatation following acute exercise. Moreover, there is evidence suggesting, that the reduced α_1-adrenergics responsiveness after exercise may be attributed to the nitric oxide production (Rao, et al. 2002).

Finally, the systolic, diastolic and heart rate elevations during resistive exercise (Williams et al., 2007) do not persist in the 24-hr after exercise, rather it was detected a significant day time diastolic ABP decrease following the combined condition. As mentioned in the previous chapters, during the aerobic exercise increases systolic blood pressure, with maintenance of diastolic blood pressure ,while during the resistance training rise the diastolic blood pressure too (Williams et al., 2007). It might be though that the rise in diastolic blood pressure during resistance exercise produces associated adaptations to the day-time diastolic ABP decrease. Nevertheless, confirmatory studies should be done.

3.5. CONCLUSION

3.5.1. Main findings

To our knowledge, this is the first study to examine 24-hr ambulatory blood pressure in trained subjects after an acute aerobic, resistive, or combined training using a repeated measure design performing an exercise protocol similar to the more typical aerobic and/or resistive training session and comparing with a non-exercise control day.

The major findings of this study discovered that trained individuals had a similar 24-hr ambulatory BP behaviors after acute aerobic, or resistance, or the combination of both exercises compared with the control condition.

Only the combined exercise was able to produce a day-time diastolic ABP lower than the other conditions. The greater effect of the combined training could be due to the exercise duration per day, because the training session of this group lasted 1-hr while the training sessions of the AG and RG groups lasted only 30-min.

Analyzing the hours immediately after and for up to 6-hr acute exercise trainings, the systolic blood pressure was unchanged while the diastolic blood pressure decreased significantly during 2-hr after conditions with aerobic exercise and 1-hr following the resistance exercise.

These findings can be only generalized to the population study: trained, middle-aged with high normal to stage 1 HTN persons.

3.5.2. Implications

Although investigators have generally given much more attention to the effects of chronic exercise on blood pressure, repeated exposure to acute bouts of exercise reducing cardiovascular reactivity may cumulatively results in cardiovascular health benefits.

Our findings suggest that aerobic and resistance training are safe for people with high normal and stage I HTN. Moreover, performing both types of training produces a diastolic hypotension during the waking period, which is very relevant as it is during the day when persons have greater stress and higher BP levels.

For this reason, the combined aerobic-resistance training, prescribed inclusive at 70% intensity, become importance as a non pharmacological approach to the prevention, treatment and control of HTN.

3.5.3. Future research

New research directions that emerge from this study and could be investigated using new randomized controlled trials as an attempt to understand the acute hypotensive effect of aerobic and resistance exercise and the physiological mechanisms involved are:

1. To clarify whether trained people are less sensitive to the PEH.
2. To compare the same three interventions of this study but using the same training time per session with the aim to clear whether the greater reduction in 24-hr ABP after ARC was due the positive addition effect of both types of exercise or because this intervention lasted more time.
3. To compare the effect of circuit program versus conventional program of isolated exercises, because the BP reduction found by us after resistance training may be due to the aerobic component of the circuit training, as it has shorter rest periods between exercises.
4. To compare the use of different aerobic and/or resistance training intensities, for example low intensity (40%) versus moderated intensity (60%) versus vigorous intensity (80%), because a particular intensity could have a greater PEH than other.

3.6. SUMMARY

OBJECTIVE: The purpose of this study was to observe the acute effect of aerobic exercise, resistance exercise and their combination on ambulatory blood pressure (ABPM). **METHODS**: 17 trained persons (15 men and 2 women, aged 55.06 ± 12.26 years, BMI 29.15 ± 4.58 and VO_2max 29.03 ± 7.31 mL/min/kg) with pre-hypertension or Grade I Hypertension but without antihypertensive medication participated in four training treatments each carried out on different days under a randomized repeated measures design: (1) AC: aerobic training condition (30-min cycling at 70% $HR_{reserve}$); (2) RC: Resistance training condition (2 circuits of 13 exercises performed at 70% 1RM) (3) ARC: aerobic and resistance training condition and (4) CC: control condition (30-min resting). Resting BP was measured 5-min before and 5-min following each condition, followed by a 24-hr ambulatory blood pressure monitoring. **RESULTS**: One-way analysis of variance detected only a significant decrease of the diastolic day-time ABPM following the ARC compared with the other three interventions ($p<0.001$), while systolic and diastolic 24-hr ABPM, systolic day-time ABPM and systolic and diastolic night-time ABPM showed no significant differences. In addition, two ways analysis of variance detected no significant interaction between the 4 conditions and the following 6-hrs systolic ABPM ($p=0.23$), but a significant interaction on the diastolic 6-hr ABPM ($p=0.02$). Post-hoc analyses showed that the diastolic significant reduction lasted one hour after the RC and two hours following the AC and ARC. **CONCLUSIONS** resistance exercise alone or in combination with aerobic exercise does not increase 24-hr ABPM, rather produces a decrease in the day time diastolic pressure post-exercise.

REFERENCES

Al-Safi, S.A. (2005). Does smoking affect blood pressure and heart rate? *Eur. J. Cardiovasc. Nurs.*, 4, 286-289.

Appel, L.J., Stason, W.B. (1993). Ambulatory blood pressure monitoring and blood pressure self-measurement in the diagnosis and management of hypertension. *Ann. Intern. Med.*, 118, 867-882.

