THE MAGNITUDE AND DURATION OF POST EXERCISE HYPOTENSION AFTER LAND AND WATER EXERCISE

BY

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DECLARATION

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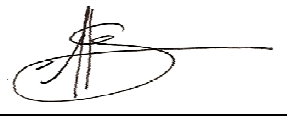
Signature: 

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SUMMARY

It is well-known that acute and chronic aerobic and resistance exercise results in decreased blood pressure (BP) in hypertensive individuals. There is little evidence that water exercise has a similar effect on BP response. There is also no certainty regarding the magnitude and duration of post exercise hypotension (PEH) after either land or water-based exercise. Most studies were also performed under controlled laboratory conditions and very few characterised the PEH response under real life conditions.

The current study endeavoured to examine the magnitude and duration of PEH after an acute session of water- and land-based exercise during free living conditions in persons with mild to moderate hypertension.

Twenty-one men and women (aged 52 ± 10 years) volunteered for the study. All participants were pre-hypertensive or hypertensive. Participants completed a no exercise control session, a water exercise session and a combined aerobic and resistance land exercise session in random order. After all three sessions, participants underwent 24 hour monitoring using an Ergoscan ambulatory BP monitoring device. Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and heart rate (HR) were monitored to determine changes from resting values after each session and to compare the PEH responses between land and water exercise.

Overall, the land exercise treatment caused a 3.6 mmHg lower average SBP over 24 hours than the control treatment ($P = 0.04$). The average difference over 24 hours between the water and control treatments was 2.2 mmHg and between land and water exercise it was 1.5 mmHg ($P > 0.05$). During daytime, both land and water exercise resulted in significantly lower SBP (12.7 and 11.3 mmHg) compared to the control session (2.3 mmHg). The PEH response lasted for 24 hours after land exercise and nine hours after water exercise. There was no difference in the daytime DBP for the three treatments ($P > 0.05$). Although all three groups showed significant reductions during night time, both exercise treatments showed greater nocturnal falls in SBP, DBP and MAP than the control treatment. There were

significant correlations between changes in the work day SBP after land ($r = 0.46$) and water ($r = 0.47$) exercise and control day SBP.

The results show that water and land-based exercise result in significant PEH during daytime hours. Although there was no significant difference in the magnitude of PEH between the land and water treatments, it seemed that land exercise resulted in a longer duration of PEH than the water exercise. This is the first study to show that the magnitude of the PEH response is similar for land and water exercise, although the duration of PEH may be longer for land exercise. These results suggest that water exercise is a safe alternative exercise modality for hypertensive individuals. Furthermore, it was shown that the magnitude of the change in SBP after land and water exercise is related to resting SBP value. Therefore, not only will individuals with the highest pre-exercise SBP values benefit the most from exercise, but they will also benefit greatly by morning exercising.

OPSOMMING

Dit is alombekend dat akute en chroniese aërobiese- en weerstandsoefening tot ‘n afname in bloeddruk (BD) lei in persone met hipertensie. Daar is egter min getuienis dat wateroefening dieselfde effek op die bloeddruk respons het. Daar is ook nie sekerheid oor die grootte en duur van post-oefening hipotensie na water- of landoefening nie. Die meeste studies is onder gekontrolleerde laboratorium omstandighede gedoen en min resultate is beskikbaar onder alledaagse lewensomstandighede.

Die huidige studie het gepoog om die grootte en duur van die post-oefening hipotensie respons in persone met ligte tot matige hipertensie onder alledaagse omstandighede na ‘n akute sessie van water- en landgebaseerde oefening te ondersoek.

Een-en-twintig mans en vrouens (ouderdom 52 ± 10 jaar) het ingewillig om aan die studie deel te neem. Alle deelnemers was hipertensief of pre-hipertensief. Alle deelnemers het ‘n kontrolesessie, ‘n wateroefeningsessie en ‘n gekombineerde aërobiese en weerstands landoefensessie, in lukrake volgorde, voltooi. Na elke sessie het die deelnemers 24 uur bloeddrukmonitering met ‘n Ergoscan wandelende bloeddruk monitor ondergaan. Sistoliese bloeddruk (SBD), diastoliese bloeddruk (DBD), gemiddelde arteriële bloeddruk en harttempo (HT) is gemonitor om die veranderinge vanaf rustende waardes na elke sessie te bepaal en om die hipotensiewe respons na land- en wateroefening te vergelyk.

Landoefening het ‘n 3.6 mmHg laer gemiddelde SBD oor 24 uur tot gevolg gehad in vergelyking met die kontrolesessie (P = 0.04). Die gemiddelde verskil oor 24 uur tussen die water- en kontrolesessies was 2.2 mmHg en 1.5 mmHg tussen die land en water oefensessies (P > 0.05). Gedurende die dag het beide die land- and wateroefening gelei tot beduidende laer SBD (12.7 en 11.3 mmHg) in vergelyking met die kontrolesessie (2.3 mmHg). Die post-oefening hipotensie het 24 uur geduur na die landoefening en nege uur na die wateroefening. Daar was geen verskil in DBD gedurende die dag tussen die drie groepe nie (P > 0.05). Alhoewel al drie groepe beduidende afnames in SBD, DBD en gemiddelde arteriële BD gedurende

die nag getoon het, het albei oefensessies groter dalings gedurende die nag tot gevolg gehad, in vergelyking met die kontrolesessie. Daar was beduidende korrelasies tussen die verandering in werksdag SBD na land ($r = 0.46$) en water ($r = 0.47$) oefening en kontrole dag SBD.

Die resultate dui daarop dat beide water- en landoefening beduidende post-oefening hipotensie gedurende die dag tot gevolg het. Alhoewel daar geen verskil in die grootte van die hipotensie tussen land- en wateroefeninge was nie, het die hipotensie na die landoefeningsessie langer geduur as na wateroefening. Hierdie is die eerste studie wat wys dat die grootte van die post-oefenings hipotensie soortgelyk is na water- en landoefening, alhoewel die duur van die hipotensie langer is na die landoefening. Die resultate dui daarop dat wateroefening ‘n veilige alternatiewe oefenmodaliteit is vir individue met matige hipertensie. Verder is gewys dat die grootte van die verandering in SBD na land en water oefening verwant is aan die rustende SBD. Dus sal individue met die hoogste rustende SBD die meeste baat by oefening, veral oefening in die oggend.

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LIST OF ABBREVIATIONS AND ACRONYMS

°	:	Degree
℃	:	Degrees Celsius
µA	:	Micro ampere
10RM	:	Ten repetition maximum
1RM	:	One repetition maximum
AAMI	:	The Association for the Advancement of Medical Instrumentation
ABP	:	Ambulatory blood pressure
ACE	:	Angiotensin converting enzyme
ACSM	:	American College of Sports Medicine
ADL	:	Activities of daily living
ANOVA	:	Analysis of variance
ATP	:	Adenosine triphosphate
$b.min^{-1}$	:	Beats per minute
BHS	:	British Hypertension Society
BIA	:	Bio-electrical impedance analysis
BMI	:	Body mass index
BP	:	Blood pressure
CAD	:	Coronary artery disease
Cm	:	Centimetre
CO	:	Cardiac output
CO_2	:	Carbon dioxide
CVD	:	Cardiovascular disease
DBP	:	Diastolic blood pressure
e.g.	:	For example

ECG	:	Electrocardiogram
ESH	:	European Society of Hypertension
h	:	Hour(s)
HDL	:	High density lipoproteins
HR	:	Heart rate
HR_{max}	:	Maximum heart rate
HRR	:	Heart rate reserve
ISAK	:	International Standards for the Advancement of Kinanthropometry
JNC-VII	:	Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure
Kcal	:	Kilocalories
$kg.m^{-2}$	:	Kilogram per square metre
kg	:	Kilogram(s)
kHz	:	Kilo hertz
$km.h^{-1}$	:	Kilometres per hour
L	:	Litre
LDL	:	Low density lipoproteins
LSD	:	Least significant difference
MAP	:	Mean arterial pressure
$ml.kg^{-1}.min^{-1}$	:	Millilitres per kilogram body weight per minute
$ml.min^{-1}$	:	Millimetres per minute
ml	:	Millilitres
mm	:	millimetre
mmHg	:	Millimetres mercury
$mmol.L^{-1}$	:	millimol per litre
MRC	:	Medical Research Council
n	:	Sample size

N_2	:	Nitrogen
NSCA	:	National Strength and Conditioning Association
O_2	:	Oxygen
P	:	Probability
PEH	:	Post exercise hypotension
POMS	:	Profile of mood states
PVC	:	Premature ventricular contractions
R	:	Respiratory quotient
r^4	:	Fourth power
RPE	:	Rate of perceived exertion
RPP	:	Rate pressure product / double product
SA	:	Sino atrial
SBP	:	Systolic blood pressure
SD	:	Standard deviation
SNA	:	Sympathetic nervous system activity
SpO_2	:	Oxygen saturation
SV	:	Stroke volume
SVR	:	Systemic vascular resistance
TPR	:	Total peripheral resistance
VO_2	:	Oxygen consumption
VO_{2max}	:	Maximum oxygen consumption
VO_{2peak}	:	Peak oxygen consumption
V_T	:	Ventilatory threshold

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CHAPTER ONE

INTRODUCTION

The World Health Organization defines health as follows: “a state of complete physical, mental and social well-being, not merely the absence of disease and infirmity” (Callahan, 1973). This definition excludes a large part of the world population as a significant number are affected by some or other chronic disease. Over the last few decades, populations worldwide have adopted urbanized lifestyles with changes in dietary habits such as increased fat and refined carbohydrate intake, increased stress and a reduction in physical activity and exercise. This has resulted in an epidemic of obesity, type 2 diabetes mellitus, hypertension and other chronic diseases (Steyn *et al*., 2008; Derman, 2008). Hypertension is one of the most prevalent chronic disorders worldwide (Chobanian *et al*., 2003).

Persons with chronic conditions often become physically inactive and / or deconditioned. This may lead to or contribute to an already impaired cardiac function, reduced muscle mass, muscle strength and exercise capacity, as well as an increased percentage body fat (Painter, 2008). This vicious cycle of deconditioning and inactivity will worsen the prognosis of the disease, ultimately resulting in an inability to perform functions of daily living, thereby negatively impacting on quality of life and further the cardiovascular risk for fatal cardiac events.

A. CLASSIFICATION OF HYPERTENSION

Hypertension is one of the major risk factors for developing cardiovascular disease and is a major health problem affecting 1 billion people globally (American College of Sports Medicine, 2006). Table 1 depicts the classification of blood pressure for adults as described by the American College of Sports Medicine (2006) and the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (Chobanian *et al*., 2003). Hypertension is classified as a systolic blood pressure (SBP) ≥ 140 mmHg and / or a diastolic blood pressure (DBP) ≥ 90 mmHg or receiving anti-hypertensive treatment.

Table 1: The classification of blood pressure (BP) for adults

BP classification	SBP (mmHg)	DBP (mmHg)	Treatment (with no other risk factors)
Normal	< 120	And < 80	Lifestyle modification
Prehypertension	120 - 139	Or 80 – 89	Lifestyle modification
Stage 1 Hypertension	140 - 159	Or 90 – 99	Lifestyle modification (6 – 12 months) and
Stage 2 Hypertension	≥ 160	Or ≥ 100	Drug therapy

BP, blood pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; mmHg, millimetre mercury

(The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, 2003)

B. CAUSES OF HYPERTENSION

Most cases (90% - 95%) of hypertension are categorized as primary hypertension and are believed to be idiopathic. Secondary hypertension is caused by endocrine or structural disorders (Brooks *et al*., 2000). Primary hypertension is hardly ever the result of a single factor, but rather a combination of genetic and environmental causes (Fagard, 2005). Common causes of primary hypertension are family history, obesity, inactivity, high sodium intake, excessive alcohol intake, psychological stress, vascular hypertrophy as well as diabetes and insulin resistance (Chiong, 2008; Steyn *et al*., 2008; Wilmore & Costill, 2004). All of the causes mentioned above will either increase cardiac output or total peripheral resistance, or both, resulting in an increased BP. An increased activity of the sympathetic nervous system and renin-angiotensin system is also associated with hypertensive persons (Pescatello *et al*., 2004).

From the available literature it can be deduced that most of the causes of hypertension can be addressed or even eliminated by changes in lifestyle, like increased physical activity, decreased intake of processed foods and stress management. Unfortunately, these adaptations are hardly applied with as much enthusiasm as pharmacological treatment. Patients are not always aware of the great impact lifestyle changes could have on chronic diseases like hypertension.

C. PREVALENCE OF HYPERTENSION

A survey done by the Medical Research Council (MRC) in 2006 showed that 55% of the South African population has hypertension. Of that group only 10% of men and 18% of women had controlled hypertension with a higher prevalence of hypertension in women (Steyn *et al*., 2006). On average about 50% of hypertensive individuals are not even aware that they have hypertension. Hypertension can go undiagnosed until it is too late, because in most cases hypertension is asymptomatic. However, hypertension can also result in some early signs and symptoms including headaches, vision change, ringing in the ears or a tingling sensation in the hands and feet. These symptoms can also be seen in normotensive individuals (Chiong, 2008).

Steyn *et al*. (2008) reported that persons with a high BMI, a family history of hypertension, obesity, stroke or excessive alcohol use were at higher risk of developing hypertension. It was also reported that persons with higher levels of education are less likely to have hypertension. Due to lifestyle habits the wealthiest quintile of the population is at higher risk than the poorer population quintiles. Furthermore, Steyn *et al*. (2008) reported that although there was no difference in the prevalence of hypertension between different ethnic groups, other socio-demographic parameter differences did account for differences in the prevalence of hypertension. This suggests that an urbanised lifestyle characterized by low physical activity and large intakes of processed foods are the main causes of hypertension.

Of major concern for the health sector is the poor level of control of hypertension which may lead to complications such as left ventricular hypertrophy and accelerated atherosclerosis resulting in high rates of cardiovascular disease (Chiong, 2008; Pescatello *et al*., 2004; Steyn *et al*., 2006; Tsai *et al*., 2002). In South Africa, research has shown that persons with a higher income had better control of hypertension than the lower income group because of private medical care and medical aid insurance. The MRC survey (2006) also showed that the older population is more aware of hypertension and health than the younger population and therefore had better control of hypertension. This is of particular concern because the younger generation may be exposed to hypertension for a long period of

time which may cause serious damage to the eyes, kidneys, coronary and cerebral arteries (Steyn *et al.*, 2008; 2006).

The high prevalence and low control of hypertension is a major concern worldwide, because the financial implications of damage control or crisis management are daunting. These negative elements could be prevented by early identification and correction of unhealthy lifestyles. Unfortunately, most people will only realize this once it is too late.

D. HYPERTENSION AND ASSOCIATED RISKS

Hypertension is one of the leading risk factors for coronary artery disease, congestive heart failure, peripheral vascular disease and renal insufficiency due to the destructive effect of an elevated BP on the arterial vessels. Hypertension contributes to atherosclerosis by damaging the vascular endothelial cells due to torsion, lateral wall pressure and shear force. This allows filtration of lipids into the arteriosclerotic lesions (Brooks *et al.*, 2000).

The risk of developing most of these diseases can be well below the diagnostic threshold of 140 / 90 mmHg. A positive relation between cardiovascular risk and blood pressure occurs at a blood pressure as low as 115 / 75 mmHg and the risk doubles for each 20 mmHg increase in SBP and / or 10 mmHg increase in DBP. Furthermore, it is known that SBP continues to increase throughout adult life, whereas DBP increases until the fifth decade and thereafter levels off or decreases slightly. Research has shown that, depending on the initial BP, BP will increase between 26% and 50% in adults over the age of 65 over a period of four years (Chobanian *et al.*, 2003). Older adults are therefore at greater risk of developing isolated systolic hypertension. In the past the danger of isolated systolic hypertension has been overlooked because the focus was mainly on the risk of elevated DBP on the cardiovascular system. However, it is gaining attention as a risk factor for cardiovascular disease, especially in the elderly (Kannel, 1999). Most persons have a 90% lifetime risk of developing hypertension after the age of 55, irrespective of BP levels at a younger age (Pescatello *et al.*, 2004; Chobanian *et al.*,

2003). Therefore, controlling and preventing hypertension is a lifelong pursuit of healthy dietary and exercise habits.

E. THE CONTROL OF HYPERTENSION

Hypertension can affect quality of life negatively, especially in later years if BP is not controlled. According to Touyz *et al*. (2004), a reduction in BP of only 3 mmHg in the general population can decrease stroke mortality and coronary artery disease mortality by 8% and 5% respectively. Thus, lowering blood pressure, preferably through lifestyle modifications, will be beneficial to all individuals who have elevated blood pressures above normal levels, especially with advancing age (ACSM, 2006; Frost & Topp, 2006).

The classification of a pre-hypertension stage (Table 1) has been introduced to emphasize the importance of reducing high blood pressure and preventing clinical hypertension through lifestyle modification at an early stage. Early identification of persons at high risk of developing high blood pressure is important so as to prevent morbidity and the adverse effect on quality of life associated with hypertension. It has been established that pre-hypertension is not directly associated with all-cause and cardiovascular disease mortality, but rather the other risk factors associated with a sedentary lifestyle. As soon as pre-hypertension progresses to hypertension, the risk for mortality is increased and is directly associated with hypertension (Mainous *et al*., 2004). The ACSM's guidelines (2006) and the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-VII) (Chobanian *et al*., 1993), recommend that the first line of defence in pre-hypertension and initial diagnosis of hypertension should be lifestyle modification for six to 12 months, before pharmacological treatment is considered. A healthy lifestyle is not just appropriate in diagnosed hypertension, but it is also critical in the prevention of hypertension (Derman, 2008; Hamer, 2006; Touyz *et al*., 2004; Chobanian *et al*., 2003; Wallace, 2003; Tsai *et al*., 2002). It has been reported that physically active persons have a 35% lower risk of developing hypertension than their inactive peers. The risk is also more pronounced in obese persons (Tsai *et al*., 2002).

Lifestyle modifications would include dietary salt restriction and weight reduction through a healthy diet containing lots of fruit, vegetables, low fat dairy products, fibre and reduced fat and cholesterol as well as physical activity and stress management. Unfortunately, non-pharmacological measures are rarely implemented correctly or consistently and the need for pharmacological treatment arises. Aggressive pharmacological treatment in persons with uncontrolled, stage 2 hypertension is warranted, but in persons with mild hypertension pharmacological treatment may not be the optimal treatment. It is well-known that pharmacological treatment reduces BP in persons with established hypertension. However, there are some adverse effects associated with drug treatment. Consequently the health benefits from the pharmacological treatment may not outweigh the risks (Tsai *et al.,* 2004). The economic and biological implications of long term pharmacological treatment justify alternative treatments, especially in mild or borderline hypertension (Painter, 2008; Quinn, 2000).

According to Costa (2002) physicians are aware of the benefits of exercise and diet to lower BP, but it is frequently not taken into account in managing high BP. This is due to a lack of patient education about the advantages of a healthy lifestyle and the disadvantages of medication. Furthermore, the compliance to lifestyle changes requires a lot of effort and patients and physicians choose to take the easier way out (Costa, 2002). Alternatively, because pharmacological treatment is so well-established in decreasing BP, it is routinely prescribed as soon as people are diagnosed with hypertension. It is difficult to persuade patients to rather try lifestyle adjustments to address health problems (Luborsky *et al*., 1982). Anti-hypertensive drugs only reduce the risk for hypertension and cardiovascular disease, but do not address the cause of the hypertension. It will never eliminate the problem completely. On the other hand, lifestyle changes also reduce other risk factors of hypertension and cardiovascular disease and are therefore more beneficial in the long run (Tsai *et al*., 2004; Costa, 2002).

F. EXERCISE AND HYPERTENSION

A sedentary lifestyle is classified as one of the seven risk factors for developing cardiovascular disease (ACSM, 2006). Aging and inactivity are also associated with

a lower resting metabolic rate, gains in fat mass, a decreased aerobic capacity and muscle wasting that contribute to the risk of cardiovascular and metabolic diseases. Muscle wasting leads to reductions in muscle strength and endurance that increases the risk for falls and decrease the ability to perform functional daily activities (Painter, 2008; Volaklis *et al*., 2007; Lehmkuhl *et* al., 2005). It is evident that slow, progressive deterioration due to inactivity is detrimental to human functioning and will go unnoticed in so many until their health is in jeopardy.

It has been known for some time that regular exercise decreases the risk of developing cardiovascular and metabolic diseases. Acute exercise and exercise training has also been prescribed in the treatment and management of hypertension and age-related diseases due to the reduction in body fat mass and improvement of the functioning of the nervous, renal and cardiovascular systems which regulate blood pressure (ACSM, 2006; Frost & Topp, 2006; Pescatello *et al*., 2004; McArdle *et al*., 2001; Brownley *et al*., 1996; Wilcox *et al*., 1982). Research has shown that regular aerobic exercise improves the control of blood pressure by lowering the requirements for anti-hypertensive medication as well as optimizing the treatment as an adjunct to standard pharmacological treatment in patients with uncontrolled hypertension. Furthermore, exercise may reduce other cardiovascular risk factors such as lowered cholesterol, decreased body fat, and decreased fasting blood glucose as well as increased muscle strength (Painter, 2008; Volaklis *et al*., 2007; Lehmkuhl *et* al., 2005; McArdle *et al*., 2001).

It has also been shown that aerobic exercise can improve maximal exercise capacity (VO_{2peak}) by 17% to 23% in patients with chronic disease. A combination of aerobic and resistance training can increase VO_{2peak} by 41% to 48% (Painter, 2008; McArdle *et al*., 2001). Increasing muscle mass by means of resistance training may optimize the response to aerobic conditioning, decrease body fat percentage, increase metabolic rate and thereby increase metabolic control in those at risk of diabetes (Marzolini *et al*., 2008; Umpierre & Stein, 2007; Volaklis *et al*., 2007; Cononie *et al*., 1991).

Evidently, exercise is not only a valuable tool in the control, initial treatment and long term control of BP, but it also has a positive impact on the other risk factors

associated with cardiovascular disease. It is time efficient and cost effective. It is easily administered and has immense variety to accommodate the needs and interests of a very diverse population. Besides the benefits that regular exercise imposes on the physiological systems, it also has potential psychological benefits. These benefits include less anxiety, depression and neuroticism, improvement in mood, self-esteem and self-concept as well as reduced stress (Painter, 2008). Besides the positive health benefits that regular exercise imposes, it will already be worthwhile if it could make one feel more energetic and lively.

CHAPTER TWO

BLOOD PRESSURE RESPONSE TO EXERCISE

A. INTRODUCTION

Blood pressure (BP) is a measure of the force exerted by the blood against the walls of the arteries. Arterial blood pressure reflects the combined effect of arterial blood flow per minute (cardiac output) and the resistance that the vessels offer to blood flow. Systolic blood pressure (SBP) reflects the work of the heart and occurs during ventricular contraction (systole) when the heart forces the blood into the aorta. Diastolic blood pressure (DBP) represents the lowest pressure in the arteries during the ventricular relaxation phase (diastole) when the ventricles fill with blood. This is an indicator of the total peripheral resistance (TPR). The pumping action of the heart causes high pressure in the aorta and the pressure difference in the arteries causes the blood to travel down the aorta to the arteries. Normal blood pressure in healthy adults is as follows: systolic blood pressure below 120 mmHg and diastolic blood pressure below 80 mmHg (Chobanian *et al*., 2003; McArdle *et al*., 2001).

B. BLOOD PRESSURE RESPONSE TO EXERCISE

During exercise the active skeletal muscles demand more oxygen than during rest. Therefore the haemodynamics of the cardiovascular system changes during exercise to meet the increased metabolic demands of the active skeletal muscles. The extent of the changes in haemodynamics is primarily determined by the mode, intensity and duration of exercise and the BP response will vary according to the type of activity.

1. Steady state dynamic exercise

During repetitive, rhythmic and dynamic activity, the demand for oxygen in the active skeletal muscle increases according to the intensity of the exercise. To accommodate this demand the peripheral vasculature dilates to allow more blood to perfuse the active muscle. Cardiac output (CO) also increases dramatically to ensure adequate oxygen to the skeletal muscle. This increase is achieved by the

withdrawal of parasympathetic tone that causes an increase in heart rate (HR) and cardiac contractility. The increase in sympathetic activity causes an increase in the vasoconstrictor activity that leads to a rise in venous return and stroke volume (SV). The rhythmic contraction of the active skeletal muscle acts as a skeletal muscle pump that forces the blood through the veins, back to the heart and therefore increases venous return and stroke volume. In a similar manner the rhythmic contraction of the respiratory muscles during breathing acts as a respiratory muscle pump that also increases venous return and stroke volume. These hemodynamic alterations during exercise are the foundation of the change in systolic and diastolic blood pressure (Mc Ardle *et al*., 2001).

The increase in cardiac output and vasoconstriction of inactive vascular beds results in the progressive increase in SBP at the commencement of exercise while the vasodilation of the vascular beds surrounding the active muscle tissue compensates for the increases and results in the constant or slightly lower DBP. The SBP will level off at 140 to 160 mmHg as the arterioles continue to dilate. The SBP can rise up to 200 mmHg, but is largely determined by the intensity of exercise. The rise in SBP ensures that blood is driven through the vasculature quickly and facilitates the delivery process to the tissue (Wilmore & Costill, 2004). After exercise, BP rapidly returns to, or even decreases below resting values (Pescatello *et al*., 2004; MacDonald, 2002; McArdle *et al*., 2001; Powers & Howley, 2004).

2. Graded dynamic exercise

At the commencement of exercise, there is a rapid rise in SBP and thereafter a gradual, linear increase in SBP as the exercise intensity increases. It is, however, often observed that SBP reaches a plateau at higher intensities of exercise (Sugimoto *et al*., 1998). The DBP will stay constant, but can decrease slightly due to the increased vasodilation of the arteries with an increase in exercise intensity. According to the ACSM's guidelines for exercise testing and prescription (2006) the normal response for BP to exercise is a progressive increase in SBP of 10 ± 2 mmHg per 3.5 $ml.min^{-1}$ increase in oxygen uptake, reaching a plateau at peak exercise. During maximal exercise the SBP may rise to 240 to 250 mmHg in trained athletes, but this rise is mainly due to the increase in cardiac output in individuals with a high

aerobic capacity (Wilmore & Costill, 2004; McArdle *et al*., 2001). In healthy men and women the SBP may vary between 160 – 220 mmHg, with men having slightly higher SBP values than women. The reason for this difference is unclear (Pescatello *et al*., 2004).

3. Resistance exercise

Isometric or straining forms of exercise exhibit a different cardiovascular response compared to dynamic exercise. Resistance exercise results in a pressure overload on the heart whereas aerobic exercise causes a volume overload on the heart (MacDougall *et al*., 1985). Exercise against a high resistance causes a mechanical compression of the arterial vessels that supply blood to the active muscles and it also causes sympathetic vasoconstriction of the inactive vascular beds. Therefore resistance exercise causes a greater rise in both systolic and diastolic BP (McArdle *et al*., 2001).

During exercise with high resistance, the Valsalva manoeuvre is often performed to stabilize the spine and improve performance which greatly increases the intrathoracic pressure (Linsenbardt *et al*., 1992). The Valsalva manoeuvre is defined as a forceful activation of the expiratory muscles that forces air against a closed glottis. This forceful contraction of the expiratory muscles compresses the veins in the thoracic region limiting venous return. The decreased blood flow activates the baroreceptors that decrease the parasympathetic and increase the sympathetic stimulation of the heart. This will result in an increased HR and SV as well as vasoconstriction of the peripheral vasculature resulting in an increase in the cardiac afterload and a decrease in venous return. In order to ensure sufficient blood supply to the muscle, the sympathetic nervous system increases the cardiac output that results in an increased SBP (Wilborn *et al*., 2004; McArdle *et al*., 2001; MacDonald, 2002; Zidermanis, 2000). MacDougall *et al*. (1985) has shown that even without performing resistance exercise and only doing the Valsalva manoeuvre, there is a substantial rise in BP. Linsenbardt *et al*. (1992) has shown that there is no difference in the BP response during exhalation or inhalation during the concentric phase of resistance training, although the Valsalva manoeuvre produced higher absolute BP increases than either inhalation or exhalation during the same exercise.

The magnitude of the hypertensive effect is directly related to the intensity of the exercise and the muscle mass involved. It has been shown that BP is higher during concentric exercise than during eccentric exercise (MacDougall *et al*., 1985). This shows that the magnitude of the BP response is more dependent on the degree of effort than the mechanical force developed (MacDougall *et al*., 1985). Blood pressure readings as high as 320 / 250 mmHg have been documented during heavy resistance training with normal breathing. During heavy resistance training, while performing the Valsalva manoeuvre, values of 480 / 350 mmHg have been recorded in highly trained athletes doing a double leg press (MacDonald, 2002; McArdle *et al*., 2001; Zidermanis, 2000; MacDougall *et al*., 1985).

It is thus important to perform low resistance exercises in persons with high BP, because high resistance exercise, especially if the exercise is accompanied by the Valsalva manoeuvre, will greatly affect persons with an already elevated resting BP. The prescription of exercise in persons with hypertension should be carefully planned and the hemodynamic response of different types of exercise should be taken into account when conducting the exercise. The program should initially be supervised to ensure correct technique during resistance and aerobic exercises and to monitor the exercise intensity that will not result in an adverse rise in BP.

C. FACTORS AFFECTING BP DURING REST AND EXERCISE

1. BP variability

The variability of BP during the day, indicating a circadian rhythm, is well documented (Jones *et al*., 2009; 2008b; Parati *et* al., 1996; Mancia *et al*., 1983, Watson *et al*., 1980). It is believed that average daytime BP can vary between 36 mmHg in normotensive persons to 55 mmHg in hypertensive individuals during free living conditions (Watson *et al*., 1980). When these activities of daily living were controlled, there was no significant circadian rhythm in persons with hypertension (Mancia *et al*., 1983) or in normotensive (Watson *et al*., 1980) subjects during intra-arterial measures of BP between 24 and 72 hours. It was concluded that it is rather the behaviour of humans and environmental factors that result in large fluctuations in the circulation. It is true that waking from sleep, adopting an upright posture, eating

food or drinking coffee and initiating physical activity all occur at the same time as the BP surge (Jones *et al*., 2008a).

Regardless of the uncertainty concerning the circadian rhythm, small short term non-uniform fluctuations during the day do exist (Mancia *et al.,* 1983). These oscillations are also noted during night time, but to a lesser extent than during the daytime hours. Older persons show larger fluctuations in the short term variability of BP than younger persons, but HR variability is less in older persons. This phenomenon could be explained by decreased arterial compliance in older individuals, resulting in greater pressure fluctuations with changes in SV and the decreased sensitivity of the arterial baroreflex that should buffer against the fluctuations in BP (Mancia *et al.,* 1983; Watson *et al*., 1980). It has been reported that SBP variability does not change at different times of the day, but that the level of physical activity and the level of mean BP do influence the absolute BP variability (Watson *et al*., 1980). Mancia *et al*. (1983) reported that although hypertensive persons show a greater absolute BP variability, there is no difference in the relative BP variability between hypertensive and normotensive persons, indicating that the level of BP does influence the variability. Watson *et al*. (1980) and Mancia *et al*. (1983) reported that the only true systematic variability in BP and HR can be seen during sleeping hours, namely a nocturnal fall or diurnal variation, but that the variability does not differ between different times of the day. Additional to the decreased pressure during sleeping hours, other researchers have reported a morning surge of increased BP just after waking (Jones *et al*., 2009; 2008a; 2008b). This could be the result of altered sympathetic nervous system activity or hemodynamic adjustments with rest and rising (Jones *et al*., 2008a; 2008b; Mancia *et al.,* 1983). It has been reported that the incidence of cardiovascular events are higher in the early morning hours than during night time hours. It has been speculated that the morning surge in BP could be a possible cause of these acute cardiac events (Jones *et al*., 2008b). It is believed that the variation in BP during the day, on top of the already elevated BP can lead to rupture of the arterial plaque, leading to an acute cardiac event. Therefore there are concerns regarding the safety of early morning exercise in persons at risk for cardiovascular events, especially in persons with hypertension (Jones *et al*., 2009). However, no pattern of exercise related cardiac events have been reported in hypertensive persons who exercise in the morning.

These circadian variations and short term oscillations in BP should be considered when analysing 24 hour BP after a bout of exercise, because changes in BP could be wrongfully attributed to the effect of the exercise instead of the normal variation in BP. Although limited evidence exists on the morning surge, and the effect it has on BP during exercise in the morning, the time of day to implement exercise should be taken into consideration to minimize the risk of cardiovascular events and prevent unnecessary complications during exercise. The time of day might not only affect the BP response during exercise, but also after exercise, either attenuating or amplifying the decrease in BP after exercise.

2. Body position

Changing body position from supine or seated to standing, at rest or during exercise, imposes an orthostatic stress on the body which results in a decreased venous return and therefore cardiac output. Reflexes prevent a decrease in arterial BP which in turn prevents syncope. Orthostatic stress can reduce arterial BP to a level in the brain that is insufficient to provide adequate perfusion, leading to syncope (Hainsworth *et al*., 2000). The total peripheral resistance is essential in maintaining normal BP during change in body position. An increase in HR also prevents large decreases in CO. Wilborn *et al*. (2004) showed that during resistance training, there was no statistically significant difference in the SBP or HR responses between exercise in a semi-recumbent position (angle leg press) and when standing upright (hack squat) at 65%, 85% or at 100% of 1RM.

It is believed that changing body position during recovery alters the central circulation haemodynamics and will affect the magnitude of post exercise hypotension (PEH) (Pescatello & Kulikowich, 2001). Thus, the changes in body position should be controlled during the recovery period of exercise studies. It is likely that the mechanisms that are responsible for the change in the central haemodynamics also contribute to the mechanisms responsible for PEH. The BP response will thus be affected differently in a seated, standing or supine recovery.

3. Active muscle mass

During exercise of the upper body, BP rises more than with leg exercise at a given percentage of VO_{2max}. The reason for the higher BP response during arm exercise is because the smaller muscle mass and vasculature involved causes greater resistance to blood flow than larger muscles and greater vasculature. This causes a greater cardiovascular strain because of the increased workload on the myocardium. This has implications for persons with cardiovascular disease or disease risk factors like hypertension. It is therefore recommended that this population group engage in dynamic types of exercise that involve large muscle groups. If upper body exercise is being used to train persons with coronary heart disease, the proper exercise levels should be recommended (McArdle *et al*., 2001).

4. Other

In a study to determine the effects of age, gender and physical training on the blood pressure response of stage 1 and 2 hypertensive participants during an exercise stress test, Sugimoto *et al*. (1998) showed that older persons and women had a greater SBP response at a similar work load of exercise. This might be the result of the smaller muscle mass in women leading to a smaller exercise capacity. The reason for the higher SBP in older persons might be attributed to less compliant arteries causing a greater pressor response and an increased activity of the sympathetic nervous system. This could be due to impaired baroreceptor function as well as decreased muscle strength that leads to a somatic reflex resulting in sympatho-excitation.

Older persons with higher BP should therefore not exercise at such a high intensity as younger adults to prevent an amplified BP response. The reasons being the deterioration in vasculature and associated changes in physiological functioning that occurs with ageing.

Other factors that could affect the BP following exercise are lifestyle related. Habits like smoking and alcohol consumption would also affect the BP after exercise. Medication use, beside anti-hypertensive medication, might also affect BP at rest.

5. Conclusion

From the available literature it can be seen that various factors affect the BP response during exercise as well as during the 24 hour period after exercise. Each individual has a unique genetic composition, body composition, different personal circumstances and a distinctive medical history that might affect the response of that person to exercise or other BP treatment. Due to these uncontrollable, delimitating factors, it is almost impossible to predict how each individual will react to BP treatment. Thus, when prescribing exercise in hypertensive individuals or persons with other chronic diseases, the exercise prescription should take contra-indications into account. The program should be well-controlled and administered correctly to ensure a desirable outcome.

D. ADAPTATIONS TO EXERCISE TRAINING

Exercise training is associated with changes in the human body which allow the individual to increase his or her exercise capacity, therefore exercising at a higher workload, but at the same physiological cost as an untrained person. The training induced adaptations also occur in diseased patients when the exercise stimulus is sufficient. These physiological adaptations are reversible and will be lost with detraining (McArdle *et al*., 2001).

1. Cardiovascular adaptations

The main adaptations during exercise training allow the body to deliver oxygen more effectively to the active skeletal muscle and for the muscle to utilize the delivered oxygen more efficiently. Thus the body functions more economically. There are numerous adaptations in the cardiovascular system that allow for the increase in efficiency (McArdle *et al*., 2001).

1.1 Plasma volume

Exercise training causes a significant increase in plasma volume after only three to six exercise sessions. This enhances the end diastolic volume. According to the

Frank-Starling mechanism, this will increase SV as well as increase oxygen transport and improve temperature regulation. With detraining, the plasma volume can decrease to normal levels within one week (McArdle *et al*., 2001).

1.2 Heart rate

Exercise training alters the sympathetic and parasympathetic activity that controls the rate of contraction of the heart. The firing rate of the sinoatrial (SA) node is slowed and will therefore result in a lower HR at rest and during sub-maximal exercise. This is further decreased by an increased stroke volume, because CO = HR x SV..... (2.1) While maintaining the same CO a lower HR will be achieved due to the improved SV. A trained person will be able to maintain a higher workload and reach higher oxygen consumption before reaching a specific submaximal HR than an untrained person (McArdle *et al*., 2001). The HR recovery after a period of exercise training is also enhanced, leading to a faster recovery after exercise (Wilmore & Costill, 2004).

1.3 Stroke volume

Training results in a higher SV at rest and during exercise. The possible mechanisms contributing to a higher SV are an increased left ventricular volume, due to plasma volume expansion, reduced cardiac stiffness, an increased diastolic filling time due to the slower HR as well as an improved cardiac contractility. All these adaptations contribute to a greater cardiac output and therefore a greater oxygen delivery to the muscle (McArdle *et al*., 2001).

1.4 Cardiac output

The greatest improvement in aerobic functioning after exercise training is due to the increase in cardiac output. The main contributor to the rise in maximal CO is the increase in SV, because maximal HR is not significantly affected by training. During submaximal exercise, a lower CO is required to maintain the same absolute workload after training compared to before exercise training. This can be explained by an increased oxygen extraction by the muscles, a more effective redistribution of blood during exercise and the enhanced ability of the muscle to generate ATP at a lower

oxygen concentration in the muscle, due to increased number of mitochondria (McArdle *et al*., 2001).

1.5 Blood flow

Trained individuals are able to maintain submaximal exercise with a lower CO than untrained individuals due to a more efficient redistribution of blood to the active skeletal muscle. Although the sympathetic nervous system activity in the splanchnic and renal areas is reduced due to training, there is still more effective redistribution of blood to the active skeletal muscle. This also allows for better thermoregulation because of the more abundant supply of blood that can precipitate heat from the skin due to the increase in cutaneous blood flow. Another contributing factor is the enlarged cross-sectional area of arteries surrounding the muscle due to the increased capillarization with exercise training. The increased ability of the muscle to extract oxygen from the blood vessel and the improved blood supply to the muscle will allow a greater quantity of oxygen to be supplied to the muscle (McArdle *et al*., 2001).

Exercise training also results in functional and structural changes in the myocardium. The cross sectional area of the coronary arteries surrounding the heart increases, allowing better perfusion to the heart. Exercise increases the ability of the heart to tolerate episodes of ischemia, making it more resistant to ischemic episodes such as myocardial infarction and to recover faster after such events (McArdle *et al*., 2001).

2. Metabolic adaptations

At the level of the tissue there are numerous adaptations that improve the metabolic functioning of the skeletal muscle. The size and number of mitochondria increases thereby improving the ability to generate ATP aerobically. The increased enzyme activity allows the person to sustain a high percentage of aerobic capacity during exercise without the accumulation of blood lactate. Thus a person can work at a higher workload before getting tired. The ability to mobilize, deliver and utilize fatty acids as energy substrate is also improved with endurance training. The enhanced fat utilization will allow the person to train at a higher intensity, for a longer duration

while conserving muscle and liver glycogen. This metabolic adaptation will greatly decrease the risk for obesity and diabetes (McArdle *et al*., 2001).