Baum, K., Rüther, T., Essfeld, D. (2003). Reduction of blood pressure response during strength training through intermittent muscle relaxation. *Int. J. Sports. Med.*, 24, 441-445.

Bjarnason-Whrens, B., Mayer-Berger, W., Meister, E.R., Baum, K., Hambrecht, R., Gielen, S. (2004). Recommendations for resistance exercise in cardiac rehabilitation. Recommendations of the German Federation for Cardiovascular Prevention and Rehabilitation. *Eur. J. Cardiovasc. Prev. and Rehabil.*, 11, 352-361.

Blair, S.N., Kohl, H.W., Barlow, C.E., Paffenbarger, R.S., Gibbons, L.W., Maera, C.A. (1995). Changes in physical-fitness and all-cause mortality: A prospective-study of healthy and unhealthy men. *JAMA*, 273, 1093-1098.

Blair, S.N., Connelly, J.C. (1996). How much physical activity should we do? The case of moderate amounts and intensities of physical activity. *Res. Q. Exerc. Sport*, 67, 193-205.

Blanchard, B., Tsongalis, G.J., Guidry, M.A., LaBelle, L.A., Poulin, M., Taylor, A.L., et al. (2006). RAAS polymorphisms alter the acute blood pressure response to aerobic exercise among men with hypertension. *Eur. J. Appl. Physiol*., 97, 26-33.

Brett, S.E., Ritter, J.M., Chowienczyk, P.J. (2000). Diastolic blood pressure changes during exercise positively correlate with serum cholesterol and insulin resistance. *Circulation*, 101, 611-615.

Brown, M.D., Dengel, D.R., Hogikyan, R.V., Supiano, M.A. (2002). Sympathetic activity and the heterogeneous blood pressure response to exercise training in hypertensive. *J. Appl. Physiol*., 92, 1434-1442.

Cardoso, Jr. C.G., Gomides, R.S., Queiroz, A.C.C., Pinto, L.G., Lobo, F.S., Tinucci, T., Mion, Jr. D., Forjaz, C.L.M. (2010). Acute and chronic effects of aerobic and resistance exercise on ambulatory blood pressure. *Clinics*, 65, 317-325.

Celis, H., Hond, E.E., Staessen, J.A. (2005). Self measurement of blood pressure at home in the management of hypertension. *CM and R*, 1, 19-26.

Cheng, H.I., Chiang, I.P. (1996). Chronic exercise decreases adrenergic agonist-induced vasoconstriction in spontaneously hypertensive rats. *Am. J. Physiol.*, 271, H977-H983.

Chobanian. A., Bakris G.L., Black, H.R., Cuschman, W.C., Green, L.A., Izzo, J.L., Jones, D.W., Materson, B.J., Oparil, S., Wright, J.T., Rocella, E.J. (2003). The 7th report of the Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure. (JNC VII). *JAMA, 289*, 2560-2572.

Collier, S.R., Kanaley, J.A., Carhart Jr, R., Frechette, V., Tobin, M.M., Hall, A.K. (2008). Effect of 4 weeks of aerobic or resistance exercise training on arterial stiffness, blood flow and blood pressure in pre- and stage-1 hypertension. *J. Hum. Hypertens.,* 22, 678-686.

Cononie, C.C., Graves, J.E., Pollock, M.L., Phillips, M.I., Sumners, C., Hagberg, J.M. (1991). Effect of exercise training on blood pressure in 70-to 79-yr-old men and women. *Med. Sci. Sports. Exerc*., 23: 505-511.

Conley, K.E., Esselman, P.C., Jubrias, S.A., Cress, M.E., Inglin, B., Mogadam, C., Schoene, R.B. (2000). Ageing, muscle properties and maximal O_2 uptake rate in humans. *J. Physiol*., 526 Pt 1:211-217.

Cornelissen, V.A., Fagard, R.H. (2005a). Effects of endurance training on blood pressure, blood pressure-regulating mechanisms and cardiovascular risk factors. *J. Hypertens.,* 45, 667-675.

Cornelissen, V.A., Fagard, R.H. (2005b). Effect of resistance training on resting blood pressure: A meta-analysis of randomized controlled trials. *J. Hypertens., 23*, 251-259.

Cornelissen, V.A., Arnout, J., Holvoet, P., Fagard, R.H. (2009). Influence of exercise at lower and higher intensity on blood pressure and cardiovascular risk factors at older age. *J. Hypertens.,* 27, 753-762.

Dickinson, H.O., Mason, J.M., Nicolson, D.J., Campbell, F., Beyer, F. R., Cook, J.V., Williams, B., Ford, G.A. (2006). Lifestyle interventions to reduce raised blood pressure: a systematic review of randomized controlled trials. *J. Hypertens*., 24, 215-233.

Dolan, E., Stanton, A., Thijs, L., Hinedi, K., Atkins, N., McClory, S., Den Hond, E., McCormack, P., Staessen, J.A., O'Brien, E. (2005). Superiority of ambulatory over clinic blood pressure measurement in predicting mortality: the Dublin outcome study. *Hypertension*, 46, 156-161.

Eicher, J.D., Maresh, C.M., Tsongalis, G.J., Thompson, P.D., Pescatello, L.S. (2010). The additive blood pressure lowering effects of exercise intensity on post-exercise hypotension. *Am. Heart. J.,* 160, 513-520.