3. Musculoskeletal adaptations

In addition to the cardiovascular adaptation, exercise training will also contribute to improved muscle fibre functioning by increasing their existing potential. With advancing age the muscle fibre type will change, converting more type II fibres to type I fibres. With the correct training this process can be slowed (Stewart *et al*., 2005; Brooks *et al*., 2000). Hypertrophy of muscle fibres can occur, leading to improved muscle strength and endurance. Especially in older individuals, this will improve their ability to perform daily activities and ensure that independence is maintained for longer (Cononie *et al*., 1991). It is speculated that endurance training might increase the muscle fibre's ability to utilize oxygen by increasing myoglobin concentration in the muscle. This process in humans is, however, still unclear (McArdle *et al*., 2001).

The change in the structural and functional physiology due to exercise training results in more efficient functioning of the human body. All the changes associated with exercise will affect the mechanisms responsible for high BP. Exercise will not necessarily ensure an extended life, but it will ensure an independent lifestyle and enhanced quality of life by increasing cardiovascular functioning and maintaining muscular integrity.

4. Altered haemodynamics during exercise in persons with hypertension

The haemodynamics of hypertensive patients differ from normotensive individuals and will vary according to the age of the person and the stage of hypertension. Ageing alone, irrespective of BP, causes significant changes in the haemodynamics at rest and during exercise. Boutcher & Stocker, (1999) reported that during light isometric tasks and dynamic exercise, older subjects, compared to younger subjects, displayed significantly higher SBP, HR and rate pressure product (RPP) throughout the dynamic exercise and the recovery period as well as higher DBP during isometric exercise even though the younger subjects exercised at a higher intensity and

workload. Substantial differences in the resting haemodynamics of the younger and older subjects have been reported as well (Boutcher & Stocker, 1999). This indicates that myocardial work at rest and during exercise is substantially (15%) higher in older individuals compared to younger persons. The higher BP at rest and during exercise could be the result of less compliant vasculature causing a greater pressor response and altered sympathetic activity due to impaired baroreceptor functioning as well as decreased receptor sensitivity (Sugimoto *et al*., 1998).

In a study to investigate the effect of six months of exercise training on the haemodynamics of healthy, older individuals, Cononie *et al*. (1991) reported that the hemodynamic response at rest was altered following both resistance and endurance training. The resistance training group showed elevated HR and reduced SV, while the endurance training group showed a reduced HR, but a slight increase in SV at rest. Although the endurance training group had a lower resting BP, there were no significant changes in cardiac output or TPR at rest in either of the groups. Exercise prescriptions for older persons should take the change in resting haemodynamics into account, especially with higher intensity exercise, to prevent an excessive myocardial load.

Irrespective of age, one of the consequences of hypertension on the cardiovascular system is that it affects the haemodynamics and therefore the functional or exercise capacity in general. One of the main differences in the haemodynamics of hypertensive and normotensive subjects at rest is the increased TPR, resulting in an increased resistance to blood flow and thus an increased afterload (Brooks *et al*., 2000). Exposure to such a high resistance for a long period of time can lead to hypertrophy of the heart and even heart failure in severe cases. The hypertrophy that results from hypertension is not the same seen during exercise training. Adaptations to long term exercise cause an enlargement of the myocardium and therefore contribute to the heart's function by increasing CO and myocardial contractility. The hypertrophy due to hypertension causes an increase in the size and number of connective tissue cells also resulting in an increased cardiac contractility, but with little or no increase in CO, due to a lower SV (Brooks *et al*., 2000; Cleroux *et al*., 1992; Montain *et al*., 1988). This places an increased load on the heart due to the enlarged size and the increased metabolic demand without an

increase in the heart's functional capacity. A chronically overloaded heart will lead to disintegration of the myocardium and ultimately myocardial ischemia (Brooks *et al*., 2000). In the later stages of hypertension the weakened myocardium will result in a reduced contractility, leading to a smaller ejection fraction and SV which will ultimately affect the maximal oxygen consumption during exercise (Brooks *et al*., 2000). The only way to avoid heart failure in persons with hypertension is to prevent chronic overload of the heart that will progress to heart failure, by reversing or controlling hypertension.

Hypertensive persons respond in a similar manner to exercise training as their normotensive peers (McArdle *et al*., 2001) and can therefore prevent or slow the destructive effect of untreated, uncontrolled hypertension. It is essential to achieve stable BP via lifestyle adaptations and, if necessary, pharmacological treatment and exercise to prevent permanent damage to the heart.

Research on the acute cardiovascular response to exercise in hypertensive persons shows the importance of the understanding of the pathophysiology of the disease when prescribing acute and chronic exercise. Montain *et al*. (1988) showed that the hemodynamic response during dynamic exercise, at 50, 60 and 70% VO_{2max}, was altered in hypertensives compared to normotensive subjects. During exercise CO was lower due to a reduced SV, but BP was higher in the hypertensive group compared to normotensive participants. In both groups the TPR decreased during exercise showing that hypertensive patients have the same relative capacity to allow vasodilation of the local vasculature. The normotensive group had lower TPR at rest compared to the hypertensive group. Cleroux *et al*., (1992) showed similar results when comparing the exercise response of hypertensive and normotensive participants. Following dynamic exercise for 30 minutes at 50% peak aerobic capacity, hypertensive subjects displayed higher SBP and DBP during exercise, although the absolute change in SBP above resting values were the same for the hypertensive and normotensive groups.

Sugimoto *et al*. (1998) also investigated the SBP response to acute exercise by using a logistic function curve analysis in hypertensive subjects. They investigated the effect of exercise training on the BP response during exercise. Although the training

resulted in decreased resting SBP, there was no change in the SBP response during exercise, although the exercise capacity did increase. This study only implemented exercise training for three weeks which might have been insufficient to induce cardiovascular changes. They also did not have a normotensive control group to compare the changes in normotensive and hypertensive individuals.

Although persons with hypertension have altered haemodynamics at rest and during exercise, they show the same response to exercise as their normotensive peers. The physiological changes and adaptations during acute exercise and exercise training are still the same and would be as beneficial as in normotensive subjects. If there is no history or underlying cause for cardiac incidents, no adverse effects should be experienced during exercise in hypertensive persons. Persons with uncontrolled BP (> 160 / 110 mmHg) should first obtain stable BP via drug treatment before commencing an exercise program.

4.1 Effect of hypertension medication on exercise capacity in persons with hypertension

In persons where anti-hypertensive medication is warranted, it is important to carefully select the type of medication that will least affect the exercise capacity of the person. The medication should decrease resting and exercise BP as well as reduce TPR. Of the many anti-hypertensive drugs on the market, angiotensin converting enzyme (ACE) inhibitors and calcium channel blockers are prescribed most frequently in persons with hypertension who exercise regularly (Pescatello *et al*., 2004). Hypertension can also be treated with diuretics and / or beta-blockers. In the case of complicated hypertension, a combination of angiotensin converting enzyme inhibitors, angiotensin II receptor blockers, alpha-blockers, beta-blockers, calcium antagonists and diuretics can be prescribed to control hypertension (Chiong, 2008).

Some anti-hypertensive agents might affect the physiological responses during exercise in hypertensive patients. This may lead to an unpleasant perception of exercise, because the patient might feel tired and fatigued, ultimately leading to non-compliance to either the medication or the exercise. The prescription of medication

should be specific for each patient and should consider the aetiology of hypertension and how the specific agent will interact with the individual as well as how the medication will influence the exercise response and tolerance (Derman, 2008).

Derman (2008) listed the effects of certain agents on the exercise response. Beta-blockers for instance decrease the HR response, RPP and CO during exercise, thus restricting exercise capacity (Sorace *et al*., 2009). The HR training zones prescribed during exercise should therefore be adjusted. It also alters fuel utilization, skeletal muscle recruitment patterns, thermoregulation and the rate of perceived exertion (RPE) during exercise. This can lead to feelings of fatigue, hyperthermia and hypoglycaemia during exercise. Thus, preventing patients from participating in long duration exercise, especially in the heat. This agent is not prescribed regularly for athletes participating in competitive sport. Nitrates cause a similar response as beta-blockers, but might increase exercise tolerance in patients with myocardial ischemia or congestive heart failure. Diuretics, on the other hand, do not alter the hemodynamic response or exercise tolerance, but it does decrease exercise BP and cause premature ventricular contractions (PVC's) due to hypokalaemia.

One of the agents most regularly prescribed for hypertensive patients participating in exercise is calcium channel blockers. This agent can lower the BP response during exercise and usually increases exercise tolerance in persons with myocardial ischemia. A longer cool down period should be incorporated in the program, because these agents can cause hypotensive episodes on rapid cessation of exercise. A similar response is seen in angiotensin-converting enzyme (ACE inhibitors) and angiotensin II receptor blockers, making these good agents to prescribe for hypertensive athletes (Derman, 2008).

From the available literature it is clear that not just any drug can be prescribed for persons with hypertension. Especially in physically active patients, the side effects of the medication and the adverse effects during exercise should be discussed with the patient to ensure awareness of the signs and symptoms so as to avoid unwanted responses. The effect of exercise and medication should be monitored to ensure that the dose of the medication is adjusted when needed.

E. EXAGGERATED BLOOD PRESSURE RESPONSE TO EXERCISE

The cardiovascular response to exercise has been shown to be an important predictor for the future development of hypertension. Thus, individuals with normal resting BP, but an exaggerated BP response during or after exercise, have a greater risk of developing hypertension (Pescatello *et al*., 2004). The exaggerated BP response can also contribute to ischemic cardiac events, including myocardial infarction and cerebrovascular incidents (Pescatello *et al*., 2004).

There is no clear definition of what constitutes an exaggerated blood pressure response, but it is mostly defined on the basis of a designated exercise SBP at maximal exertion or at a given work load. An exaggerated response should take the resting SBP, gender and age of the person into account (Miyai *et al*., 2000). According to the ACSM's guidelines for exercise testing and prescription (2006), an exercise test or session should be terminated when SBP reaches 250 mmHg and DBP reaches 115 mmHg or when SBP decreases by more than 10 mmHg with an increase in workload. However, when signs and symptoms of ischemia occur before these endpoints, exercise should also be terminated.

In a study of middle-aged normotensive men, it was reported that those individuals with a disproportionate BP response during exercise had a three times higher risk of developing hypertension during a 4.7 year follow-up period (Miyai *et al*., 2002). Similarly, Miyai *et al*. (2000) reported a positive correlation between an exaggerated BP response and the development of future hypertension in subjects (42 ± 6 years) with high-normal resting BP. The subjects were followed up for an average duration of 5.1 years. Although an exercise test alone cannot be used to predict future hypertension, it can serve as an adjunct if BP data is available (Miyai *et al*., 2002). Other predictors of future hypertension include resting BP, family history of hypertension or cardiovascular disease, a high body mass index (BMI), physical inactivity and weight gain of more than 5kg (Pescatello *et al*., 2004; Miyai *et al*., 2000).

There is also controversy concerning the mechanism of an exaggerated BP response during exercise. It is believed that persons with an exaggerated response do not

have sufficient vasodilation in the arteries to decrease TPR to compensate for the rise in CO during exercise. These responses could be attributed to hyper-reactivity of sympathetic nerves, increased vascular responsiveness to adrenergic stimulation or the thickening of the arteriolar wall that alters its ability to respond to vasoconstrictor stimuli. The rise in CO and the reduced vasodilation causes an increase in systolic and diastolic BP similar to patients with established hypertension (Miyai *et al*., 2002; 2000).

Currently the use of routine exercise testing to predict future hypertension from an exaggerated exercise BP response is not advised due to a number of limitations in the research. For instance, there is no standardized exercise test protocol and the definition of an exaggerated BP response is lacking. In previous studies confounding variables were not always controlled for and the use of non-invasive BP measures during exercise have limitations. However, when exercise testing is done and the BP data is available, it can be used as additional screening information to pro-actively prevent high risk patients from developing hypertension (Pescatello *et al*., 2004).

F. EXERCISE IN PERSONS WITH CARDIOVASCULAR DISEASE RISK FACTORS

In certain cases the benefits of acute or chronic exercise do not outweigh the risks associated with it. Before initiating exercise in high risk individuals, the relative risk screening should be conducted and medical treatment should be initiated to stabilize the major risk factors before exercise is considered. The American College of Sports Medicine (2006) listed the following contra-indications to exercise testing: a recent myocardial event or myocardial infarction, unstable angina, uncontrolled dysrhythmias, severe aortic stenosis, uncontrolled heart failure, an acute pulmonary embolus or pulmonary infarction, acute myocarditis or pericarditis, a suspected dissecting aneurysm and an acute systemic infection, fever or swollen lymph glands.

Even if individuals do not present with the above signs or symptoms, each situation should be carefully evaluated and the risks of exercise should never outweigh the benefits. In persons with risk factors for cardiovascular disease and with compromised coronary circulation, the increased myocardial oxygen demand and

shortening of diastole during strenuous activity may evoke an oxygen deficiency and can cause arrhythmia. Strenuous physical activity can also trigger an acute myocardial infarction or sudden cardiac death, especially in individuals with known heart disease and sedentary lifestyles (Pescatello *et al*., 2004). Persons with a resting BP of 160 / 100 mmHg should be advised not to initiate exercise until BP is controlled and stabilized by pharmacological treatment (American college of Sports Medicine, 2006). The situation of each individual should be carefully evaluated and discussed with a cardiologist before an exercise program is initiated. Therefore a careful medical screening, including an exercise stress test, prior to starting an exercise program is essential.

This does not mean that persons with cardiovascular disease risk factors cannot exercise at all. Numerous studies have been conducted in persons with cardiovascular disease or cardiovascular disease risk factors, without any adverse events. Exercise training is well accepted as a non-pharmacological treatment in persons with stable coronary artery disease. Exercise training also leads to an improved endothelial-dependent vasodilation and vascular function resulting in an increased functional capacity (Walsh *et al*., 2003). A normal endothelium is important in the regulation of vascular tone, thrombogenesis, lipid breakdown, inflammation and vessel growth. Therefore a dysfunctional endothelium is believed to play a key role in the development and progressions of atherosclerosis. A chronically dysfunctional endothelium may lead to the disruption of atherosclerotic plaque and cause potential vascular events. A dysfunctional endothelium is also associated with essential hypertension, because of an altered production of nitric oxide and endothelin-1. A decrease in the nitric oxide release can cause an increase in vascular resistance and ultimately lead to vascular and cardiac hypertrophy, stroke and coronary artery disease (Perticone *et al*., 2001). Thus by improving endothelial functioning, the risk for cardiac events decreases.

CHAPTER THREE

HYPERTENSION AND EXERCISE

A. INTRODUCTION

Exercise and lifestyle adjustments are, without additional drug treatment, the only prescriptions for persons with borderline hypertension. Lifestyle modifications are also prescribes, along with pharmacological treatment, for persons with stage 1 and 2 hypertension (Chobanian *et al*., 2003). The importance of exercise treatment in the prevention and control of hypertension cannot be denied. It is important to understand the mechanisms of exercise training that are responsible for the decrease in resting BP so as to ensure that lifestyle and exercise habits are put into practice successfully. The successful implementation of an exercise training programme will lead to decreased BP, consequently avoiding the necessity for initiating drug treatment or enhance antihypertensive drug efficacy (Chobanian *et al*., 2003).

B. THE MANAGEMENT AND CONTROL OF HYPERTENSION

1. Exercise

There is abundant evidence in the available literature that regular exercise decreases resting BP in most hypertensive subjects (Tsai *et al*., 2004; Pescatello *et al*., 2004; Tsai *et al*., 2002; Hagberg *et al*., 2000; Sugimoto *et al*., 1998; Brownley *et al*., 1996). It is well established that an acute exercise session, exercise training and even just physical activity through lifestyle can reduce high BP with up to 10 mmHg for SBP and eight mmHg for DBP (Tsai *et al*., 2004; Hagberg *et al*., 2000; Wilcox *et al*. 1982). Costa, (2002) also showed that persons participating in regular physical activity can have an up to five mmHg lower resting BP than their inactive peers.

Exercise treatment alone is not sufficient for all hypertensive adults. It has been reported that only about 75% of all hypertensive patients will respond to exercise

treatment and that BP reductions will vary between patients depending on the pathophysiology of the condition and / or the genetic differences between individuals (Brown *et al*., 2002; Fagard, 2001). Another reason why exercise alone does not always have BP lowering effects is that there is no certainty regarding the optimal amount of exercise to induce significant BP changes. Numerous studies have examined different exercise characteristics as well as combinations of exercise. These methodological inconsistencies may be the reason why not all exercise interventions show positive effects on BP.

2. Exercise and weight loss

Hypertension is usually associated with a number of other cardiovascular risk factors i.e. overweight, obesity, inactivity and diabetes. With the rising prevalence of obesity worldwide, the risk for hypertension in this population increases because it has been shown that obesity, among others, is a contributing factor for the development of hypertension (Chobanian *et al*., 2003). It has been reported that obese women with a BMI > 30 $kg.m^{-2}$ have a 16% higher prevalence of hypertension than women with a normal BMI (Costa, 2002). Similarly it has been reported that a one kg reduction in body weight can result in a 1.6 mmHg decrease in SBP and a 1.3 mmHg reduction in DBP. Therefore a four to eight percent decrease in body weight will decrease BP by 3 mmHg (Touyz *et al*., 2004; Costa, 2002). Chobanian *et al*. (2003) also reported that a 4.5 kg decrease in body weight will reduce or prevent high BP in overweight persons.

Fortmann *et al*. (1988) compared the effects of a one year weight loss and exercise intervention on BP and showed that both interventions significantly reduced ambulatory BP. Although the exercise group did not show a significantly greater reduction in BP than the diet group, the diastolic evening BP reductions for the exercise only group was significantly greater. The magnitude of the decrease in BP was small, but the health implications of such changes are potentially large considering that the subjects were normotensive. In a hypertensive population, the effects are likely to be greater.

Blumenthal *et al*. (2000) compared the effect of a combined weight loss and exercise program to an exercise only program in hypertensive, sedentary, obese individuals. The intervention lasted six months and the subjects in the two intervention groups performed the same amount of exercise. The exercise program consisted of aerobic exercise (cycling or walking / jogging), 3 to 4 times a week at 70% – 85% heart rate reserve (HRR). After six months both intervention groups had higher aerobic capacities, although the weight management group lost significantly more weight than the exercise only and control groups. Both intervention groups showed a similar and significant reduction in resting systolic and diastolic BP compared to the control group. The weight management group showed a significantly greater reduction in DBP than the exercise only group. Although exercise alone is sufficient in lowering BP, the weight management group reported a larger reduction in both resting and ambulatory BP which supports the contention that a program of weight loss significantly augments the results of an aerobic exercise program.

Fagard, (2005) concluded in a meta-analysis that weight reduction by diet alone is more effective in lowering BP than exercise alone. When exercise was combined with dietary weight reductions, there was a greater decrease in BMI, but no additional BP reductions. However, weight reduction is easier said than done. It has been shown that most women will regain the weight they have lost through diet alone (Costa, 2002). It may therefore be easier to maintain or reduce weight through a healthy diet and regular exercise (Touyz *et al.,* 2004) and in the process also improve the functioning of the nervous, renal and cardiovascular systems, which play an important role in the regulation of BP (Derman, 2008; ACSM, 2006; Frost & Topp, 2006; Chobanian, 2003; Wallace, 2003).

3. Exercise and medication

Some researchers believe that exercise in combination with diet has similar blood pressure lowering effects as pharmacological therapy in persons with borderline or stage 1 hypertension (Chiong, 2008; Frost & Topp, 2006; Pescatello *et al*., 2004; Fagard, 2001; Brooks *et al*., 2000). Radaelli *et al*. (1992) compared the effects of an exercise program, atenolol (β-blocker) treatment and a combination of exercise and

drug treatment on the magnitude of BP reductions in twelve hypertensive men and women aged 33 to 58 years. Despite an increase in cardiovascular fitness, exercise training alone had minimal effects on SBP, DBP and HR. Patients in the medication group showed no improvements in cardiovascular capacity, but significant decreases in resting BP and HR. In the combination group (exercise and medication), there was a significant decrease in resting BP and HR, but it was not significantly greater than the medication treatment alone. It was deduced that physical training on its own has a modest effect on persons with hypertension, but that the training effect on BP can be enhanced with antihypertensive drugs (Radaelli *et al*., 1992). This study only included 12 participants and the exercise treatment was only applied over three weeks. Although acute reductions in BP can be seen after a single session of exercise, significant long term adaptations will hardly occur after only three weeks of training. It has been reported that BP reductions are evident within 10 weeks of starting a training program, but that the lowering effects only become clear after 11 to 20 weeks of training (Hagberg *et al*., 2000).

Despite evidence that exercise training alone is not enough to prevent or effectively treat hypertension, exercise must still be considered an important adjunct to anti-hypertensive treatment. Exercise is cost-effective, it does not have pharmacological side effects and it also imposes a positive influence on other cardiovascular risk factors such as plasma lipoprotein levels, glucose and insulin metabolism and left ventricular hypertrophy. Overall, regular exercise training decreases cardiovascular disease mortality (Tsai *et al*., 2004; Hagberg *et al*., 2000).

Furthermore, exercise attenuates physical deconditioning and the development of secondary cardiovascular risk factors (Meredith-Jones *et al*., 2009). Exercise improves overall wellbeing, physical functioning and hence optimizes quality of life. In persons with chronic diseases, the natural course of functioning is deterioration, but exercise can decrease the rate of deterioration or maintain the current functioning. If slight improvements or maintenance are seen in such cases, it is considered a positive result (Painter, 2008; Chiong, 2008; Volaklis *et al*., 2007; Pescatello *et al*., 2004).

The question arises how exercise facilitates all these beneficial effects on health and well being. Especially whether the change in BP after an endurance training program is the result of the accumulated effect of the acute exercise sessions that cause a sustained BP reduction (i.e. the effect of exercise *per se*) or whether it is the result of a different mechanism than that of the acute reduction. Another possibility is that it is related to the effects that exercise has on other risk factors such as glucose concentration, lipid profile, obesity and other morphological factors (Hamer, 2006).

C. PHYSIOLOGICAL MECHANISMS UNDERLYING THE CHANGE IN BLOOD PRESSURE WITH EXERCISE TRAINING

The precise mechanism of how regular exercise lowers resting BP is uncertain mainly because the aetiology of hypertension could be the result of many factors (Brown *et al*., 2002; Kiyonaga *et al*., 1985). Furthermore, genetic differences between individuals may contribute to the mechanism and magnitude of BP reductions (Hamer 2006; Pescatello *et al*., 2004; McArdle *et al*., 2001), and may explain why all people do not respond equally to an exercise intervention.

Pescatello *et al*. (2004) stated that the decreased resting BP after exercise training must be mediated by either CO or TPR. Resting CO does not really change after chronic exercise, even in healthy individuals, although resting HR might decrease. The increase in SV, typically found after training, could be due to an increased venous return to the heart, an increase in the size of the left ventricle, an increase in cardiac contractility or a decreased afterload. The increase in SV is therefore counterbalanced by the decrease in resting HR and will not result in major changes in the CO. It seems then that the decrease in BP is therefore rather the result of changes in the peripheral vascular resistance than alterations in CO (Cornelissen & Fagard, 2005; Fagard, 2001). The change in peripheral vascular resistance is believed to be mediated by changes in the sympathetic nervous system and / or structural adaptations at the level of the tissue (Hamer, 2006).

1. Sympathetic nervous system

One of the main characteristics in persons with hypertension is the increased sympathetic nervous system activity (SNA) (Pescatello *et al*., 2004). Altering the latter will result in changes in BP. The peripheral vasculature is directly affected by the change in sympathetic nervous system activity, resulting in altered vasoconstriction of vessels. Changes in the vasodilatory substances produced as a result of alterations in the SNA as well as the vascular responsiveness to these substances after exercise has been documented to affect BP (Pescatello *et al*., 2004; Brown *et al*., 2002). However, the specific effect of exercise on these changes remains controversial due to limitations in the non-invasive measurement of sympathetic nervous system activity (Hamer, 2006).

It has been reported that the concentration of vasoconstrictor substances such as plasma norepinephrine and endothelin-1 are reduced after exercise training (Hamer, 2006; Pescatello *et al*., 2004; Brown *et al*., 2002). Kiyonaga *et al*. (1985) also reported a decrease in plasma catecholamine levels after 20 weeks of exercise training in primary hypertensive patients. Although no significant relationship could be established between decreased plasma catecholamines and BP reductions, it is still indicative of reduced SNA. The lack of significant results might have been due to the small sample size. It has also been shown that exercise decreases the vascular responsiveness to vasoconstrictors like norepinephrine and endothelin-1, resulting in less TPR (Pescatello *et al*., 2004; Brown *et al*., 2002), but the evidence in humans is limited (Hamer, 2006). An increased release of local vasodilatory substances, like nitric oxide, has also been noted (Hamer, 2006; Pescatello *et al*., 2004). Exercise training therefore attenuates vasoconstriction by reducing the activity of the SNA (Pescatello *et al*., 2004).

Another possibility in mediating a decreased BP that has been observed after exercise training is increased insulin sensitivity. Impaired insulin sensitivity and insulin resistance is regularly associated with hypertension. It affects BP through altered renal sodium dynamics and increased SNA. Increased insulin sensitivity after exercise training has been reported (Painter, 2008; Pescatello *et al*., 2004; Brown *et al*., 2002). This might also indicate a reduction in the SNA. In a six months exercise

study, Brown *et al*. (2002) also showed increased insulin sensitivity after six months of exercise training, but the decrease in MAP could not be directly attributed to a change in the insulin sensitivity in older hypertensive individuals. The researchers concluded that the insulin sensitivity might indirectly influence BP by altering SNA.

2. Structural adaptations

As a result of exercise training, several changes occur at the level of the tissue, such as changes in the vasculature structure of the muscle tissue. These changes include increased length, cross-sectional area and diameter of already existing arteries as well as angiogenesis (formation of new blood vessels) that increases the capillary density. The increased capillary density, greater distensibility of the vessels and a larger lumen diameter are all structural adaptations that possibly contribute to the lower TPR (Pescatello *et al*., 2004; McArdle *et al*., 2001; Brooks *et al*., 2000). Besides the changes in the vasculature surrounding the muscle, changes also occur in the muscle fibre itself. Painter (2008) reported changes in the muscle structure and neuromuscular control after six months of combined aerobic and resistance training. There were improvements in the cross-sectional area of type I and II muscle fibres and an improved mitochondrial density. Although the muscle fibre type did not change, the potential of each muscle fibre increases.

3. Other

It has been speculated that a reduction in the resting levels of plasma renin and angiotensin II, a powerful vasoconstrictor, might contribute to decreased BP after exercise training, but currently the evidence is not conclusive (Brown *et al*., 2002). Cononie *et al*. (1991) reported no change in plasma angiotensin I and II after six months of training in 70 – 79 year old normotensive men and women. Kiyonaga *et al*. (1985) studied the hormonal response to exercise in middle aged, hypertensive patients. They also reported no change in the renin-angiotensin system or ACE activity after 10 to 20 weeks of aerobic exercise training. This suggests the decreased renin and angiotensin concentrations are not major contributors to decreased BP after exercise training in normotensive and hypertensive individuals. Altered renal functioning that facilitates the kidney's elimination of sodium might also

be affected by exercise, resulting in a reduced fluid volume and hence BP (Kiyonaga *et al*., 1985). Again no conclusive results are available (Hamer 2006; Pescatello *et al*., 2004; McArdle *et al*., 2001).

Exercise training is regularly associated with decreased body fat percentage and increased aerobic capacity which could contribute to a reduction in BP. It has been shown that obesity and insulin resistance are associated with increased sympathetic nervous system activity and renal sodium reabsorption that contributes to an elevated BP (Brown *et al*., 2002). It can be deduced that by decreasing body mass, body fat and increasing insulin homeostasis, BP should be reduced as well. Brown *et al*. (2002) showed that although exercise did have a positive effect on all the above mentioned variables, none of the factors were major predictors of changes in BP.

4. Conclusion

The anti-hypertensive effect of exercise is poorly understood, but the mechanism that mediates reductions in BP might be linked to long-term physiological adaptations that occur during exercise training (Tsai *et al*., 2004). It is clear that a single mechanism is not solely responsible for reductions in BP after exercise training, but rather a complex interaction between multiple mechanisms. Even if some of the physiological adaptations caused by exercise are not directly attributable to a decreased BP, it might indirectly affect other mechanisms responsible for a decreased BP. Hamer (2006) suggested that it is possibly the same mechanisms that are responsible for the acute BP reduction, such as endothelial-dependant vasodilation and altered sympathetic vascular regulation that are also the drive behind chronic BP reductions.

The structural adaptations in the cardiovascular and musculoskeletal systems also contribute largely to BP reductions, but genetic factors, the associated risk factors and the training status of the person will largely determine the mechanisms involved in reducing BP. In order to obtain significant reductions in BP after exercise training, a well-designed exercise program should be prescribed. Although BP reductions can be seen within one to ten weeks of a program, the effects are immediately reversible with the cessation of exercise (Hagberg *et al*., 2000; Gilders *et al*., 1989; Kiyonaga *et*

al., 1985). Therefore, exercise prescription should not be a short term solution, but a lifelong habit.

D. LAND BASED ENDURANCE EXERCISE

1. Introduction

Aerobic exercise is most often prescribed for the management of hypertension, either prior to the chronic prescription of drugs or in conjunction with pharmacological treatment. The ACSM (2006) recommends moderate intensity dynamic aerobic exercise for at least 30 minutes a day on most days of the week to maintain current health or prevent cardiovascular disease. There is, however, little evidence that this is sufficient to decrease BP in persons with hypertension. There is also no conclusion on what the magnitude of the reduction in BP might be (Cooper *et al*., 2000). Due to differences in experimental design and the numerous factors that could influence BP, it is difficult to compare studies to one another and to draw a conclusion on the most optimal volume of exercise to induce maximal BP reductions.

2. Effect of aerobic exercise in normotensive individuals

A reduction in resting BP after exercise training is not a common feature in normotensive individuals, although it has been shown occasionally (Cornelissen & Fagard, 2005; Wallace, 2003; Pescatello & Kulikowich, 2001). Gilders *et al*. (1989) showed that after 16 weeks of aerobic exercise training at 80% HR_{max}, normotensive (46 years) subjects showed no change in resting or ambulatory BP, irrespective of the increase in aerobic power. Jessup *et al*. (1998) investigated the effects of a 16 week exercise program on resting and ambulatory BP in 61 to 77 year old normotensive adults. The program consisted of three one hour sessions of dynamic exercise a week. A non-significant reduction in resting BP was reported in the exercise group, but not in the control group after the exercise program. The researchers also compared 24 h ambulatory BP monitoring before and after the 16 week exercise program. It was reported that the systolic and diastolic ambulatory BP was significantly lower for 24 hours following the exercise program. This was seen only in the exercise group. The researchers concluded that endurance training

decreases the day to day BP in healthy older adults and prevents the rise in BP associated with ageing.

Similarly Goldberg *et al*. (1994) examined the cardiovascular effects of 16 weeks of aerobic and weight training in adult normotensive men. They showed no significant change in resting SBP after the weight training or aerobic exercise groups. When the ambulatory BP before and after the exercise program was compared, the exercise group showed a mean 24 h SBP decrease of 7.9 mmHg and diastolic decrease of 3.6 mmHg, while the control group showed no significant changes in 24 h BP. The ambulatory BP of the exercise groups was lower at every hour compared to the control group. Gilders *et al*. (1994) also showed that moderate (70% VO_{2peak}) and high intensity (80 – 85% VO_{2peak}) aerobic exercise can significantly reduce resting SBP (8 mmHg) and DBP (7 mmHg) in normotensive elderly subjects after 26 weeks of training, despite no change in body composition.

It can be concluded that exercise training does show hypotensive capabilities in resting and ambulatory BP in normotensive subjects. Two studies showed that the ambulatory BP is reduced during activities of daily living in younger and older adults. This strengthens the evidence that regular exercise contributes to the prevention of hypertension by reducing day to day BP even in a healthy, normotensive population.

3. Effect of aerobic exercise in hypertensive individuals

Tsai *et al*. (2002) examined the effect of a 12 week dynamic aerobic exercise program on resting BP in hypertensive, middle-aged subjects. They showed a significant maximal decrease in both SBP and DBP of 18 mmHg and 10 mmHg, respectively, compared to baseline values. The researchers concluded that the reductions were equivalent to the effect of anti-hypertensive medication and that the BP can decrease to normal levels after an endurance exercise program. The BP was, however, only measured by auscultation and not by an automated device and thus could have been subjected to observer bias.

Tsai *et al*. (2004) also reported significant decreases in SBP and DBP after a 10 week moderate intensity, dynamic exercise program in hypertensive, middle-aged

patients. In this case, the maximal reduction in SBP was 13.1 mmHg for SBP and 6.3 mmHg for DBP. Similarly Kiyonaga *et al*. (1985) showed a 14 mmHg and 9 mmHg decrease in SBP and DBP respectively after 10 weeks of moderate intensity aerobic exercise in stage 1 and 2 hypertensive adults between the ages of 34 and 56 years. After 20 weeks of exercise, a further reduction of 3 and 4 mmHg was observed in the SBP and DBP respectively, but not all participants completed 20 weeks of training. Interestingly, not all subjects responded positively to exercise. It was shown that the responders showed lower plasma renin values, which could have contributed to the larger decreases in BP.

Van Hoof *et al*. (1989) examined the effect of exercise training on resting BP over 24 hours of daily activities in pre-hypertensive individuals. It was shown that resting BP tended to decrease, but only for DBP (5 mmHg). Although the SBP decreased by 4 mmHg at rest, the reduction was not significant after the 48 training sessions of intermittent aerobic and calisthenics exercises. DBP during the daytime hours decreased significantly by 5 mmHg.

Although exercise is widely prescribed in the management of stage 1 hypertension, not all studies show a positive effect of exercise on BP. Blumenthal *et al*. (1991) tested the effect of a 16 week exercise program in mildly hypertensive men and women. The participants were randomly divided into three groups, namely the aerobic exercise group, the strength and flexibility group and the control group. The flexibility and strength group was included to control for non-specific effects, such as social stimulation and attention from trainers, or of being in an exercise group. They completed 20 minutes of flexibility training and 30 minutes of circuit weight training with no cardiovascular exercise. The aerobic exercise group completed three exercise sessions a week (walking or jogging at 70% VO_{2max}). The control group continued daily routines with no intervention. Although all three groups showed significant decreases in resting BP, the aerobic exercise group had no clear advantage in BP reductions. They also showed no difference in change in ambulatory BP before and after the 16 week training. They did, however, find a correlation between an increase in aerobic capacity and a decrease in DBP. The group with the highest gains in VO_{2max} also showed the greatest reductions in DBP. They concluded that there is little support for the anti-hypertensive effects of aerobic

exercise training alone and that the mere involvement in a research study might have resulted in the decline in BP in the non-exercise group. It was therefore concluded that aerobic exercise alone should not be considered as a replacement for pharmacological treatment in mild hypertensive patients.

Cooper *et al*. (2000) investigated the magnitude of BP reductions after a home-based, unsupervised moderate intensity walking program in persons with hypertension. The participants were asked to expend, in addition to their normal daily activities, 150 to 200 kcal of energy during 30 minutes of aerobic activity. Participants exercised five days per week for six weeks. Compared to baseline, participants in the exercise group showed a decrease in ambulatory BP after the exercise while the control participants showed a small, non-significant increase in BP. The differences between the control and intervention groups failed to reach statistical significance. The researchers concluded that moderate intensity exercise alone is not enough to cause a significant decrease in BP in hypertensive patients. The reduction in BP (2.8 and 1.9 mmHg for SBP and DBP respectively), is however, important in decreasing the risk for cardiovascular disease and can be an adjunct to pharmacological treatment. An important consideration in this study is that the exercise program was home-based and, although participants wore accelerometers, the report of physical activity might not have been accurate. Furthermore, the intensity and volume of exercise was not controlled and might not have been sufficient to result in a significant decrease in BP. The control group was instructed not to change its physical activity, but the levels of physical activity were not monitored.

Gilders *et al*. (1989) also showed that after 16 weeks of aerobic exercise training in hypertensive adults (46 years) there was no change in resting or ambulatory BP, despite the increase in aerobic power. They showed no change in CO or TPR after the training period, supporting the notion that there was no change in BP after exercise training.

The differences in the BP changes after exercise training can be attributed to variations in study design and methodological matters. In some of the studies automated BP devices were not used and the investigator was not blinded to the

exercise and control groups. Not all studies randomized the control and treatment periods and daily activities were not monitored. In some cases the ambulatory monitoring commenced directly following the last training session, therefore the decrease in ambulatory BP might not have been the result of the exercise training, but rather due to PEH after an acute session. The difference in exercise intensity and study duration might also contribute to the differences in the magnitude of the BP response observed. Furthermore, individual differences also influence the response to exercise training and will contribute to the varying results found in the literature.

Even though some studies do not show a reduction in BP after exercise, the majority of the literature supports the notion of a decreased BP after exercise training. In a meta-analysis conducted by Cornelissen & Fagard (2005), it was concluded that resting as well as ambulatory BP significantly decreased with endurance training. Reported decreases in systolic and diastolic BP was an average of 3 / 2.4 mmHg, respectively, for resting BP whereas daytime ambulatory BP is decreased by 3.3 / 3.5 mmHg respectively in the general population, which included normotensive and hypertensive individuals. They also concluded that these reductions are most pronounced in individuals with hypertension and higher baseline BP reaching a reduction of 6.9 and 4.9 mmHg for SBP and DBP respectively. It was also reported that after the exercise intervention, daytime BP was significantly lower, but that night time BP was much less affected.

4. Other beneficial effects of aerobic exercise

Although aerobic exercise alone does not always have significant BP lowering effects in hypertensive individuals, regular aerobic exercise also has other known health benefits. For instance, exercise training triggers morphological and functional modifications which result in an increased exercise capacity and a less severe acute exercise response. It has been reported that endurance exercise training for four weeks and longer causes significant increases in cardiovascular fitness (Cornelissen & Fagard. 2005; Fagard, 2005; Tsai *et al*., 2004; 2002; Jessup *et al*., 1998; Braith *et al*., 1994; Seals & Reiling, 1991; Blumenthal *et al*., 1991; Gilders *et al*., 1989) despite the fact that the increase in fitness is not necessarily accompanied by changes in anthropometric measures. Some researchers reported significant changes in BMI,

body mass, waist circumference and body fat percentage (Cornelissen & Fagard, 2005; Fagard, 2005; Braith *et al*., 1994) while others reported no change in any of the body composition parameters (Tsai *et al*., 2002; Jessup *et al*., 1998; Seals & Reiling, 1991; Blumenthal *et al*., 1991; Kiyonaga *et al*., 1985). The absence of decreased weight or body fat percentage could be the result of insufficient training intensity or duration in some of the studies. Other positive effects of endurance training are a decreased resting HR (Cornelissen & Fagard, 2005; Fagard, 2005; Goldberg *et al*., 1994; Gilders *et al*., 1989; Kiyonaga *et al*., 1985) and improved glucose and insulin metabolism (Cornelissen & Fagard, 2005; Hagberg *et al*., 2000). Significant decreases have also been reported in total cholesterol, triglycerides, LDL-cholesterol (14%) and a statistically significant increase of 12% in HDL-cholesterol (Tsai *et al*., 2002). Other reported benefits of exercise training include an improvement in left ventricular mass index, ejection fraction, CO, SV and HR variability. All these adaptations will lower the mortality and morbidity rate in hypertensive individuals (Painter, 2008).

Further important changes that have been reported after endurance exercise training are an improved quality of life and less bodily pain when assessed by means of psychological questionnaires (Tsai *et al*., 2004). It is unclear whether hypertension medication would have equivalent effects on quality of life. Furthermore, whether patients experience improved quality of life due to lowered BP or because of the exercise *per se* is still open to debate.

5. Influence of type, frequency and intensity of exercise training on BP reductions

According to available literature, exercise variables such as intensity, frequency and duration only explain 4.9% of the variance in the response of SBP and 1.1% of DBP. This was concluded when comparing studies that examined the effect of different training variables on the magnitude of BP reductions (Fagard, 2001). Similarly Cornelissen & Fagard (2005) as well as Fagard (2005) concluded from their meta-analysis that despite the large range of different training characteristics of the studies, they could not find any significant relationship between BP response and

training characteristics. They did, however, conclude that the magnitude of BP reduction was associated with a gain in aerobic capacity.