Fagard, R.H. (2001) Exercise characteristics and the blood pressure response to dynamic physical training. *Med. Sci. Sports. Exerc.*, 33, S484-S492.

Fagard, R.H. (2002). Physical exercise and coronary artery disease. *Acta Cardiol., 57, 91-100.*

Fagard, R.H. (2005a). Effects of exercise, diet and their combination on blood pressure. *J. Hum. Hypertens.,* 19, S20-S24.

Fagard, R. H. (2005b). Physical activity, physical fitness and the incidence of hypertension. *J. Hypertens.*, *23*, 265-267.

Fagard, R.H., Björnstad, H.H., Børjesson, M., Carré, F., Deligiannis, A., Vanhees, L. (2005). ESC study group of sports cardiology recommendations for participation in leisure-time physical activities and competitive sports for patients with hypertension. *Eur. J. Cardiovasc. Prev. Rehabil.*, 12, 326-331.

Fagard, R. H. (2006). Exercise is good for your blood pressure: Effects of endurance training and resistance training. *Clin. Exp. Pharmacol. Physiol., 33*, 853-856.

Fagard, R.H and Cornelissen, V.A. (2007). Effect of exercise on blood pressure control in hypertensive patients. *Eur. J. Cardiovasc. Prev. Rehabil.*, 14, 12-17.

Fisher, J.P. and Fadel, P.J. (2010). Therapeutic strategies for targeting excessive central sympathetic activation in human hypertension. *Exp. Physiol.*, 95, 572-580.

Franz, I.-W. (2003). Blutdruckverhalten während Ergometrie. *Dtsch. Z. Sportmed.*, 54, 55-56.

Friedman, R.H., Kazis, L.E., Jette, A., Smith, M.B., Stollerman, J., Torgerson, J., Carey, K. (1996). A telecommunications system for monitoring and counseling patients with hypertension. Impact on medication adherence and blood pressure control. *Am. J. Hypertens.*, 9, 285-292.

Glass, G.V., McGaw, B., Smith, M.L. (1981). *Meta-analysis in social research*. Newberry Park, California: Sage Publications, Inc.

Grassi, G., Seravalle, G., Dell'Oro, R., Turri, C., Bolla, G.B., Mancia, G. (2000). Adrenergic and reflex abnormalities in obesity-related hypertension. *Hypertension*, 36, 538-542.

Grassi, G. (2009). Assessment of sympathetic cardiovascular drive in human hypertension: achievements and perspectives. *Hypertension*, 54, 690-697.

Grassi, G., Seravalle, G., Quarti-Trevano, F. (2010). The 'neuroadrenergic hypothesis' in hypertension: current evidence. *Exp. Physiol.*, 95, 581-586.

Guidry, M.A., Blanchard, B.E., Thompson, P.D., Maresh, C.M., Seip, R.L., Taylor, A.L., Pescatello, L.S. (2006). The influence of short and long duration on the blood pressure response to an acute bout of dynamic exercise. *Am. Heart. J.*, 151, 1322.e5-1322.e12.

Halliwill, J.R., Taylor, J.A., Eckberg, D.L. (1996). Impaired sympathetic vascular regulation in humans after acute dynamic exercise. *J. Physiol.*, 495, 279-288.

Hardy, D.O., Tucker, L.A. (1998). The effect of a single bout of strength training on ambulatory blood pressure levels in 24 mildly hypertensive men. *Am. J. Health Promot.*, 13, 69-72.

Harris, K., Holly, R. (1987). Physiological response to circuit weight training in borderline hypertensive subjects. *Med. Sci. Sports. Exerc.*, 19, 246-252.

He, J., Klag, M.J.M., Caballero, B., Appel, L.J., Charleston, J., Whelton, P.K. (1999). Plasma insulin levels and incidence of hypertension in African Americans and whites. *Arch. Intern. Med*., 159, 498-503.

He, J., Whelton, P.K., Appel, L.J., Charleston, J., Klag, M.J. (2000). Long-term effects of weight loss and dietary sodium reduction on incidence of hypertension. *Hypertension,* 35, 544-549.

Heck, H., Rost, R., Hollmann, W. (1984). Normalwerte des Blutdrucks bei der Fahrradergometrie. *Dtsch. Z. Sportmed.*, 35, 243.

Hill, D.W., Collins, M.A., Cureton, K.J., Demello, J.J. (1989). Blood pressure response after weight training exercise. *J. Appl. Sport Sci. Res.,* 3, 44-47.

Ishikawa-Takata, K., Ohta, T., Tanaka, H. (2003). How much exercise is required to reduce blood pressure in essential hypertensives: a dose-response study. *Am. J. Hypertens.,* 16, 629-633.

Jones, H., Prichard, C., George, K., Edwards, B., Atkinson, G. (2008). The acute post-exercise response of blood pressure varies with time of day. *Eur. J. Appl. Physiol*., 104, 481-489.

Karvonen, M., Kentala, K., Mustala, O. (1957). The effects of training heart rate: a longitudinal study. *Ann. Med. Exp. Biol. Fenn.,* 35, 307-315.

Kaplan, N.M. (1990). *Clinical Hypertension*. Baltimore, MD: Williams and Wilkins.