Seals & Reiling (1991) investigated the effect of regular low-intensity aerobic exercise on 24 hour arterial BP in hypertensive adults over the age of 50. All subjects participated in a six month aerobic exercise intervention at an intensity of 40%-50% HRR. A sub-group of subjects continued the exercise intervention for an additional six months at a higher intensity and training volume than the first six months. After the first six months, it was reported that there was a reduction in the resting BP in the exercise group, but that it was not significantly different from the reduction seen in the control group. In the 24 h ambulatory BP measures there was no significant reduction in the daytime or mean BP in either of the groups indicating that an exercise intensity of 40%-50% HRR is not sufficient to decrease resting or ambulatory BP. After the next six months of exercise there was a significant decrease in 24 h SBP and mean BP during the day time hours compared to baseline and control values. There was no change in the DBP or any of the measures during day or night time. This study showed that low intensity exercise over six months was not sufficient to induce BP reductions in adult hypertensive individuals, but that a higher intensity might be more beneficial. It may be possible that a low intensity exercise program will be sufficient if the exercise frequency is more and of a longer duration.

There seems to be agreement that exercise of moderate intensity (40 – 70% VO_{2max}) significantly lowers BP in most hypertensive individuals (Tsai *et al*., 2004; Fagard, 2001). Furthermore, exercise intensities greater that 70 – 85% VO_{2max} may not provoke any additional benefits (Pescatello *et al*., 2004; Touyz *et al*., 2004; Hagberg *et al*., 2000) and may also carry a higher risk for possible injury and adverse cardiac events (Tsai *et al*., 2004).

Another training variable that can be altered in an exercise program is the frequency of training per week. Most studies reported exercise programs consisting of three exercise sessions per week (Tsai *et al*., 2004; 2002; Seals & Reiling, 1991; Cononie *et al*., 1991) with a variety of results (positive and negative) reported from these studies. Some researchers suggest that higher training frequencies will result in

greater reductions in BP (Pescatello *et al*., 2004), however, there is no evidence that higher training frequencies, more than 3 times per week, will result in a greater decrease in BP (Wallace, 2003; Fagard, 2001). It seems then that exercise frequency is not a major determinant of the BP response to exercise, or the magnitude of BP reductions after training.

Indications are that the magnitude of resting BP reductions is also not dependent on the duration of an exercise session. Exercise sessions lasting between 30 and 60 minutes elicited similar reductions in BP (Pescatello *et al*., 2004). Most studies implemented exercise programs lasting between 10 and 16 weeks (Fagard, 2006). It was concluded that exercise training lasting longer than 16 weeks did not result in additional decreases in BP. In fact, the SBP reduction became less pronounced, possibly due to a decrease in the compliance of patients (Fagard, 2006; Wallace, 2003; Fagard, 2001). Hagberg *et al*. (2000) concluded that a significant reduction in SBP could be observed within one to 10 weeks of exercise training.

It can be concluded from the literature that the intensity of exercise is a more important consideration in the constitution of a training program than the frequency or duration of the exercise. Furthermore, exercise training should become a lifestyle, not just a temporary solution for a couple of weeks. Therefore the issue that should rather be addressed is what the optimal volume of training per week should be to have long lasting effects on high BP.

E. LAND-BASED RESISTANCE TRAINING

1. Introduction

The recommendations for aerobic exercise training for individuals with a high risk for cardiovascular disease (CVD) are widely known. Although resistance training is gaining more support for persons with a high risk for CVD, the prescription when combined with aerobic training remains unclear. Aerobic training alone increases cardiorespiratory fitness (VO_{2max}), but the combination with resistance training might have added benefits, particularly for the musculoskeletal system.

2. Motivation for resistance exercise

Ageing and inactivity (especially from the fifth decade of life) are associated with a lower resting metabolic rate, gains in fat mass, a decreased aerobic capacity and muscle wasting that increases the risk for cardiovascular and metabolic diseases (Cononie *et al*., 1991). Muscle wasting leads to reductions in muscle strength and endurance, increasing the risk for falls and decreasing the ability to perform functional daily activities. Through resistance training one can increase muscle mass and optimize the response to aerobic conditioning, decrease body fat percentage, increase metabolic rate and thereby increase metabolic control in those at risk for diabetes (Marzolini *et al*., 2008; Umpierre & Stein, 2007; Wilborn *et al*., 2004; Goldberg *et al*., 1994; Cononie *et al*., 1991). It has also been reported that resistance training will contribute to a lower HR and BP after exercise, improved balance, increased strength of connective tissue and increased bone density (Wilborn *et al*., 2004). The ACSM therefore recommends resistance training as an adjunct to aerobic exercise to prevent or treat hypertension (American College of Sports Medicine, 2006; Pescatello *et al*., 2004).

According to Winett and Carpinelli (2001) the required amount of resistance exercise to produce adaptations is not necessarily a large volume of exercise, because there is no linear relationship between volume of exercise and strength gains. Wallace (2003) is of opinion that hypertensive individuals should aim at improving muscle endurance rather than muscle strength. Programs that therefore focus on high repetitions and low absolute weights should be adopted.

Resistance training is considered a very time-efficient and cost-effective training alternative with very few contra-indications. Resistance training can thus be implemented safely and effectively without major risk to persons with or at risk of cardiovascular disease (Wilborn *et al*., 2004).

3. Effects of resistance training on BP reductions

There is no conclusive evidence that resistance training exacerbates high BP or is associated with adverse cardiac events, but it is advised that resistance training not

be prescribed as the primary mode of exercise, but as an adjunct to cardiovascular exercise and that DBP should be monitored continuously throughout training (ACSM, 2006).

Several studies have been conducted to examine the effect of combined aerobic and resistance exercise on BP response. Although resistance training significantly increases muscle strength and endurance (Marzolini *et al*., 2008; Stewart *et al*., 2005; Goldberg *et al*., 1994; Cononie *et al*., 1991), some authors found that it is not enough to cause reductions in resting BP (Goldberg *et al*., 1994; Cononie *et al*. 1991). A meta-analysis by Cornelissen & Fagard (2005) revealed that resistance training causes significantly lower DBP, but not necessarily SBP. On the other hand it has been shown that aerobic training alone (Goldberg *et al*., 1994; Cononie *et al*. 1991), as well as combined resistance and aerobic exercise, significantly decreases BP (Marzolini *et al*., 2008; Stewart *et al*., 2005). Combined resistance and aerobic training also resulted in greater increases in aerobic fitness, muscular strength, and lean body mass compared to aerobic or resistance exercise alone (Marzolini *et al*., 2008; Stewart *et al*., 2005). Goldberg *et al*. (1994) reported that after 16 weeks of endurance and weight training there was a significant reduction in RPP and therefore the myocardial oxygen demand during exercise decreased. Researchers also showed a decrease in submaximal HR after endurance and resistance training.

3.1 Circuit weight training

If there are concerns that resistance exercise may have adverse effects in persons with high BP, or that the cardiovascular benefits are not sufficient, one can always consider circuit weight training. Circuit weight training is the performance of several different resistance exercises using lighter weights with minimal rest between exercises. This type of program therefore aims to improve endurance more than absolute strength or muscle mass.

Harris & Holly (1987) evaluated the efficacy and safety of a circuit weight training program in individuals with hypertension. The subjects performed three sets of 10 different exercises, three times a week, for a nine week period. There were significant improvements in the 1RM for bench and leg press in the exercise group,

as well as a significant absolute increase in VO_{2max} of 11%. The exercise group also showed a 7% decrease in DBP, but no change in resting HR or SBP. It appears that an adequate training stimulus and short rest intervals are key components in weight training programs so as to elicit improvements in cardiovascular capacity.

The available literature gives abundant information about the effectiveness of resistance training in persons with hypertension, persons with other chronic disease and in older individuals. There is no reason to believe that resistance exercise will be hazardous to high risk individuals if implemented correctly and under supervision of a trained person. In fact, the benefits gained from resistance training are a key component in maintaining a healthy metabolic rate, muscular endurance and strength and to maintain a high quality of life and an independent lifestyle for as long as possible. Even though resistance training is not prescribed as the main focus of hypertension management and prevention programs, it would still contribute positively to some of the mechanisms possibly responsible for decreased BP after exercise training.

F. WATER-BASED EXERCISE

1. Introduction

Water exercise is regularly prescribed as a means of increasing cardiovascular fitness and it is gaining popularity as a vertical exercise medium during aquatic therapy (Meredith-Jones *et al*., 2009; Barbosa *et al*., 2007; Darby & Yaekle, 2000). It is especially well-suited for persons with injuries to the ankles, knees, hip and back. It decreases the load on the injured body part due to a decrease in the compressive forces on these joints. It is also prescribed for older persons, persons with impaired balance and persons with impaired exercise ability due to the decreased gravity acceleration and increased density with water immersion. This ensures that all activities are slowed down and are less demanding and traumatic on the person. In obese or overweight individuals the load on the joints is reduced because of the buoyancy of the water. Exercise can involve the upper and lower extremities through an optimal range of motion while restricting joint stress. Water immersion demands a different response of the muscular apparatus which lowers the mechanical work

performed by the muscle. The involvement of anaerobic metabolism is therefore less than with land activities making it a safe environment for low-impact activity (Barbosa *et* al., 2009; 2007; Broman *et al*., 2006; Benelli *et al*., 2004; D'Acquisto *et al*., 2001; Hall *et al*., 1998; Whitley & Schoene, 1987).

The advantages of water exercise are not limited to injured or older individuals. Healthy individuals also make utilize water exercise to increase cardiovascular fitness, flexibility, conditioning and wellness. Aquatic exercise utilizes different muscle groups compared to land-based exercise and it ensures variety in an individual's exercise routine. It is also a valuable tool in the enhancement of sport performance and rehabilitation of injuries in elite athletes (Broman *et al*., 2006). It has been reported that a session of shallow water (submerged to hip level) exercise in younger and older women is sufficient to meet the intensity guidelines set by the American College of Sports Medicine (2006) to produce health benefits such as improved cardiovascular fitness and muscular endurance as well as a reduced risk of developing premature chronic disease (Barbosa *et* al., 2009; Benelli *et al*., 2004; Campbell *et al*., 2003; D'Acquisto *et al*., 2001; Darby & Yaekle, 2000; Hall *et al*., 1998; Eckerson & Anderson, 1992; Whitley & Schoene, 1987).

2. Physiological response during water exercise

2.1 Acute hemodynamic changes during water immersion

2.1.1 *Changes during water immersion at rest*

The hemodynamic responses at rest and during water immersion exercise are altered due to the effect of hydrostatic pressure and the temperature of the water (Broman *et al*., 2006). Water immersion results in an approximately 10 $b.min^{-1}$ lower resting HR than on land (Barbosa *et al*., 2007; Darby & Yaekle, 2000). Fujisawa *et al*. (1995) also reported lower HR measured in the seated position at rest during water immersion than compared to a similar measurement on land. This is possibly the effect of the hydrostatic pressure resulting in a redistribution of blood volume away from the extremities to the abdominal and thoracic cavities. In healthy subjects immersed in thermo-neutral (35℃) water, the hydrostatic pressure causes a

redistribution of approximately 700ml of blood away from the limbs to the thoracic cavity (Park *et al*., 1999). This increased thoracic blood volume results in a higher central venous pressure leading to an increased pre-load. According to the Frank-Starling mechanism, this results in a rise in SV and therefore an augmented CO (Cider *et al*., 2005; Šrámek *et al*., 2000; Park *et al*., 1999).

During water immersion at rest, Park *et al*. (1999) reported a significantly elevated SBP (8% – 11%) and DBP (15%). This elevated BP response at rest happened in spite of a decreased TPR at rest. The decreased TPR implies that peripheral vasodilation occurs during water immersion. However these findings are not universal, as some authors have also reported a decrease (Šrámek *et al*., 2000; Darby & Yaekle, 2000) or no change (Cider *et al*., 2005; Hall *et al*., 1998; Fujisawa *et al*., 1995) in resting BP with water immersion.

2.1.2 *Changes during exercise*

Darby & Yaekle (2000), as well as Hall *et al*. (1998), reported higher energy expenditure and HR in chest deep water exercise (deep water immersion) than land exercise during submaximal exercise. Darby & Yaekle (2000) showed that at the same HR, deep water exercise at 30°C elicited a 21% higher VO_2 in female college students than the same type of exercise on land. For the same VO_2, HR during water exercise was 7 – 13 $b.min^{-1}$ lower than land exercise at submaximal intensities, but not at higher intensities. The authors also reported higher caloric expenditure during water exercise than land-based exercise and concluded that participants were working harder in the water at the same HR than on land. This could be the result of the added resistance component of the water. Hall *et al*. (1998) reported similar results for treadmill walking, i.e. a higher VO_2, HR and RPE during water exercise compared to the same speed of treadmill walking on land at higher speeds. At a slow treadmill speed, when drag forces were minimal, the HR during water exercise was lower than during land exercise. When walking in the water at the same speed as on land, it is expected that the HR and energy cost would be higher during the water exercise due to the increased drag force of the water, hence the higher RPE during water exercise. Therefore it can be concluded from this study that the relative

workload was much higher in the water compared to on land, hence the higher HR and RPE.

During shallow water walking Whitley & Schoene (1987) also reported higher exercise HR for a set speed, distance and duration when compared to land treadmill walking in healthy female college students. The pre-selected speeds of 2.55, 2.77, 3.02 and 3.31 $km.h^{-1}$ were of sufficient intensity to improve cardiorespiratory fitness in water, but not during treadmill walking on land. The increase in HR for the treadmill walking on land was only 13% compared to resting HR at the fastest speed, whereas for the water walking the HR increased by 135% from rest. This could be expected, because the buoyancy and decreased movement speeds in the water add a resistance component that decreases work efficiency therefore requiring more work effort in the water compared to on land. A higher treadmill speed should have been selected on land to induce the same, sufficient exercise stimulus to increase HR in a similar manner than during the water exercise. The results of this study should therefore be interpreted with care.

On the other hand, Silvers *et al*., (2007) reported that during peak treadmill exercise in shallow water and on land, there was no significant difference in maximal HR and oxygen consumption. Although the breathing frequency and minute ventilation was higher during water exercise, there was no difference in the peak cardiorespiratory results and are therefore comparable for land and water exercise. The possible explanation for the greater minute ventilation and breathing frequency is that the work of breathing in water is greater than on land. The muscles used in breathing consume more oxygen causing a decrease in tidal volume. The breathing frequency and minute ventilation has to increase to obtain the same VO_2.

Eckerson & Anderson (1992) reported that when doing sub-maximal water exercise at an intensity to the equivalent of 82% of HR_{max} during a maximal treadmill test on land, subjects only obtain VO_2 values of 48% comparable to land exercise. This means that at the same relative HR for land and water exercise, oxygen consumption was lower in the water than on land. Broman *et al*. (2006) also reported a lower HR and VO_2 during water treadmill running compared to land treadmill running. This shows that compared to land exercise the relationship between HR and VO_2 during

water exercise is altered. Therefore at submaximal loads, water exercise must be performed at higher intensity to obtain the same relative oxygen consumption compared to on land. Even though HR might be comparable to land exercise, the oxygen consumption during water exercise is lower than during land exercise and cannot be predicted from HR. Water exercise must be performed at a higher intensity to improve cardiovascular fitness and have a transfer effect to land exercise.

It is difficult to compare the results between studies, because most of the studies used different exercise intensities and the hemodynamic response during water exercise is largely dependent on the intensity of the effort. A sound conclusion regarding the HR – VO_2 relationship during water exercise cannot be drawn. The biomechanics of water treadmill, deep water running with a flotation device and water aerobics also largely differs and will surely affect the energy cost during exercise. Other extraneous variables like body position, water depth and water temperature might also complicate the interpretation and comparison of results.

Furthermore, when comparing shallow and deep water exercise to land-based exercise, it has been reported that water exercise induces a higher RPE, but a lower HR and lactate concentration (Barbosa *et al*., 2007; Benelli *et al*., 2004). The differences in land versus water-based exercise can be explained by the change in thermoregulation and conduction of body heat, the decrease in activity of the postural muscles due to a decreased gravitational force and the decreased cardiovascular work due to the hydrostatic pressure of water immersion (Barbosa *et al*., 2009; 2007; Benelli *et al*., 2004). This confirms the suggestion that exercise intensity should be adjusted when exercising in water to ensure that the same training effect is achieved.

When exercise intensity during water exercise is adjusted to induce the same cardiovascular effect as on land, it is expected that the BP will change accordingly. Park *et al*. (1999) showed that SBP increased significantly from rest during both land and water exercise, while DBP stayed relatively constant compared to resting values. This is similar to the BP changes observed during land exercise (McArdle *et al*., 2001). This suggests that the hemodynamic and cardiovascular adjustment during dynamic exercise was not changed by water immersion despite the increase in cardiac preload (Park *et al*., 1999). In contrast Hall *et al*. (1998) reported a greater

rise in SBP in water exercise than on land, especially in warmer water (35.8℃). However, the exercise intensities for the land and water exercises in this study were not the same. Due to the drag force of the water, the intensity in the water was expected to be higher than on land for the same treadmill walking speed, hence the greater rise in SBP during water exercise. The DBP did not change after exercise in 28.2℃ water, but a higher water temperature (35.8℃) resulted in a significant decrease in DBP. Fujisawa *et al*. (1995) measured the change in BP during isometric exercise on land and in water. They showed that although the exercise in water tended to show lower mean BP than on land, there was no significant difference. The order of the exercise in this study was not randomized and the slight differences in results could be due to a learning effect.

It can be concluded that water exercise will result in a higher resting SBP and DBP merely due to the effect of water immersion, but that the relative increase in SBP during exercise is similar or slightly higher than in the case of land exercise. The peak SBP would be higher during water exercise mainly due to the elevated resting values. The DBP would also increase at rest and therefore be higher during exercise, but would not show further increases during exercise. This is similar to land exercise. There is, however, no evidence that water exercise has any adverse effects on the BP response.

2.2 Factors affecting the acute adaptations to water immersion exercise

2.2.1 *The effect of water temperature*

Body temperature is greatly affected by the environment and exercise which in turn affects the physiological responses of the body. During immersion in water, body temperature changes due to conduction and convection caused by the high thermal conductivity of water. Water has 25 times the convection capacity of air and therefore body heat is lost four times faster in water than in air at the same temperature (Drinkwater, 2008). The colder the water, the faster heat would be lost from the body (Barbosa *et al*., 2009). Consequently a number of studies investigated the effect of exercise at different water temperatures on the physiological responses in humans.

Cold water exposure of about 14℃, when compared to air temperature, causes an increase in HR of 5%, SBP of 7% and DBP of 8% due to an increased activity of the sympathetic nervous system. In neutral water temperature (~ 32℃), metabolic rate is unchanged, but HR, SBP and DBP is lowered by 15%, 11% and 12% respectively (Šrámek *et al*., 2000). In contrast Park *et al*. (1999) reported a lower SV and a higher HR and CO in thermo-neutral water (34.5℃) compared to colder water (30℃). Similarly Hall *et al*. (1998) showed that HR was significantly higher in 35.8℃ water than in 28.2℃ at the same treadmill walking speed, but that VO_2 and RPE did not differ between the temperatures. It seems that at temperatures higher than 34℃ the HR is significantly higher compared to temperatures below 32℃.
It is speculated that in colder water, peripheral vasoconstriction increases the TPR, resulting in an increased central blood volume. This enhances SV and results in a reflex bradycardia (Hall *et al*., 1998). Consequently the SBP, DBP and MAP are higher in colder water than in neutral water (Park *et al*., 1999). During hot water immersion (about 40℃), HR is increased during and after immersion in spite of a decrease in sympathetic nervous system activity. This is due to the slowed enzymatic processes caused by the hot water, which causes slower nerve conduction; this impairs the rate of force development and decreases muscular endurance during dynamic exercise (Drinkwater, 2008). In a cold environment, blood will be redistributed away from the skin and extremities whereas in a hot environment there will be competition for blood flow between the muscles to deliver oxygen and nutrients and the skin to facilitate heat loss. Thus HR and BP responses, as well as VO_2 will be affected by the temperature of the environment.

Water temperatures around 27 - 30℃ are most frequently prescribed for water exercise (Broman *et al*., 2006). At these temperatures the physiological responses are closely related to land-based exercise. The influence of exercise mode, intensity, duration and the population performing the exercise will also affect the physiological responses during exercise. It is necessary to maintain a water temperature that ensures no discomfort and thermoregulation stress to the person. In older populations and more flexible types of exercise, a warmer temperature should be considered whereas when working with a younger population doing cardiovascular fitness types of activity, a cooler temperature should be considered (Barbosa *et al*., 2009).