Kjeldsen, S.E., Aksnes, T.A., Fagard, R.H., Mancia, G. (2009). *Hypertension*. In A.J. Camm, T.F. Lüscher, P.W. Serruys (Eds.), ESC Textbook of Cardiovascular Medicine. 2° Ed. (pp. 437-464). Oxford University Press. England.

Kelley, G.A., McClellan, P. (1994). Antihypertensive effects of aerobic exercise: A brief meta-analysis review of randomized controlled trials. *Am. J. Hypertens.,* 7, 115-119.

Kelley, G.A. (1995). Effects of aerobic exercise in normotensive adults: a brief meta-analytic review of controlled clinical trials. *South. Med. J.,* 88, 42-46.

Kelley, G. (1996). Effects of aerobic exercise on ambulatory blood pressure: a meta-analysis. *Sport Med. Training and Rehab., 7,* 115-131.

Kelley, G. (1997). Dynamic resistance exercise and resting blood pressure in adults: a meta-analysis. *J. Appl. Physiol., 82,* 1552-1565.

Kelley, G.A. (1999). Aerobic exercise and resting blood pressure among women: a meta-analysis. *Prev. Med.,* 28, 264-275.

Kelley, G.A., Kelley, K.S. (2000). Progressive resistance exercise and resting blood pressure: a meta-analysis of randomized controlled trials. *Hypertension, 35,* 838-843.

Kelley, G.A., Kelley, K.S., Tran, Z.V. (2001). Walking and resting blood pressure in adults: a meta-analysis. *Prev. Med.,* 33, 120-127.

Kingwell, B.A. (2000). Nitric oxide-mediated metabolic regulation during exercise: effects of training in health and cardiovascular disease. *FASEB J.*, 14, 1685-1696.

Laterza, M.C., de Matos, L.D., Trombetta, I.C., Braga, A.M., Roveda, F., Alves, M.J., Krieer, E.M., Negrao, C.E., Rondom, M.U. (2007). Exercise training restores baroreflex sensitivity in never-treated hypertensive patients. *Hypertension*, 49, 1298-1306.

Law, M.R., Wald, N.J. Morris, J.K., Jordan, R.E. (2003). Value of low dose combination treatment with blood pressure lowering drugs: analysis of 354 randomized trials. *BMJ*, 326, 1427-1434.

Lewington, S., Clarke, R., Qizilbash, N., Peto, R., Collins, R. (2002). Age-specific relevance of usual blood pressure to vascular mortality. *Lancet*, 358, 1903-1913.

MacDonald, J.R., Mac Dougall, J.D., Hogben, C.D. (2000). The effects of exercise duration on post-exercise hypotension. *J. Hum. Hypertens.*, 14, 125-129.

Mac Dougall, J.D., Tuxen, D.S.D.G., Moroz, J.R., Sutton, J.R. (1985). Arterial blood pressure response to heavy resistance exercise. *J. Appl. Physiol.*, 58, 785-790.

Mac Dougall, J.D., Mc Kelvie, R.S., Moroz, D.E., Sale, D.G., McCartney, N., Buick, F. (1992). Factors affecting blood pressure during heavy weightlifting and static contractions. *J. Appl. Physiol.*, 73, 1590-1597.

Maeda, S., Miyauchi, T., Kakiyama, T., Sugawara, J., Iemitsu, M., Irukayama-Tomobe, Y., Murakami, H., Kumagai, Y., Kuno, S., Matsuda, M. (2001). Effects of exercise training of 8 weeks and detraining on plasma levels of endothelium-derived factors, endothelin-1 and nitric oxide, in healthy young humans. *Life Sci.*, 69, 1005-1016.

Mairona, A., O'Driscoll, G.,Taylor, R., Green, D. (2003). Exercise and the nitric oxide vasodilator system. *Sport Med.*, 33, 1013-1035.

Manolio, T.A., Burke, G.L., Savage, P.J., Gardin, J.M., Oberman, A. (1994). Exercise blood pressure response and 5-years risk of elevated blood pressure in a cohort of young adults: the CARDIA study. *Am. J. Hypertens.*, 7, 234-241.

Marchiando, R.J., Elston, M. P. (2003). Automated ambulatory blood pressure monitoring: clinical utility in the family practice setting. *Am. Fam. Physicians.*, 67: 2343-2350.

Mathews, C.E., Pate, R.R., Jackson, K.L., Ward, D.S., Macera, C.A., Kohl, H.W., Blair, S.N. (1998). *J. Clin. Epidemiol.*, 51, 29-35.

Mc Cartney, N., Mc Kelvie, R.S., Martin, J. (1993). Weight-training-induced attenuation of the circulatory response of older males to weight lifting. *J. Appl. Physiol.*, 74, 1056-1060.

McGuigan, M.R.M., Bronks, R., Newton, R.U., Sharman, M.J., Graham, J.O., Cody, D.V., Kraemer, W.J. (2001). Resistance training in patients with peripheral

arterial disease: effects on myosin isoforms, fibre type distribution and capillary supply to skeletal muscle. *J. Gerontol. A. Biol. Sci. Med. Sci.*, 56, B302-310.

Melo, C.M., Alenear Filho, A.C., Tinucci, T., Mion, D. Jr., Forjaz, C.L. (2006). Postexercise hypotension induced by low-intensity resistance exercise in hypertensive women receiving captopril. *Blood Press Monit.*, 11:183-189.

Messerli, F. (2007). Hypertension, uncontrolled and conquering the world. *Lancet,* 370-539.

Mohrman, D.E., Heller L.J. (2006). *Cardiovascular Physiology*. (6th Ed) United States of America: McGraw-Hill Companies, Inc.

Mundal, R., Kjeldsen, S.E., Sandvik, L., Erikssen, G., Thaulow, E., Erikssen, J. (1996). Exercise blood pressure predicts mortality from myocardial infarction. *Hypertension,* 27, 324-329.

Murphy, H.M; Nevill, A.M., Murtagh, E.M., Holder, R.L. (2007). The effect of walking on fitness, fatness and resting blood pressure: a meta-analysis of randomized, controlled trials. *Prev. Med.,* 44, 377-385.

O'Brien, E., Coats, A., Owens, P., Petrie, J., Padfield, P.L., Littler, W.A., de Swiet, M, Mee, F. (2000). Use and interpretation of ambulatory blood pressure monitoring: recommendations of the British Hypertension Society. *BMJ*, 320, 1128-1134.

O'Brien, E., Asmar, R., Beilin, L., Imai, Y., Mallion, J.M., Mancia, G., Mengden, T., Myers, M., Padfield, P., Palatini, P., Parati, G., Pickering, T., Redon, J., Staessen, J., Stergiou, G., Verdecchia, P. (2003). European Society of Hypertension recommendations for conventional, ambulatory and home blood pressure measurement. *J. Hypertens.*, 21, 821-848.

Pescatello, L.S., Fargo, A.E., Leach, Jr C.N., et al. (1991). Short term effect of dynamic exercise on arterial blood pressure. *Circulation*, 83, 1557-1561.

Pescatello, L.S., Miller, B., Danias, P.G., Werner, M., Hess, M., Baker, C., Jane De Souza, M. (1999). Dynamic exercise normalizes resting blood pressure in mildly hypertensive premenopausal women. *Am. Heart J*., 138 (5Pt 1), 916-921.

Pescatello, L.S., Kulikowich, J.M. (2001). The after effects of dynamic exercise on ambulatory blood pressure. *Med. Sci. Sports Exerc*., 33, 1855-1861.

Pescatello, L.S., Franklin, B.A., Fagard, R., Farquhar, W.B., Kelley, G.A., Ray, C.A. (2004). American College of Sport Medicine, position stand: Exercise and Hypertension. *Med. Sci. Sport Exerc., 36*, 533-553.

Pescatello, L.S., Guidry, M.A., Blanchard, B.E., Kerr, A., Taylor, A.L., Johnson, A.N., Maresh, C.M., Rodríguez, N., Thompson, P.D. (2004). Exercise intensity alters postexercise hypotensioon. *J. Hypertens*., 22, 1881-1888.

Pescatello, L.S., Blanchard, B.E., Van Heest, J.L., Maresh, C.M., Gordish-Dressman, H., Thompson. P.D. (2008). The metabolic syndrome and the immediate antihypertensive effects of aerobic exercise: a randomized control design. *BMC Cardiovasc. Disord*., 10; 8:12.

Predel, H.G. (2007). Bluthochdruck und Sport. *Dtsch. Z. Sportmed.*, 9, 328-333.

Pickering, T.G., James, G.D., Boddie, C., Harshfield, G.A. (1988). How common is white coat hypertension? *JAMA, 259,* 225-228.

Pickering, T.G., Phil, D., Shimbo, D., Hass, D. (2006). Ambulatory blood pressure monitoring. *N. Engl. J. Med.,* 354, 2368-2374.

Pollock, M.L., Gaesser, G.A., Butcher, J.D., Després, J.P., Dishman, R.K., Franklin, B.A., Garber, C.E. (1998). American College of Sport Medicine Position Stand: the recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness and flexibility in healthy adults. *Med. Sci. Sport Exerc*., 30, 975-991.

Pollock, M.L., Franklin, B.A., Balady, G.J., Chaitman, B.L., Fleg, J.L., Fletcher, B., Limacher, M., Piña, I.L., Stein, R.A., Williams, M., Bazzarre, T. (2000). Resistance exercise in individuals with and without cardiovascular disease: benefits, rationale, safety and prescription. An advisory from the Committee on Exercise, Rehabilitation and Prevention, Council on Clinical Cardiology, American Heat Association. *Circulation,* 101, 828-833.

Queiroz, A.C.C., Gagliardi, J.F.L., Forjaz, C.L.M., Rezk, C.C. (2009). Clinic and ambulatory blood pressure responses after resistance exercise. *J. Strength Cond. Res*., 23, 571-578.

Quinn.T.J. (2000). Twenty four hour, ambulatory blood pressure responses following acute exercise: impact of exercise intensity. *J. Hum. Hypertens.* 14, 547-553.

Rao, S.P., Collins, H.L., Dicarlo, S.E. (2002). Postexercise alpha-adrenergic receptor hyporesposiveness in hypertensive rats is due to nitric oxide. *Am. J. Physiol. Regul. Integr. Comp. Physiol*., 282, R960-R968.

Rashid, M., Fuentes, F., Touchon, R., Wehner, P. (2003). Obesity and the risk for cardiovascular disease. *Prev. Cardiol.*, 6, 42-47.

Sale, D.G. (1988). Neural adaptation to resistance training. *Med. Sci. Sports Exerc*. 20: S135-S145.