2.2.2 *The effect of water depth*

Exercise effort is perceived differently when done at different levels of immersion. Shallow water exercise (up to hip level) results in a higher HR, RPE, VO_2 and energy expenditure compared to deep water exercise (immersion to chest level) (Barbosa *et al*., 2009; 2007; Benelli *et al*., 2004). The increase in RPE at hip level could be explained by the higher drag forces in the lower limbs compared to the trunk and upper limbs during deep water immersion. There is also an increased ground reaction force due to decreased buoyancy with shallow water immersion. The change in neuromuscular patterns in the active muscles at different levels of immersion may also contribute to the change in perception (Barbosa *et al*., 2007).

Benelli *et al*. (2004) showed that deep water immersion exercise resulted in a lower heart rate (48 $b.min^{-1}$) than land exercise whereas shallow water exercise produced a slightly lower heart rate (7.5 $b.min^{-1}$) compared to land-based exercise. These results were confirmed by Barbosa *et al*. (2007). It can be concluded that the deeper the water, the less the physiological demand. This can be explained by the increased volume of blood redistributed to the trunk area due the increased hydrostatic pressure which enhances heart filling during diastole and in turn leading to a greater SV (Barbosa *et al*., 2009). These findings imply that shallow water exercise is more advantageous to maintain cardiovascular fitness than deep water exercise because the ground reaction force and gravitational acceleration in shallow water is still higher than during deep water exercise. This has implications for obese persons and persons with lower limb injuries where the least possible impact during exercise is preferred. Deep water exercise also allows for exercise of the upper body and trunk musculature whereas shallow water exercise only provides an exercise stimulus to the lower extremities.

2.2.3 *The influence of exercise modality*

Similar to land based training, the type of activity in the water will affect the physiological demands during exercise. The faster the movement through the water (i.e. walking compared to running), the greater the drag force and therefore the resistance in the water. This demands a higher force production to overcome the

external load leading to a higher HR, VO_2 and RPE (Darby & Yaekle, 2000; Hall *et al*., 1998). Therefore, increasing exercise intensity in the water places a greater load on the cardiovascular system, just as during land based exercise.

The number of body segments involved in the exercise will also influence the metabolic and cardiovascular demand. If arm and leg exercises are combined during a deep water immersion exercise, a higher HR, lactate concentration and RPE are observed when compared to leg only exercise in both land and water (Darby & Yaekle, 2000). Combining arm and leg exercise results in a higher drag force and this promotes a higher oxygen and nutrient demand with concomitant increase in HR and metabolite production (Hall *et al*., 1998). Therefore, combining arm and leg exercise will increase the exercise intensity and result in greater gains in cardiovascular fitness when compared to leg exercise alone.

One can also incorporate different training tools during water exercise to adjust the level of difficulty of the exercise and incorporate variety into the program. These tools include flotation devices, water weights, ankle cuffs and aqua steps. The flotation device is frequently used when exercising in deep water. This allows the person to be submerged, but still stay upright while performing the activities. Exercising in deep water without a flotation device is more demanding on the system than with the device, because the person has to maintain buoyancy while performing the exercise. In inexperienced persons, performing exercise without a flotation device thus results in a higher VO_2, VE, HR and RPE. The physiological response will further be influenced by body composition, the amount of drag force, and the amount of propulsive force (Barbosa *et al*., 2009).

Changing the body position during exercise will also have an effect on the physiological response. HR will be lower during exercise in a horizontal compared to a vertical position in the water, because of the increased filling time during diastole, therefore increasing stroke volume. Cardiac output will thus remain constant (Barbosa *et al*., 2009; Benelli *et al*., 2004).

Numerous factors should be considered when prescribing water exercise in the maintenance of cardiovascular fitness. Firstly the physiological characteristics and

training status of the person should guide the intensity of the exercise. The water temperature, depth, body position and exercise tools used will also influence the exercise stimulus and therefore the hemodynamic response. There is no evidence that exercise in any temperature or depth of water resulted in adverse cardiovascular response. There is, however, no certainty regarding the acute BP response during and especially after exercise in normotensive or hypertensive persons.

3 Chronic adaptations to water exercise

3.1 Cardiovascular adaptations

The adaptations of the cardiovascular and metabolic systems to water-based exercise are of special interest, because if sufficient, it could play a role in the prevention of chronic diseases such as coronary artery disease, hypertension, diabetes and obesity (Meredith-Jones *et al*., 2009).

The mechanisms responsible for the adaptation of the cardiovascular system is usually explained as adaptations in the peripheral skeletal muscles suggesting an increased arterial-venous oxygen difference, increased capillary density and increased mitochondrial enzymes. Broman *et al*. (2006) reported that the hydrostatic pressure during water immersion has a stimulatory effect on capillary proliferation and oxidative enzyme activity and one would thus expect an improvement in exercise capacity.

Some studies reported a significant increase in VO_{2max} after a deep water and shallow water training program in untrained, elderly women (Meredith-Jones *et al*., 2009; Broman *et al*., 2006; Taunton *et al*., 1996). In competitive athletes no change in maximal exercise capacity has been reported (Barbosa *et al*., 2009), while Pechter *et al*. (2003) also failed to induce higher VO_{2max} results in patients with chronic kidney disease after a 12 week low intensity water based exercise program. As is the case with land-based exercise, the change in cardiovascular fitness and aerobic capacity depends largely on the initial training status of the person and the intensity and duration of the training program.

It has also been speculated that resting HR was lower after an aquatic training program (Barbosa *et al*., 2009). Broman *et al*. (2006) also showed that during a submaximal work capacity test the submaximal HR decreased between four and 15 $b.min^{-1}$ after a deep water running program indicating an improved submaximal work capacity on land. Unlike HR, there was no change in resting SBP or DBP after an eight week deep water running program in healthy elderly women. On the other hand, Colado *et al*. (2009) reported a decrease in resting DBP after 24 weeks of land and water resistance training. The difference in results could be attributed to the different modes of exercise as well as the duration of training. In the study by Broman *et al*. (2006) subjects only trained twice a week for eight weeks, whereas Colado *et al.* (2009) trained twice a week for the first 12 weeks and three times a week thereafter. Both studies implemented high intensity training, thus it is still inconclusive what the effects of lower intensity water exercise will be on BP. Most research on land exercise reports greater reductions in BP after low to moderate intensity exercise compared to high intensity exercise. Although there is not a lot of evidence on the effects of water exercise on resting BP, there is no reason to believe that it will adversely affect BP. Other cardiovascular benefits, which can influence resting BP, have been reported after water exercise training protocols,

3.2 Muscular strength adaptations

It is feasible that water exercise would provide a sufficient stimulus to improve muscle strength since the drag effect of limb movement in water provides resistance to the muscle movement. Water exercise is therefore also an alternative medium for land resistance exercise and has added advantages of less compressive forces on the joints compared to land exercise. Colado *et al*. (2009) showed that when comparing resistance exercise in water to resistance exercise with elastic bands on land, both interventions significantly increased physical capacity, body composition and decreased resting DBP. Another study on resistance exercise showed that muscle activation is similar or even enhanced in water compared to land exercise and therefore direct comparisons between water resistance exercise and land resistance exercise are possible (Colado *et al*., 2008).

Meredith-Jones *et al*. (2009) reported a 20% increase in upper body strength (chest press exercise) and a 30% increase in lower body strength (knee flexion and extension), tested on land, after a 12 week circuit-based water exercise (resistance training combined with deep water running) in overweight elderly women. The lack of a control group in this study means that results should be interpreted with care, as improvement in muscle strength could be due to a learning effect. Colado *et al*. (2009) reported similar results in a 24 week resistance training program in water, while there was no change in the control group. In contrast, Taunton *et al*. (1996) reported no change in muscle strength or endurance after 12 weeks of water exercise. The researcher concluded that the program was not specific enough to cause increases in muscle strength and that the training program might have been too short to notice any change. This study also lacked a control group. Again, the change in muscle strength in response to a water-based training program will be dependent on the initial training status of the person, the volume of training and the type of exercise performed (Barbosa *et al*., 2009).

3.3 Other

Research has shown that participants improve significantly on the sit-and-reach test after a period of shallow water aerobic and strength exercises. This can be explained by the properties of warm water that result in the reduction of spasticity and improvement in a range of motion resulting in improved flexibility (Barbosa *et al*., 2009; 2007). However, the improvements are dependent on the initial level of flexibility and the training status of the person. There may be no improvement when these studies were repeated in well conditioned athletes.

There are some indications that water exercise may cause a reduction in body fat percentage, but very little conclusive evidence exists (Barbosa *et al*., 2009; Meredith-Jones *et al*., 2009; Volaklis *et al*., 2007). This could be the result of differences in study design, a lack of a controlled diet, different methodologies to measure body fat percentage, a short duration of exercise intervention that is insufficient to produce body composition changes and using non-responsive study populations such as elite athletes (Barbosa *et al*., 2009).

Meredith-Jones *et al*. (2009) reported a decrease in waist and hip circumference after 12 weeks of combined resistance and aerobic deep water exercise in overweight elderly women, suggesting a decrease in abdominal obesity. They failed to show any changes in body mass or BMI. Colado *et al*. (2009) reported decreases in fat mass and waist circumference in postmenopausal women after 24 weeks of resistance training. Additionally, their subjects presented with an increase in fat free mass and a decrease in body mass and BMI. These positive results could be due to the longer duration of their intervention (24 weeks) compared to the 12 week intervention of Meredith-Jones *et al*. (2009).

Taunton *et al*. (1996) reported no change in flexibility, muscle strength or body composition after 12 weeks of land-based and water-based exercise in elderly, sedentary women. They speculated that a generic exercise program prescribed for the elderly is not sufficient to cause any adaptations and that the program needs to be more specific in order to show improvements. It was also concluded that the program was not of sufficient duration and intensity to produce significant changes. The results of this study should be interpreted with care due to the lack of a control group.

Compared to land-based exercise, there is no conclusive evidence on the exact beneficial effects of water exercise. The differences in study design and exercise variables make it very difficult to compare studies and draw conclusions. However, it is clear that there are no adverse effects on BP associated with water- based exercise and that the results are comparable to that of land-based aerobic and resistance training.

4. Water exercise in persons with chronic disease

For many years water activities and swimming have not been prescribed for persons with cardiovascular diseases due to the assumption that it would cause unwarranted increases in left ventricular volume and ultimately worsen the prognosis of the disease. In recent years scientific evidence confirmed that water-based activities can be safely implemented in the rehabilitation and exercise programs of persons with

cardiovascular disease and other risk factors (Volaklis *et al*., 2007; Cider *et al*., 2005).

Hanna *et al*. (1993) showed that similar to healthy individuals, the cardiac preload is also increased during water immersion in men with a history of myocardial infarction. In healthy men it has been shown that although CO is increased during water exercise, the oxygen consumption is decreased at the same relative HR. Hanna *et al*. (1993) showed that this is not the case in men with previous myocardial infarction and that the SV during exercise was not elevated during water exercise. The researchers argued that the scar formation during ischemia resulted in abnormalities of left ventricular systolic and diastolic function that might have contributed to these alterations. Another difference reported in these men was that HR during land and water exercise was the same at all workloads up to 75% of VO_{2peak}.

Both Cider *et al*. (2005) and Pechter *et al*. (2003) successfully implemented a water-based training program in patients with chronic heart failure and chronic kidney disease. Pechter *et al*. (2003) found that a 12 week low intensity water-based program improved the cardiorespiratory reserve, renal function and physical capacity of renal failure patients. There was also a significant reduction in resting SBP and DBP, but not in BMI or blood lipid profile. The researchers concluded that water-based training is safe for persons with renal disease and is more suited for patients who are obese, older and less motivated than is the case with land-based exercise (Pechter *et al*., 2003).

Volaklis *et al*. (2009) implemented a 12 week exercise intervention, (combined aerobic and resistance training) involving persons with coronary artery disease. They compared land-based and water-based programs and also included a non-exercise control group. Both exercise groups showed significant improvements in exercise tolerance, RPP, muscular strength, body composition and lipid profile with no changes in the control group. There was no significant difference in the adaptations observed in the two exercise groups, therefore the researchers concluded that both exercise routines were effective in producing an exercise response without any adverse effects on health status. It is important to note that the

water-based immersion intervention elicited the same adaptations as the land-based program.

5. Conclusion

The available literature suggests that water exercise is a valid alternative to land-based exercise in healthy and diseased persons. No research is available on the effect of water exercise in persons with hypertension. Research regarding the BP response after water-based exercise compared to land-based exercise in hypertensive individuals is therefore warranted.

It is speculated that water exercise would have similar effects as land exercise on the resting BP after training. The acute BP response to water exercise might be different to land exercise due to the changes in haemodynamics as a result of water immersion *per se*. The possible rise in resting HR, SBP and DBP during water immersion might result in a higher absolute exercise BP response. Furthermore, research indicates that during water exercise, persons should exercise at a higher intensity to reach the same workload as during land exercise. The higher resting BP levels and higher exercise BP response due to more intense exercise might be dangerous to individuals with already elevated BP. However, no exaggerated BP response during water exercise under normal circumstance has been reported.

It is hypothesized that the decrease in TPR in the extremities during water immersion might contribute to decreased DBP after exercise leading to a greater PEH than seen during land exercise. The elevated SV, and thus resting CO, might result in an attenuated drop in SBP as opposed to land exercise. The prediction of PEH from the resting and exercise responses might not be accurate.

CHAPTER FOUR

POST EXERCISE HYPOTENSION

A. INTRODUCTION

It is well accepted that chronic exercise training lowers resting blood pressure, but research has also shown that an acute bout of exercise can result in transient reductions in BP as well (MacDonald *et al*., 1999). This temporary decrease in BP is termed post exercise hypotension (PEH) and is defined as the transient decrease in resting BP in the minutes or hours after an acute bout of exercise. Kenney & Seals (1993) defined PEH as a reduction in systolic and / or diastolic arterial BP below resting levels after a single bout of exercise. This decrease in BP is due to a rapid decline in cardiac output from high exercising values to resting values, while the systemic vascular resistance does not recover as quickly and causes a drop in pressure (Halliwill, 2001). Exercise has been explored as a possible alternative treatment for high BP by numerous researchers.

There are no clearly defined criteria for the magnitude or duration of the decreased pressure response. One of the main questions regarding PEH is whether it is clinically significant or just an interesting physiological phenomenon. For PEH to be clinically significant, it needs to produce a significant decrease in arterial BP which must be sustained under activities and stressors of daily living for a significant duration. Most studies have only measured PEH under controlled laboratory conditions, i.e. conditions where the effects of daily stressors and free living activities are eliminated or greatly minimized. If PEH is shown to be clinically significant, it would be sufficient to aid in the treatment and control of hypertension in addition to or as an adjunct to pharmacological treatment (Kenney & Seals, 1993). If PEH lasts for several hours during the day, the patient is protected when it is most needed. Furthermore, the time of day to initiate exercise might contribute to controlling high BP.

B. PHYSIOLOGICAL MECHANISMS UNDERLYING PEH

The exact physiological mechanism of PEH is unclear and most researchers believe it is multifactorial. There are inconsistencies in the available literature regarding the systemic and regional haemodynamics following acute exercise and therefore different mechanisms to explain the PEH have been suggested (Pescatello *et al*., 2004). Some of the reasons for these inconsistencies could be explained by methodological issues, for instance, the differences in age and health status of the participants, the duration of the pre-exercise rest period before baseline measures are taken and the experimental protocols such as different exercise variables, the duration of PEH measurement, the body position during measurement and whether the subject was in a controlled or free-living environment (Cleroux *et al*., 1992).

Another factor that will influence the mechanism of PEH is the cause of hypertension in each person. Mean arterial pressure is determined by cardiac output and total peripheral resistance, therefore a reduction in arterial pressure must be mediated by reductions in one or both of these variables (Kenney & Seals, 1993). Cardiac output is determined by heart rate and stroke volume and because HR is usually elevated after exercise, a reduction in CO must be mediated by a reduced SV (Kenney & Seals, 1993). However, CO is often reported to be elevated after exercise due to a sustained tachycardia, therefore the decrease in arterial pressure is probably the result of a reduction in TPR (Cleroux *et al*., 1992). According to Poiseuille's equation vessel resistance is directly proportional to blood viscosity, length of the blood vessel and inversely proportional to the radius to the fourth power (r^4) of the blood vessel (Brooks *et al*., 2000). Because the blood viscosity and the length of the blood vessel are not altered by acute exercise, the diameter of the blood vessel is the only variable that could influence TPR provided that the individual does not dehydrate (Pescatello *et al*., 2004). Decreases in plasma volume have also been considered in the mechanism underlying PEH, but the drop in plasma volume after exercise is inconsistent and does not contribute to a decreased stroke volume. Therefore, a decrease in plasma volume is unlikely to be a major determinant of PEH (MacDonald, 2002; Kenney & Seals, 1993; Cleroux *et al*., 1992).

Cleroux *et al*. (1992) has shown that the reduction in SBP and DBP after 30 minutes of cycling at 50% VO_{2peak} was the result of a decrease in TPR and a sustained decrease in forearm vascular resistance and plasma norepinephrine in hypertensive subjects. Although the TPR decreased in the normotensive group by 15% as well, the hypertensive group had a more pronounced decrease of about 27% compared to before exercise. CO increased more in the hypertensive group than in the normotensive group due to a greater increase in stroke volume. Both heart rate and cardiac contractility were similar for both groups after exercise. The increased SV could only be explained by a reduction in afterload because the pre-load was not affected. This study concluded that the anti-hypertensive effect of exercise can be explained by a greater decrease in total peripheral resistance in hypertensive subjects.

Similarly Rueckert *et al*. (1996) measured the hemodynamic patterns for two hours after 45 minutes of treadmill walking at an intensity of 70% HRR in Stage 1 and 2 hypertensive subjects. They showed that the acute decrease in BP was due to a significant decrease in the calf vascular resistance after the cessation of exercise. However, they reported that TPR normalized during the first hour after exercise, but that there was a significant decrease in CO in the second hour after exercise which was attributed to a fall in HR 50 minutes post exercise. The authors thus concluded that the mechanism of PEH was biphasic. Initially a drop in regional vascular resistance occurs, and later a fall in CO while TPR returns to pre-exercise values. The haemodynamics was only measured for two hours after exercise, therefore the mechanism responsible for a sustained hypotensive response is not known.

There is more evidence supporting the decrease in CO after exercise due to a decrease in stroke volume in the first 30 minutes post exercise in elderly hypertensive patients. This reduction in stroke volume might be mediated by a reduction in left ventricular end-diastolic volume. From 60 to 90 minutes after exercise, PEH seems to be the result of a continued reduction in SV and a progressive reduction in HR. A decrease in venous return or blood volume could explain the change in end-diastolic volume. It was also shown in this study that cardiac work was significantly decreased after exercise in the hypertensive group. In normotensive individuals there was no change in cardiac output or SV. Consequently

there was no change in BP after exercise. In contrast, this study showed no change in TPR in the 90 minutes after exercise (Brandão Rondon *et al*., 2002). This could possibly be the result of different mechanisms underlying the change in BP in younger and older individuals. In older patients vascular compliance is decreased, thus decreased TPR is not always possible, therefore PEH depends primarily on changes in CO (Brandão Rondon *et al*., 2002).

A decrease in TPR after exercise is most likely the result of a combination of neural and vascular changes that cause vasodilation of the skeletal muscle vascular beds. Numerous mediators of vasodilation have been investigated, but to date there is no evidence that a single mechanism is solely responsible for the decreased TPR that occurs after exercise. Two possible mechanisms that are believed to play a major part in PEH are altered sympathetic nervous system activity and altered vascular responsiveness after exercise (Pescatello *et al*., 2004).

1. Neural influences

At rest the muscle sympathetic nerve activity is controlled by the arterial baroreflex and cardiopulmonary receptor reflexes. The arterial baroreflex is the main mechanism of short term arterial BP regulation (Ichinose *et al*., 2008). This is designed to maintain arterial BP by altering peripheral vascular resistance and controlling CO via an inotropic effect and HR. During exercise, when the oxygen demand increases, the sympathetic activity increases and the baroreflex is set to operate at a higher pressure and HR (Ichinose *et al*., 2008). After exercise it is believed that there is a reduced outflow in the sympathetic vasoconstrictor nerve activity to skeletal muscle vascular beds and that the arterial and baroreflex are reset to a lower BP level than pre-exercise values (Floras *et al*., 1989). Floras *et al*. (1989) suggested that the possible mechanisms for a decreased sympathetic nerve activity to the muscle after exercise are related to: 1) suppression of the efferent sympathetic nerve activity due to prolonged increase in BP, 2) the reflex decrease in sympathetic nerve activity due to cardiopulmonary baroreceptors and 3) opioid and serotonergic systems that inhibit sympathetic outflow by central baroreceptor reflexes. Other researchers also suggested that the sympathoinhibition may be the result of the activation of the endogenous opioid receptor pathways in the central nervous system

(Halliwill, 2001; Kenney & Seals, 1993), but recent evidence has shown that this is not the case (Pescatello *et al*., 2004).