Sega, R., Facchetti, R., Bombelli, M., Cesana, G., Corrao, G., Grassi, G., Mancia, G. (2005). Prognostic value of ambulatory and home blood pressures compared with office blood pressure in the general population: follow-up results from the Pressioni Arteriose Monitorate e Loro Associazioni (PAMELA) study. *Circulation*, 111, 1777-1783.

Scher, L.M., Ferriolli, E., Moriguti, J.C., Scher, R., Lima, N.K. (2010). The effect of different volumes of acute resistance exercise on elderly individuals with treated hypertension. *J. Strength Cond. Res.* 22 (Epub ahead of print).

Singh, J.P., Larsoon, M.G., Manoli, T.A., O'Donnell, C.J., Lauer, M., Evans, J.C., Levy, D. (1999). Blood pressure response during treadmill testing as a risk factor for new-onset hypertension. The Framingham heart study. *Circulation*, 99, 1831-1836.

Staessen, J.A., Thijs, L., Fagard, R., O'Brien, E.T., Clement, T., de Leeuw, P.W., Mancia, G., Nachev, C., Palatini, P., Parati, G., Tuomilehto, J., Webster, J. (1999). Predictiong cardiovascular risk using conventional vs ambulatory blood pressure in older patients with systolic hypertension. *JAMA*, 282, 539-546.

Staessen, J.A., O'Brien, E.T., Thijs, L., Fagard, R.H. (2000). Modern approaches to blood pressure measurement. *Occup. Environ. Med.*, 57, 510-320.

Strasser, B., Haber, P., Strehblow, D., Cauza, E. (2008). The benefits of strength training on arterial blood pressure in patients with type 2 diabetes mellitus measured with ambulatory 24-hour blood pressure systems. *Wien. Med. Wochenschr*., 158, 379-384.

Thibonnier. M. (1992). Ambulatory blood pressure monitoring. When is it warranted? *Postgrad. Med., 91*, 263-272.

Thompson, P.D., Crouse, S.F., Goodpaster, B., Kelley, D., Moyna, N., Pescatello, L. (2001).The acute versus the chronic response to exercise. *Med. Sci. Sports Exerc., 33 (suppl 6)*, S438-S445.

Trombetta, I.C., Betalha, L.T., Rondon, M.U., Laterza, M.C., Kuniyoshi, F.H., Gowdak, M.M., Barretto, A.C., Halpern, A., Villares, S.M., Negrão, C.E. (2003). Weight loss improves neurovascular and muscle metaboreflex control in obesity. *Am. J. Physiol. Heart Circ. Physiol.*, 285, H974-H982.

Van Hoof, R., Macor, F., Lijnen, P., Staessen, J., Thijs, L., Vanhees, L. et al. (1996). Effect of strength training on blood pressure measured in various conditions in sedentary men. *Int. J. Sports Med.*, 17, 415-422.

Verdecchia, P., Porcellati, C., Schillaci, G., Borgiioni, C., Ciucci, A., Battistelli, M., Guerrieri, M., Gateschi, C., Zampi, I., Santucci, A., Santucci, C., Revoldi, G. (1994). Ambulatory blood pressure: an independent predictor of prognosis in essential hypertension. *Hypertension*, 24, 793-801.

Verdecchia, P. (2000). Prognostic value of ambulatory blood pressure: current evidence and clinical implications. *Hypertension*, 35, 844-851.

Vollmer, W.M., Sacks, F.M., Ard, J., Appel, L.J., Bray, G.A., Simons-Morton, D.G., Conlin, P.R., Svetkey, L.P., Erlinger, T.P., Moore, T.J., Karanja, N., DASH Collaborative Research Group. (2001). Effects of diet and sodium intake on blood pressure: subgroup analysis of the DASH-sodium trial. *Ann. Intern. Med.*, 135, 1019-1028.

Wallace, J.P., Bogle, P.G., King, B.A., Krasnoff, J.B., Jastremski, C.A. (1999). The magnitude and duration of ambulatory blood pressure reduction following acute exercise. *J. Hum. Hypertens*., 13, 361-366.

Whelton, S.P., He, J., Appel, L.J., Cutler, J.A., Havas, S., Kotchen, T.A, et al. (2002a). Primary prevention of hypertension: clinical and public health advisory from the National High Blood Pressure Education Program. *JAMA, 288*, 1882-1888.

Whelton, S.P., Chin, A., Xin, X., He, J. (2002b). Effect of aerobic exercise on blood pressure: a meta-analysis of randomized, controlled trials. *Ann. Intern. Med.*, 136, 493-503.

Wendelin-Saarenhovi, M., Isoaho, R., Hartiala, J., Helenius, H., Kivelä, S-L; Hietanen, E (2001). Long-term reproducibility of ambulatory blood pressure in unselected elderly subjects. *Clin. Physiol.*, 21, 3, 316-323.

Westhoff, T.H., Franke, N., Schmidt, S., Vallvracht-Israng, K., Meissner, R., Yildirim, H., Schlattmann, P., Zideck, W., Dimeo, F., van der Giet, M. (2007). Too old to benefit from sports? The cardiovascular Effects of Exercise Training in Elderly Subjects Treated for Isolated Systolic Hypertension. *Kidney and Blood Press. Res., 30,* 240-247.

Williams, M.A., Haskell, W.L., Ades, P. A., Amsterdam, E.A., Bittner, V., Franklin, B.A., Gulanick, M., Laing, S.T., Stewart, K.J. (2007). Resistance Exercise in Individuals with and without cardiovascular diseases: 2007 Update. A scientific statement from the American Heart Association Council on Clinical Cardiology and Council on Nutrition, Physical Activity and Metabolism. *Circulation*, 116, 572-584.

Willmore, J.H., Costill, D.L. (2005). Physiology of Sport and Exercise. (3rd Ed). Champaign, IL; Human Kinetics,

WHO (1995). Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. WHO Technical Report Series 854. Geneva: World Health Organization.

Xin, X., He, J., Frontini, M.G., Ogden, L.G., Motsamai, O.I., Whelton, P.K. (2001). Effects of alcohol reduction on blood pressure: a meta-analysis of randomized controlled trials. *Hypertension*, 38, 1112-1117.

CURRICULUM VITAE

PERSONAL INFORMATION:

Name:	Andrea Solera Herrera
Nationality:	Costa Rican
Date of birth:	19.12.1975
Place of birth:	San José, Costa Rica
Native language:	Spanish
Foreign language:	German (intermediate level) English (intermediate level)

EDUCATION :

1982 until 1987:	Elementary School: "Escuela República de Guatemala", Alajuela, Costa Rica
1988 until 1992:	Grammar School: "Instituto de Alajuela", Alajuela, Costa Rica
1993 until 1997:	Bachelor in Physical Education at the Universidad de Costa Rica, San José, Costa Rica
1998 until 2000:	Master´s Program in Human Movement Science at the Universidad de Costa Rica, San José, Costa Rica
2005 until 2006:	International Coaching Course, from August 29th 2005 to January 30th 2006 in the specialized field of Basketball, at the Sports Scientific Faculty of the University Leipzig, Leipzig, Germany
2007 until 2011:	Ph.D. at the German Sport University Cologne, Germany

WORK EXPERIENCE:

2001 until 2003: Costa Rican Sport Institute (ICODER)

- *Instructing and supervising* the National Senior's Exercises Program

1999 until 2007: Universidad de Costa Rica (UCR)

- *Teaching*: Research Methods, Statistics in undergraduate and graduate programs
- *Teaching*: Basketball, Swimming, Popular Dance, at undergraduate levels
- *Coordinating*: Symposiums and Conferences in the physical activities field
- *Developing*: research projects

AWARDS:

1998: Best student's score at the Physical Education School at the Universidad de Costa Rica

2000: Honors score in the Academics Master's Program of the Universidad de Costa Rica

2001: First prize for the best research project presented at the IV National Physical Education Congress. San José, Costa Rica

2009: Young investigator award for the research project presented at the 33th Congress of the Germany Blood Pressure League

INTERNATIONAL CONGRESS PRESENTATIONS:

1999: Speaker at the 46th Annual Meeting of the American College of Sport Medicine. Seattle, USA. Topic: "Influence of dehydration and rehydration on cognitive processes"

2003: Speaker at the 50th Annual Meeting of the American College of Sport Medicine. San Francisco, USA. Topic: "Influence of dehydration and rehydration on basketball free throw accuracy"

2004: Speaker at the 51st Annual Meeting of the American College of Sport Medicine. Indianapolis, USA. Topic: "Fluid balance during exercise in the heat: are there predictors of overhydrations?"

2009: Speaker at the 33th Congress of the Germany Blood Pressure League. Topic: "The hemodynamic effect of 12 weeks aerobic exercise vs resistance exercise vs the combination aerobic-resistance exercise on patients with arterial hypertension"

WORKSHOP PARTICIPATIONS:

2005: Participant in the course Arterial Hypertension: implications, treatment and prevention.

SCHOLARSHIPS:

2005: Scholarship from the Faculty of Sports Sciences at the Universität Leipzig, Germany.

2006: Scholarship from the German Academic Exchange Service (DAAD).

PUBLICATIONS:

Solera Herrera, A; Aragón Vargas, L .F. (2006). Deshidratación y sobrehidratación voluntaria durante el ejercicio en el calor: posibles factores relacionados. *Revista de Ciencias del Ejercicio y la Salud,* 4(1), 22-23.

Solera, A. (2003). Efectos de la deshidratación y la rehidratación sobre la precisión del tiro libre de baloncesto. *Revista de Ciencias del Ejercicio y la Salud* 3(1), 35-42. Topic: "Influence of dehydration and rehydration on basketball free throw accuracy"

Moncada, J; Solera, A; Salazar W. (2002). Fuentes de varianza e índices de varianza explicada en las ciencias del movimiento humano. *Revista de Ciencias del Ejercicio y la Salud* 2(2), 70-74. Topic: "Sources of explained variance in movement science"

Solera, A; Salazar W. (2001). Efectos de la deshidratación y la rehidratación sobre los procesos cognitivos de tiempo de reacción, memoria auditiva y percepción visual. *Revista de Ciencias del Ejercicio y la Salud* 1(1), 1-9. Topic: "Effects of dehydration and rehydration on cognitive processes: reaction time, auditive memory and visual perception".

INTERESTS:

- Continue teaching and researching at the Universidad de Costa Rica (UCR).
- Continue researching with elderly population and persons with chronic diseases.

SUMMARY

The present thesis had the purpose to clarify, in two randomized controlled trials, the acute and chronic effects of aerobic training vs. resistance training vs. the combination of both on the ambulatory blood pressure monitoring (ABPM).