Norepinephrine is another measure of the activity of the sympathetic nervous system. Cleroux *et al*. (1992) reported decreased plasma norepinephrine levels after exercise in hypertensive subjects suggesting that a reduced sympathetic activity to skeletal muscle and skin is one of the important contributors to PEH. The mechanisms involved in the baroreflex resetting and reduced sympathetic outflow are still unknown and research findings are inconclusive (Kirkman, 2007; MacDonald, 2002; Halliwill, 2001; Kenney & Seals, 1993).

2. Vascular influences

Various vasodilatory metabolites (i.e. histamine, nitric oxide and prostaglandins) have been considered to play a role in PEH (Lockwood *et al*., 2005). At tissue level, circulating hormones and / or metabolic factors may contribute to PEH. Acute exercise causes the release of local and circulating vasodilator substances from the vascular endothelium that blunt the effect of the vasoconstrictor responses and causes vasodilation of the skeletal muscle vascular beds. It is believed that vessels are less responsive to sympathetic vasoconstrictors after exercise due to impaired α-adrenergic receptor sensitivity. It is also possible that there is an increased sensitivity of the vasodilatory receptors (Lockwood *et al*., 2005). The change in vessel responsiveness after exercise can be the result of competition of the local and circulating vasodilator substances to bind to the receptors or by modulation of the α-adrenergic pathway (MacDonald, 2002; Halliwill, 2001; Kenney & Seals, 1993).

Although numerous vasodilators have been investigated, none could be shown to independently mediate a reduction in BP after exercise. It is unlikely that one mechanism or vasodilator is solely responsible for PEH, but it could be argued that all of the vasodilators play a small part in a complex interaction of a cascade of events.

3. Other influences

There are a number of other factors that have been considered to explain the mechanisms behind PEH. Some of the contributors might not directly affect the mechanism responsible for PEH, but rather indirectly influence the sympathetic nervous system activity, central haemodynamics or local vasodilator release.

3.1 Age

Only two studies have shown that exercise affects haemodynamics differently in older hypertensive individuals compared to younger individuals. Older populations presented an increase in TPR and a decrease in CO after exercise, while younger populations show opposite relations. The decrease in cardiac output is possibly mediated by a decreased SV which could be the result of a change in cardiac contractility or a fall in venous return to the heart. No changes in pre-load or afterload were observed compared to baseline values, so the change was possibly mediated by altered cardiac contractility. Speculation is that this could be the result of the seated recovery (Brandão Rondon *et al*., 2002; Kenny & Seals, 1993). It was also shown that PEH became less prevalent with advancing age. Speculation is that this was the result of less sensitive vascular smooth muscle to catecholamine induced vasodilation and a decreased parasympathetic nervous system activity in older persons (Fleg & Lakatta, 1986). However, Brandão Rondon *et al*. (2002) have confirmed the occurrence of PEH in the elderly.

3.2 Body position

PEH has been studied under different recovery circumstances. Different body positions during the recovery period can impose different stressors on the body and orthostatic stress can affect the mechanism of PEH. Postural hypotension causes venous pooling due to a decrease in the action of the skeletal muscle pump during standing or seated recovery. During seated or standing recovery the major vascular beds are below the level of the heart and the decreased venous return can result in reflex tachycardia, increased CO and TPR. Supine recovery on the other hand might decrease TPR (Kirkman, 2007; Halliwill, 2001; Kenny & Seals, 1993). Others

speculated that the decrease in venous return would decrease SV and thus result in a greater PEH (Halliwill, 2001).

3.3 Core temperature

During exercise, metabolic heat production causes a rise in internal body temperature. The body responds by increasing vasodilation of the cutaneous blood vessels to dissipate heat. In theory, this thermoregulatory induced blood flow to the skin might contribute to a decreased systemic vascular resistance and thus a lower arterial BP (MacDonald, 2002; Rueckert *et al*., 1996; Kenney & Seals, 1993). Lockwood *et al*. (2005), however, reported that PEH is not due to cutaneous vasodilation, but rather skeletal muscle vasodilation. An increased body temperature after exercise is not sustained for a prolonged period of time and would only contribute to PEH for a short while after the cessation of exercise, until body temperature has returned to pre-exercise values. Therefore, although thermoregulation might contribute to PEH immediately after exercise, it is unlikely that it would cause a sustained decrease in BP.

3.4 Exercise modality

Although it has been reported that dynamic and resistance exercise produce similar reductions in BP after exercise (MacDonald, 2002), the hemodynamic mechanisms following resistance exercise might be different to those seen after dynamic aerobic exercise. It has been speculated that resistance exercise produces metabolites that could enhance muscle vasodilation, leading to a reduction in systemic vascular resistance and BP. Resistance exercise can also cause a decrease in plasma volume due to the mechanical compression of the muscle on the vasculature. This forces blood plasma into the interstitial spaces which can result in decreased SV and CO leading to reduced BP (Rezk *et al*., 2006; MacDonald, 2002). It was shown that resistance exercise resulted in a decreased BP after exercise due to a reduction in CO which was mediated by a reduced SV. The decreased SV was probably the result of a decreased pre-load due to the shift of plasma volume to the interstitial space. Although HR was increased after the resistance exercise, it was not large

enough to account for the reduction in SV, resulting in a reduced CO (Rezk *et al*., 2006).

3.5 Exercise intensity

Exercise intensity influences the change in haemodynamics during exercise. Higher intensity exercise results in greater rises in HR, CO and thus BP. It is therefore expected that the intensity of exercise would also influence the mechanisms responsible for PEH. Forjaz *et al*. (2004) measured the hemodynamic response in 24 year old normotensive subjects for 90 minutes after dynamic exercise at 30%, 50% and 75% of VO_{2peak}. They showed that systemic vascular resistance (SVR) and CO changes did not differ between the various exercise intensities. After exercise, the SVR decreased and CO increased in all the exercise sessions, but the hemodynamic factors for the increase in CO were different. After the no exercise control session and low intensity exercise the CO changed due to an increase in SV, while after the more intense exercise it was due to increases in HR. The hemodynamic patterns varied from one individual to another and the authors concluded that haemodynamics are not affected by exercise intensity, but rather by individual differences.

The reason for the inconsistent hemodynamic changes after a single bout of exercise cannot be explained solely by the exercise characteristics such as intensity, mode or duration of the session, because changes in both CO and TPR have been observed at the same intensity of exercise (Kenney & Seals, 1993).

4. Conclusion

The inconsistent hemodynamic changes that are reported after an acute bout of exercise remain a mystery. It is possible that the population studied might affect the changes in haemodynamics because it is not known which mechanisms are responsible for a greater PEH in persons with hypertension compared to their normotensive peers (Kenney & Seals, 1993). Most of the factors affecting BP, such as TPR, CO and sympathetic activity, cannot be measured directly. Thus, it is almost impossible to determine the exact contribution of each of the factors described above, especially during exercise. From the available literature, it can be deduced

that the physiological mechanisms underlying PEH are very complex and cannot be ascribed to a single factor. More research is warranted to determine the complex interaction of the various mechanisms resulting in altered BP after exercise.

C. MAGNITUDE OF PEH

Due to differences in study design, there is uncertainty regarding the true magnitude and duration of PEH. Researchers have examined the effect of numerous variables on PEH, i.e. different exercise intensities, duration, environmental factors and population differences, but inconclusive results have been found (Halliwill, 2001; Forjaz *et al*., 1998; Fagard, 2001; Rueckert *et al*., 1996). Some studies showed that a considerable proportion of individuals do not present with reduced BP after chronic or acute exercise. It is inconclusive whether these individuals are non-responders, or whether the absence of PEH can be attributed to different study designs, study populations, inadequate sample size or inaccurate BP measures (Lehmkuhl *et* al., 2005; Pescatello & Kulikowich, 2001; Brownley *et al*., 1996).

It seems that the most important variable that determines the magnitude of PEH is the baseline or pre-exercise BP values (Pescatello *et al*., 2004). Numerous studies have shown that PEH occurs in hypertensive as well as normotensive subjects, but greater reductions are reported in individuals with hypertension (Pescatello *et al*., 2004, Pescatello & Kulikowich, 2001; Kenney & Seals, 1993; Somers *et al*., 1991). Wilcox *et al.* (1982) have shown that individuals with hypertension showed a smaller absolute increase in SBP during exercise at a HR of 120 $b.min^{-1}$, and that the decrease in SBP after exercise was greater compared to normotensive controls. The reason for the smaller increase in SBP during exercise is unknown. It has also been shown that after the same intensity of exercise only persons with mild or borderline hypertension showed a reduction in resting BP while normotensive individuals showed no reductions in BP after exercise (Forjaz *et al*., 2004; Brandão Rondon *et al*., 2002; Brownley *et* al., 1996; Cleroux *et al*., 1992; Pescatello *et al*., 1991). In normotensive individuals the magnitude of PEH ranges from no reduction to decreases of 8 mmHg for SBP while in hypertensives, reductions of up to 20 mmHg have been reported. DBP decreases of 5 and 9 mmHg have been reported for normotensive and hypertensive subjects respectively (Forjaz *et* al., 1998; Kenney &

Seals, 1993). Studies using ambulatory or automated BP monitoring showed smaller reductions in BP after exercise than studies using manual auscultatory measures. This is probably because automated measures are more sensitive, reliable and less subjected to observer bias (Pescatello *et al*., 2004).

Gender does not seem to affect PEH. It has been reported that there are no differences in the magnitude of BP reductions between hypertensive men and women following the same acute bout of exercise (Queiroz *et al*., 2009; Ciolac *et al*., 2008; Quinn, 2000; Kenney & Seals, 1993). No studies have controlled the effect of the menstrual cycle in women in the study designs (Pescatello *et al*., 2004) it is therefore not known if the menstrual cycle would have an effect on PEH.

Bulbulian & Ebert (1994) studied the effect of personality type on the BP response after 20 minutes of dynamic exercise in young normotensive women. It is known that the aggressive, competitive and stressed characteristics of type A personalities are more prone to cardiovascular disease. It was shown that the post exercise reductions in BP were similar between Type A and B personality groups. However, SBP and DBP during exercise were lower for type B personalities suggesting that the hemodynamic responses may differ according to behavioural types.

It seems that the main predictor of the magnitude of PEH is baseline BP. Persons with a higher resting BP will show greater reductions than normotensive subjects, probably due to the protective effect of the baroreflex in normotensive subjects. The aetiology of BP in individuals might also affect the magnitude and mechanism of BP reductions after exercise. The device used to measure BP might affect the outcome of the study with studies using automated devices reporting smaller reductions. Other genetic factors such as gender and personality type do not seem to affect the magnitude of PEH.

D. DURATION OF PEH

Besides the importance of the magnitude of PEH, the duration of the reduction in BP also determines the clinical significance of the exercise response. Even if the reduction is relatively small, but is sustained during the working hours of the day, it

might have a cardio protective effect. The onset of hypotension after exercise occurs within the initial minutes to an hour after the cessation of exercise and lasts from three minutes and up to 22 hours after exercise in hypertensive subjects (Pescatello *et al*., 2004; Brownley *et al*., 1996; Fleg & Lakatta, 1986). Most studies measured BP after exercise under very controlled conditions in the laboratory for a period of 60 to 120 minutes. Data for the period after the initial 120 minutes are limited (MacDonald, 2002; Forjaz *et al*., 1998; Cleroux *et al*., 1992; MacDonald *et al*., 1999). PEH will only be clinically relevant if it is sustained for a significant period of time during free living conditions.

There are only a few studies that used ambulatory BP monitoring for nine to 24 hours after dynamic exercise to determine the duration of PEH (Ciolac *et al*., 2008; Guidry *et al*., 2006; Brandão Rondon *et al*., 2002; Quinn, 2000; Brownley *et* al., 1996; Rueckert *et al*., 1996; Somers *et al*., 1991; Pescatello *et* al., 1991). Although significant reductions in BP under free living conditions have been reported for up to 22 hours in hypertensive individuals, the results of most studies on the duration and magnitude of PEH during activities of daily living are contradictory. Only one study controlled the post exercise activities, but the auscultatory methods that were used to determine BP in this study may have provided inaccurate results (MacDonald, 2001). There is also limited evidence available on the time taken to reach pre-exercise values again, because only a few studies have continued measurements to the standardized end point where BP returns to pre-exercise values (Kenney & Seals, 1993, Brownley *et al*., 1996).

In the only study where post-exercise activities were controlled, MacDonald *et al*. (2001) measured the duration of PEH during simulated activities of daily living after an exercise bout of 30 minutes cycling at 70% of VO_{2peak} in young adults with borderline systolic hypertension. The 70 minutes post exercise consisted of controlled activities of light exercise and simulated activities of daily living. They reported a significant decrease in SBP, DBP and MAP of 16, 5 and 8 mmHg respectively after exercise. The greatest BP drop of 26 mmHg was seen during a 5 minute period of quiet standing after exercise, probably due to venous pooling. Although the BP was only measured for 70 minutes after exercise, it remained lower than pre-exercise values for the exercise group and did not show a trend towards

returning to baseline values after the 70 minutes. It is unknown what the BP response would be with continued daily tasks. The researchers concluded that moderate intensity exercise is sufficient to cause PEH that has the potential of being sustained during activities of daily living.

Brandão Rondon *et al*. (2002) measured the duration of PEH after 45 minutes of cycling at 50% VO_{2peak} in elderly hypertensive and normotensive patients. They reported that PEH was sustained for SBP, DBP and mean BP for 22 hours after exercise only in the hypertensive group during day and night time, but not for the normotensive group. Similarly Pescatello *et al*. (1991) measured the ambulatory BP after a control and exercise session (in six normotensive and six mildly hypertensive, middle aged men). Subjects were allowed to continue with their normal daily activities after the exercise or control session. They found that SBP was 6mmHg lower after exercise compared to baseline for eight hours in hypertensive participants while DBP and MAP was 9 mmHg and 8 mmHg lower respectively for 12 hours after exercise. The normotensive group showed a 5mmHg increase in SBP and no change in DBP or MAP after exercise. The results of this study could have been confounded because the sample size was very small and there might have been methodological bias, because the exercise and control sessions were not randomized.

Brownley *et al*. (1996) compared the effect of moderate aerobic exercise on the ambulatory BP of 20 normotensive and 11 hypertensive men and women (21 - 47 years). Only the hypertensive group showed a significant decrease in SBP, DBP and MAP of 5.8, 3.1 and 5.6 mmHg respectively compared to the non-exercise day. This lasted for five hours after exercise. Subjects were allowed to resume work and normal daily activities to examine if PEH was sustained during a normal working day. The researchers concluded that morning exercise only has an effect during the working hours and does not affect sleeping hours. They also examined the effect of exercise on stress levels by the profile of mood states (POMS) to determine if exercise decreases stress and therefore contribute to the lower BP. They found that the decreased BP is independent of stress and psychological state.

Most studies only measured PEH in patients that were not receiving drug treatment for high BP. It is of utmost importance to know whether pharmacologically treated patients would respond in a similar fashion. Ciolac *et al*. (2008) measured 24 hour ambulatory BP in middle-aged hypertensive subjects who had been pharmacologically treated for approximately nine years. This was the only study not to include a wash-out period before the start of exercise. It was reported that the average 24 hour SBP and DBP was 3 mmHg and 1.9 mmHg lower after the acute exercise session. Daytime DBP (1.6 mmHg), night time DBP (3.4 mmHg) and night time SBP (4 mmHg) was also significantly lower after the exercise. Although not statistically significant, daytime SBP was also 2 mmHg lower. Twenty-six of the 50 patients were still hypertensive at the commencement of the study despite the drug treatment, but after the exercise 12 more patients reached normal BP levels.

Despite the results showing a sustained PEH, not all research supports this notion. Some studies found that the hypotensive response was not sustained during daily living. Somers *et al*. (1991) examined the duration of PEH in normotensive (26 ± 3 years) and mild hypertensive (32 ± 4 years) men and women after a graded maximal exercise test. The exercise test lasted between 27 and 46 minutes. In the first hour after the exercise BP was monitored every five minutes in the supine position. Thereafter the subjects returned home and continued with normal daily activities. BP was monitored with a semi-automated sphygmomanometer three times every two hours for 12 hours. These values were compared to BP values taken at the same time of day on a non-exercise control day. There was a significant decrease in BP in the first hour after exercise in the supine position compared to control values in both the normotensive and hypertensive groups. SBP and DBP were 18 mmHg and 14 mmHg lower in the hypertensive group and 8 mmHg (both SBP and DBP) in the normotensive group. There was no sustained PEH when the participants returned home. The researchers suggested that PEH might have lasted longer if the subjects spent more time in the supine position directly after exercise as the mechanisms for PEH may have been overridden when the subjects were no longer in the supine position.

It is also possible that a graded maximal exercise protocol is not of optimal intensity and duration to elicit a PEH response. Previous studies have shown that exercise of

a longer duration and at a lower intensity is more likely to induce and sustain PEH (Pescatello *et al*., 2004). It is also possible that the manner of BP measurements could have affected the outcome of the study, because subjects had to measure their own BP at home with a semi-automated sphygmomanometer. BP readings taken at home may not be as accurate and reliable as the measurements taken in the laboratory (Somers *et al*., 1991).

Rueckert *et al*. (1996) measured the duration of PEH in 18 hypertensive, 50 year old men and women. After a 15 minute rest period, the subjects walked on a treadmill for 45 minutes at an intensity of 70% HRR. After the exercise the haemodynamics of the subjects were monitored in the laboratory for two hours in a semi-recumbent position. Thereafter the subjects returned home with an ambulatory BP monitor and continued with their normal daily activities. There was a statistically significant reduction of 14 mmHg in SBP within the first two hours after exercise. Afterwards the values returned to baseline values. MAP and TPR also decreased significantly by 7mmHg and 3.7 units respectively, but this only lasted for the first 20 minutes after exercise. These results correlate with those of Somers *et al*. (1999) who also showed that PEH is transient and will not last an entire day.

Forjaz *et al*. (2004) examined the effect of exercise intensity on PEH in young normotensive subjects. They showed that after all three exercise sessions at 30%, 50% and 75% VO_{2peak}, PEH occurred during the recovery period. The PEH was greater and lasted longer after the more intense exercise, but lasted only for 90 minutes in the controlled laboratory setting in a seated recovery position. The hypotensive effect was not observed during the 24 h monitoring period when the subjects returned home.

The reason for the inconsistencies in the duration and magnitude of PEH is unclear. Individual differences and BP monitoring devices might explain some of the differences in research findings. All of the studies implemented different methodological designs and exercise variables, that also might have influenced the magnitude and duration of PEH.

E. EXERCISE FACTORS AFFECTING THE MAGNITUDE AND DURATION OF PEH

Numerous studies have investigated the different exercise variables in an effort to find the most effective mode, intensity and duration of exercise to induce a clinically significant reduction in BP, reduce other cardiovascular risk factors, ensure compliance of the patient as well as minimize the risk for other diseases or injury. There is still no agreement on the ideal volume of exercise for patients with hypertension as well as the effect of the exercise characteristics like intensity, duration, mode and frequency on the magnitude and duration of PEH.

The American College of Sports Medicine (2006) has specific recommendations with regard to exercise prescription for persons with hypertension. Exercise must consist of dynamic activity of large muscle groups at an intensity of 40% - 70% of VO_2 reserve or HRR for 30 to 60 minutes on three to seven days per week. Resistance training is not recommended as the primary form of exercise training for hypertensive individuals, but should be combined with aerobic training and incorporated in the program on two to three days. Resistance exercise should consist of eight to 10 exercises of major muscle groups at a low resistance with high repetitions.

There are a number of special considerations that should be taken into account when working with persons with hypertension. Persons with a BP greater than 200 / 110 mmHg should not commence exercise before BP has been stabilized. Medication may alter the normal hemodynamic responses to exercise; therefore patients should be informed about signs and symptoms such as heat intolerance and ischemia. A gradual cool down period should be incorporated to prevent excessive hypotension due to medication. Exercise should be terminated if BP exceeds 220 / 105 mmHg during exercise.