Chronic effect: 62 sedentary patients (17 females and 45 males, aged 54.4 ± 11.4 yrs.), with grade I hypertension without antihypertensive medication were randomly-allocated to 4 groups: (1) AG: aerobic training group; (2) RG: resistance training group; (3) ARG: aerobic-resistance training group; (4) CG: control group. Apart from the CG, all subjects trained 12-wk, 3 d/wk at 50-75% intensity. Before and after the experimental phase were measured the 24-h systolic and diastolic ambulatory blood pressure (sABPM and dABPM, respectively). The two-way ANOVA detected a significant sABPM reduction following AG, RG and ARG (-3.29, -4.93 and -5.82 mmHg respectively, p=0.03), whereas BP of the CG remained unchanged. A trend towards reduction on the 24-h dABPM was found after three exercise interventions compared to CG; however, this change did not reach statistical significance (p=0.16).

Acute effect: 17 trained subjects (15 males and 2 females, aged 55.06 ± 12.26 yrs.) with pre-hypertension or Grade I Hypertension without antihypertensive medication participated in 4 training treatments, each carried out on different days under a randomized repeated measures design as follows: (1) AC: aerobic training condition (30-min cycling at 70% $HR_{reserve}$); (2) RC: resistance training condition (2 sets of 14 exercises at 70% 1RM); (3) ARC: aerobic and resistance training condition; and (4) CC: control condition (30-min resting). Resting BP was measured 5-min before and 5-min following each experimental condition, followed by a 24-hr ABPM. One-way ANOVA showed no significant differences between the 24-hr ABPM for the experimental conditions. In addition, a two-way ANOVA detected no significant interaction between the 4 conditions and the following 6-hr sABPM (p=0.23), but a significant interaction on the 6-hr dABPM (p=0.02). Post-hoc analyses showed that the diastolic significant reduction lasted one-hour with the RC and two-hours following the AC and ARC.

Conclusion: resistance training alone or in combination with aerobic training reduces the sABPM following 12-wk of exercise and the dABPM during 1-h after training in patients with mild hypertension.

ZUSAMMENFASSUNG

Diese Dissertation hatte das Ziel, die chronische und die akute Auswirkung des Ausdauertrainings bzw. Krafttrainings sowie einer Kombination aus beiden auf den Langzeitblutdruck (ABPM) zu untersuchen.

Chronischer Effekt: 62 sportlich inaktive Patienten (Alter: 54.4±11.4 J) mit arterieller Hypertonie Grad I, die keine antihypertensive Medikation in den letzten drei Monaten eingenommen hatten, wurden in 4 Gruppen randomisiert: (1) AT (Ausdauertraining), (2) KT (Krafttraining), (3) AKT (kombiniertes Ausdauer- und Krafttraining) und (4) KG (Kontrollgruppe). Das Training erfolgte individualisiert in vergleichbarer Dosierung (Training 3x/Woche) und Intensität (50-75% maximale Intensität). Vor und nach den 12 Trainingswochen wurde der 24 Std. systolischen und diastolischen Langzeitblutdruck (sABDM und dABDM) gemessen. Mittels zweifaktorieller Varianzanalysen wurde bestimmt, dass die 3 Trainingsformen eine signifikante Reduktion des sABDM erreichten (-3,29, -4.93 and -5.98 mmHg bzw. p=0.03) im Gegensatz zur Kontrollgruppe, deren sABDM keine Reduktion zeigte. Bei der dABDM gab es keine Reduktion, obwohl die Werte der 3 Trainingsgruppen zu einer Senkung neigen (p=0.16). **Akuter Effekt**: 17 sportlich aktive Patienten (15 Männer und 2 Frauen, Alter: 55±12 J), mit hoch-normalem Blutdruck aber ohne antihypertensive Medikation nahmen an der Studie teil. Vier Untersuchungseinheiten wurden an vier verschiedenen Tagen unter einer randomisierten Messwiederholung durchgeführt: (1) AT: 30-min Radfahren bei 70% HF-Reserve; (2) KT: 2 mal 14 Einzelübungen bei 70% 1RM, (3) AKT: beide Trainingsformen (Ausdauer und Krafttraining zusammen) (4) KK: Kontrollkondition 30 min in Ruhe. Die BP-Messungen erfolgten 5 min vor und 5 min nach jeder Einheit, gefolgt von einer 24 Std. ABDM. Die einfaktorielle Varianzanalyse zeigt keine Unterschiede zwischen dem Mittelwert des Langzeitblutdrucks nach den 4 Trainingsformen, und zwar weder die sABPM noch die dABPM. Mittels zweifaktorieller Varianzanalyse wurde eine signifikante Interaktion auf den diastolischen 6 Stunden-Blutdruck (p = 0.02) bestimmt. Post-hoc-Analysen zeigten, dass die diastolische signifikante Reduktion 1 Stunde nach dem KT (-6 mmHg) und zwei Stunden nach dem AT (-3.5 mmHg) und AKT (-5mmHg) andauerte.

Schlussfolgerung: Krafttraining allein, bzw. in Kombination mit Ausdauertraining ist in der Lage die sABDM nach 12 Trainingswochen und die dABDM sofort nach dem Training zu reduzieren. Dies gilt für Patienten mit milder arterieller Hypertonie.