1. Exercise modality

PEH has been observed after numerous types of dynamic exercise including walking, running, leg ergometry, cycling and arm ergometry. There is no conclusive evidence

regarding the most effective exercise modality that would elicit the greatest magnitude or duration PEH (MacDonald, 2002).

MacDonald *et al*. (2000) compared the effects of leg and arm exercise for one hour after exercise in nine borderline recreationally active men and women to investigate the effect of different amounts of active muscle mass on the magnitude of PEH. It has been documented that leg musculature comprises 32% of body mass while upper limb mass is 7.6%. Therefore, at the same relative exercise intensity, the muscle mass involved during lower body activities is a lot higher and will result in a greater metabolic rate. The authors speculated that if PEH was mediated by peripheral factors like local vasodilator substances, PEH would be greater following leg exercise, because the absolute production of these substances would be more with the greater muscle mass involved. Although the magnitude of PEH was not influenced by the muscle mass, it was noted that at the end of the 60 minute post exercise period, BP started returning to resting levels for the arm exercise while with the leg exercise the BP was still decreasing. The researchers proposed that the muscle mass might have an effect on the duration of PEH. Concerning the mechanism of PEH, they concluded that it was not the result of a local vasodilator and that central control mechanisms might be involved in mediating PEH.

1.1 Resistance exercise

Cornelissen & Fagard (2005) reviewed the literature on the effect of resistance training on BP and concluded that most studies found that systolic and diastolic BP was reduced after resistance exercise. Resistance exercise does not increase resting BP and is therefore not dangerous to persons with hypertension. Umpierre & Stein (2007) found that a single resistance training session can cause PEH in normotensive and hypertensive individuals irrespective of the exercise intensity. Consequently, it is suggested that resistance exercise can have similar outcomes in persons with high BP than aerobic exercise (Boroujerdi *et al*., 2009; Umpierre & Stein, 2007; Cornelissen & Fagard, 2005; Pescatello, 2004; Fisher, 2001; MacDonald *et al*., 1999), either on its own, but definitely as part a comprehensive exercise program (Meredith-Jones *et al*., 2009; Colado *et al*., 2009; Marzolini, 2008; Rezk *et al*., 2006). In fact, some authors are of the opinion that programs where resistance

exercise is incorporated, aerobic training programs lead to more successful adaptations of the cardiovascular system than with cardiovascular activity alone (Volaklis *et al*., 2007). The addition of resistance exercise may therefore enhance the effects of a dynamic exercise program (Colado *et al*., 2009).

Similar to aerobic exercise, one must consider the effect of the intensity, duration and frequency of resistance training on the overall BP response and specifically the magnitude of PEH. Of these three variables, the intensity of exercise is probably most important as one would expect higher BP responses with greater exercise intensities. When performing resistance training, the intensity of exercise is usually expressed as a percentage of the maximal weight that could be lifted in one repetition, namely the one repetition maximum (1 RM) or as a specified repetition maximum, which is the heaviest load lifted for a certain number of repetitions (Earle & Beachle, 2003).

Rezk *et al*. (2006) showed that SBP decreased after resistance exercise sessions at 40% and 80% of 1RM in a similar manner, but that only the session at 40% of 1RM decreased the DBP. This could be explained by the different haemodynamics observed after the two exercise sessions. After the 40% 1 RM session, CO was lowered due to a reduction in SV. This caused the decreases in SBP and DBP. After the 80% 1 RM session, the CO was also decreased, but systemic vascular resistance showed an increase after exercise due to the larger mechanical compression which could explain the decrease in SBP and lack of decrease in DBP. Importantly, however, was that the magnitude of the BP reduction for the resistance exercise was similar to that reported after an aerobic exercise session and lasted for at least 90 minutes under controlled conditions in the seated position. Boroujerdi *et al*. (2009) also showed significant reductions in BP following resistance training at 42.5% and 85% of 1RM. They reported that SBP and DBP showed a longer duration of PEH after more intense exercise, although the DBP returned to baseline within 30 minutes post exercise. However, they only measured the post exercise response for 60 minutes. The reason for the difference in results from Rezk *et al*. (2006) is not clear, but it might be ascribed to the fact that Boroujerdi *et al*. (2009) used normotensive body builders in their study that were experienced in weight training. The same results might not have been observed in novice, hypertensive patients.

In a study by MacDonald *et al.* (1999) PEH was compared between resistance (unilateral leg press at 65% of 1RM) and submaximal dynamic exercise (cycling at 65% of VO_{2max}). Both exercise trials lasted less than 15 minutes. There were similar and statistically significant reductions in SBP after both activities, but no change in DBP. SBP reached its lowest point at 10 minutes after exercise (20 mmHg reduction) and MAP was 7 mmHg lower than baseline values between 30 and 60 minutes post exercise. Unfortunately BP was only monitored for 60 minutes post exercise, so it is not clear how long the hypotensive effect lasted. Queiroz *et al*. (2009) also found significant reductions in BP after a single session of low intensity resistance training (3 x six resistance exercises at 50% 1RM until fatigue) in young normotensive men and women and this PEH response also lasted for 60 minutes under controlled laboratory conditions. Once the subjects left the laboratory and continued with activities of daily living, the reduction in BP was diminished.

From the above studies it is clear that resistance exercise has the potential to lower BP in a way similar to aerobic exercise. Furthermore, Cornelissen & Fagard (2005) could not find any conclusive evidence that the intensity of the exercise had a significant effect on the magnitude of the BP reduction. One aspect that needs further study is the duration of the PEH effect after resistance exercise since in all the previous studies BP was only monitored for 60 – 90 minutes post-exercise.

1.2 Dynamic aerobic exercise

Numerous studies have demonstrated that aerobic exercise of different modalities, intensities and duration is sufficient to induce BP reductions (Jones *et al*., 2007; Forjaz *et al*., 2004; Fagard, 2001; Quinn, 2000; Pescatello *et al*., 1991). What is less clear is how much exercise an individual should do to produce the greatest reduction in BP (Pescatello *et al*., 2004).

Since most studies on PEH have utilized acute aerobic exercise, the following discussion on the effect of intensity, duration and frequency of exercise on the magnitude and duration of PEH is limited to aerobic exercise.

2. Exercise intensity

Previous studies have shown that the PEH response is present after dynamic exercise of intensities that range from 30 to 100% VO_{2max}, HRmax or heart rate reserve (HRR) (Jones *et al*., 2007; Syme *et al*., 2006; Smelker *et al*., 2004; Forjaz *et al*., 2004; Fagard, 2001; Quinn *et al*., 2000; MacDonald *et al*., 1999; Forjaz *et al*., 1998; Marceau *et al*., 1993; Pescatello *et al*., 1991). PEH was also observed after maximal exercise to exhaustion (Kenney & Seals, 1993; MacDonald, 2002; Forjaz *et al*., 1998; Somers *et al*., 1991; Quinn, 2000). Most studies only measured the effect of the training session for a few hours after exercise (Jones *et al*., 2007; Smelker *et al*., 2004; MacDonald *et al*., 2001; MacDonald *et al*., 1999; Forjaz *et al*., 1998; Rueckert *et al*., 1996.)

Some studies documented that the magnitude of the PEH response is independent of the intensity of the exercise (Guidry *et al*., 2006; Forjaz *et al*., 1998). Both Pescatello & Kulikowich (2001) and Kenney & Seals (1993) reported that the initial BP as well as the patient's BP classification was more powerful predictors of the magnitude of the BP reduction after exercise. Those individuals with the highest baseline BP had the greatest decline in BP after exercise (Cleroux *et al*., 1992; Somers *et al*., 1991; Brownley *et al*., 1991; Pescatello *et al*., 1991). Other studies showed that doing aerobic exercise at a higher percentage of VO_{2max} causes a greater change in magnitude and duration of the hypotensive effect, especially during the evening and sleeping hours (Forjaz *et al*., 2004; MacDonald, 2002; Quinn, 2000).

Table 2 summarizes the different study outcomes on the effect of exercise intensity on the magnitude and duration of PEH. Physiologically one would expect a relationship between exercise intensity and the BP response. It has been shown that the sympathetic nervous system activity, norepinephrine release and the changes in the hemodynamic response during exercise are related to exercise intensity. It is possible that the intensity of exercise also has an effect on the cardiovascular changes after exercise (Forjaz *et al*., 2004; Forjaz *et al*., 1998; Rueckert *et al*., 1996; Cleroux *et al*., 1992). As we still do not completely understand the mechanism of PEH, it is unclear whether exercise intensity will affect the magnitude of PEH (Smelker *et al*., 2004).

Syme *et al*. (2006) examined whether peak SBP attained during a maximal exercise test was related to the PEH response in adult hypertensive men. SBP was monitored for 10 hours after two cycling exercise sessions at 40 (light) and 60% (moderate) VO_{2max}. The group that had an exaggerated BP response during the maximal exercise test presented with a 7.3 mmHg lower SBP after light and 5 mmHg lower SBP after moderate exercise. In men with normal peak SBP response during the maximal exercise test, there was only a lower SBP (6.3 mmHg) after the moderate exercise session. Therefore in men with a high peak SBP response, only light exercise is needed to bring about PEH, whereas in men with a normal peak SBP, moderate exercise intensity is needed to elicit PEH. The authors speculated that the light exercise intensity interacted favourably with the pathophysiology of the men with high peak SBP, because men with high peak SBP might have endothelial dysfunction. The light exercise does not induce such a great vasoconstrictor effect on the already damaged endothelium. The group with the normal peak SBP response could counteract the greater vasoconstriction because the endothelium is intact, but they needed a higher intensity exercise to facilitate a more sustained reduction in peripheral vascular resistance. It is important to provide an adequate stimulus to each specific population to control the various factors that could affect PEH, because over-exercising could lead to an increase in BP instead of hypotension.

From Table 2 it can be concluded that there is no certainty whether exercise intensity affects the magnitude of BP reductions. It seems that the intensity might have an effect on the duration of the PEH response as some studies utilizing a higher intensity showed a longer duration of PEH (Forjaz *et al*., 2004; Quinn, 2000). In a training study by Marceau *et al*. (1993), it was also shown that higher intensity exercise lowered BP during day and night time whereas lower intensity exercise only lowered BP during the day time. Other training studies support the notion that more intense exercise is less effective than moderate exercise on lowering BP and that it imposes fewer risk factors for persons with high BP (Touyz *et al*., 2004; Tsai *et al*., 2004; Fagard, 2001; Hagberg *et al*., 2000). Most of the acute exercise studies only monitored BP for two hours after exercise, and no conclusions can be drawn on the sustained BP reductions after more intense exercise.

Table 2: A summary of the effect of exercise intensity on the magnitude and duration of PEH in normotensive (N) and hypertensive (H) individuals.

Study	**Intensity** (VO_{2peak})	**Duration**	**Population** H / N	**Magnitude** **SBP**/**DBP**(mmHg)	**Duration**
Guidry *et al*. (2006)	40%	15 min	H	5.6/0	No difference in duration of PEH, all lasted up to 9h
	40%	30 min		4.3/2.4	
	60%	15 min		4.1/2.1	
	60%	30 min		4.9/3.6	
Forjaz *et al*. (2004)	30%	45 min	N	Not significant	
	50%		N	4.8/3.6	90 min for SBP and 30 min for DBP
	75%			8.4/4.4	90 min for SBP and 60 min for DBP
Smelker *et al*. (2004)	70% V_T	25 min	H	Not significant	
	80% V_T			<15	120 min
	90% V_T			< 15	120 min
	100% V_T			15	120 min
Quinn (2000)	50%	30 min	H	4/5	4 h
	75%			9/7	13/11 h
MacDonald *et al*.(1999)	50%	30 min	N	8/5	15 min for SBP and 45 min for DBP
	75%			8/5	
Forjaz *et al.* (1998)	30%	45 min	N	No significant difference in SBP or DBP between exercise intensities. All showed PEH. Average decrease 5/2.5 mmHg at 90min post exercise	
	50%				
	80%				
Rueckert *et al*. (1996)	70% HRR	45 min	H	11/4	120/20 min
Cleroux *et al*. (1992)	50%	30 min	N & H	No effect (N)	
				11/4 (H)	3h (H)
Somers *et al*. (1991)	Increment al exercise	27 - 46 minutes	N & H	18/14 (H)	1h (supine)
				8/8 (N)	Not sustained in ADL
Brownley *et al*.(1991)	60 – 70% HR_{max}	20 min	N & H	6.5/1.9 (H)	5 h (trends of reduced BP after 5 h)
				No effect (N)	
Pescatello *et al*.(1991)	40%	30 min	N & H	No reduction (N)	No difference in intensities 8.7/12.7 h
	70%			5.1/8 (H)	

VO_{2peak}, maximal aerobic capacity; mmHg, millimetres; min, minutes; h, hours; V_T, ventilatory threshold; HRR, heart rate reserve; HR_{max}, maximal heart rate

3. Exercise duration

The duration of the exercise may also influence the magnitude and the duration of PEH with the expectation that longer exercise may cause a more favourable PEH response. It is difficult to compare study results, because the study designs differ dramatically in terms of intensity, modality of exercise and BP monitoring techniques (MacDonald, 2002). Evidence shows that an exercise bout as short as three to 20 minutes is sufficient to lower BP, but that the longer duration exercise cause a greater magnitude and duration of the PEH response (Guidry *et al*. 2006).

MacDonald *et al*. (2000) conducted two studies to investigate the effect of exercise duration on the magnitude of PEH in normotensive and borderline hypertensive individuals. They tested cycle ergometry exercise at 70% VO_{2peak} for 15, 30 and 45 minutes in normotensive in the one study and for 10 and 30 minutes in hypertensive individuals in the other study. They showed that the maximal SBP reduction was 12 mmHg and 4.6 mmHg from pre-exercise values between 5 and 45 minutes post exercise in all the exercise bouts in the normotensive group. Exercise duration had no effect on the decrements in DBP in the normotensive group. The SBP was also reduced by 14 mmHg and DBP by 8 mmHg for 60 minutes post exercise in the hypertensive group. At all time points the magnitude of hypotension seems to be lower after the 30 minute trial, but it was not significantly different from the 10 minute trial. The researchers concluded that the duration of exercise had no significant effect on the magnitude of PEH in normotensive or hypertensive individuals, but no conclusion could be drawn on the duration of PEH, because they only measured BP for an hour after exercise. They speculated that the duration of exercise might cause a longer duration of PEH.

Guidry *et al*. (2006) also examined the effect of different intensities and duration of the magnitude and duration of PEH (Table 2). They showed that SBP was lowered after all the exercise sessions for most of the daytime hours, but that DBP had greater and longer lasting reductions after longer exercise bouts. They concluded that short duration exercise at a light intensity is as effective as a longer duration or higher intensity exercise to induce BP lowering effects.

The ACSM (2006) prescribes continuous or intermittent exercise in the treatment and control of high BP, but they do not conclude on the efficacy of intermittent versus continuous exercise. Most studies have measured the BP response after continuous exercise (Ciolac *et al*., 2008; MacDonald *et al*., 1999; Forjaz *et al*., 1998; Rueckert *et al*., 1996). Park *et al*., (2008; 2006) and Jones *et al*. (2009) examined the effect of intermittent exercise on the magnitude of PEH in hypertensive individuals. Park *et al*. (2006) reported that although the absolute reduction in BP after the intermittent and continuous exercise was the same, the SBP and DBP reductions lasted 11 h and 10 h respectively after the intermittent exercise (4 x 10 minute walking bouts, over four hours), whereas following continuous exercise (40 minutes of continuous walking) both SBP and DBP were reduced for 7 h. The two protocols were introduced at different times of the day (09:30 vs. 12:00) which could have influenced the outcome of the study.

Jones *et al*. (2009) showed that intermittent exercise resulted in an 8 mmHg greater decrease in MAP compared to continuous exercise at 70% VO_{2peak} in normotensive adults. The rest periods between the 10 minute exercise bouts in the intermittent protocol were spent in a seated position which might have increased venous pooling, thus contributing to PEH. A different result might have been observed if the subjects continued with activities of daily living between the exercise bouts. The researchers also showed that when conducting intermittent exercise at different times of the day, it was less influenced by diurnal variation than continuous exercise. This study had a very small sample size (n = 8), and the post exercise BP was only monitored for 20 minutes after exercise, therefore the results should be interpreted with care.
The results show that intermittent exercise is just as effective, if not more effective, than continuous exercise in reducing BP. This has implications for public health where individuals with time constraints can accumulate short bouts of exercise during the day that will be beneficial in reducing BP.

4. Total work done

Jones et al. (2007) argued that it is the total amount of work done that determines the magnitude of PEH rather than the intensity or duration of the exercise. In their study, all subjects (normotensive) performed 3 cycling exercise trials and one control trial of

seated rest for 30 minutes. The first exercise session consisted of 30 minutes of cycling at 70% VO_{2peak}, (intense), the second session was also 30 minutes at 40% VO_{2peak} (moderate) and the third session was at 40% VO_{2peak}, (long moderate), but lasted until an equivalent dose of work was completed as in the intense trial. There was no significant difference between the SBP reductions of the intense (5 mmHg) or long moderate trial (1mmHg), but both these trials had significantly greater decreases in BP than the short moderate trial (5 mmHg SBP). Only the intense and long moderate trials produced statistically significant greater BP reductions than the control session. There were no statistically significant differences between the DBP values of any of the exercise sessions compared to the control session. The results from this study showed that it is not the intensity or the duration that determines PEH, but rather the total work (dose = intensity x duration) done in a session of exercise. This shows that exercise can be prescribed at low intensity, but the duration must then be prolonged (Jones *et al*., 2007).

5. Time of day to exercise

Resting BP exhibits circadian rhythms characterized by nocturnal decreases in BP and an increase in BP in the morning when rising (Mancia *et al*., 1983, Watson *et al*., 1980). The question arises whether these changes in BP are affected by exercising at different times of the day. Jones *et al*. (2008 a; 2008b) addressed this question and measured PEH for one hour after exercise. They showed that PEH was more pronounced after exercise in the afternoon (16:00) than during morning hours (08:00). The mean BP decreased by 7 mmHg following afternoon exercise while there was a 3 mmHg increase after morning exercise (Jones *et al*., 2008b). MAP and DBP showed similar trends to mean BP. The SBP in the morning did show a hypotensive response (10 mmHg), but to a much lesser extent than seen after afternoon exercise (22 mmHg). The authors concluded that PEH is dependent on the time of day that exercise is initiated. Higher values of HR and CO were observed at baseline during the morning hours probably due to the morning surge of the circadian rhythm (Jones *et al*., 2009; 2008b). The TPR after morning exercise also increased above baseline whereas following afternoon exercise the TPR decreased and remained below baseline levels for the remainder of the measuring period. It was suggested that individuals with hypertension should not exercise in the morning

due to the risk of a rise in BP (Jones *et al*., 2008b). This study only measured BP in normotensive individuals. There was no control group to compare the BP responses in the morning and afternoon without the influence of exercise. Similar results were reported in the study by Jones *et al*. (2009) where the PEH after exercise in the afternoon was greater than in the morning. Although both exercise sessions showed decreases in SBP and MAP, the effect was larger following afternoon exercise. The BP was only monitored for 20 minutes after exercise in eight normotensive subjects.

Not all studies support the notion of a morning surge (Mancia *et al*., 1983, Watson *et al*., 1980). It is speculated that it is the behaviour of humans rather than a circadian rhythm that results in the morning surge. The only true rhythm observed has been a nocturnal fall in BP. Most studies examining the BP response after exercise did not record the time of day of the exercise, but it is highly unlikely that all of the studies would have been conducted in the afternoon. No other study has reported detrimental effects on BP caused by exercise in the morning. Therefore the results of the studies by Jones *et al*., (2009; 2008a; 2008b) should be interpreted with care. Marceau *et al*. (1993) has shown that after 10 weeks of exercise training in the morning, the morning rise in BP was blunted, thereby decreasing the risk for cardiac events. It is logical to think that if a morning surge does exist, that exercise in the morning would have a cardioprotective effect if a reduction in BP occurs after exercise. The person is therefore protected during the daytime hours when daily stressors contribute to higher BP. If PEH occurs during night time hours, following afternoon exercise, the positive effects of the exercise would be diminished by the next morning. The positive effects of the exercise would occur during sleeping hours when the BP is already lowered.

This wasted benefit of afternoon exercise is supported by Lehmkuhl *et al*. (2005) who showed that when comparing the 24 h SBP measures of a 50% VO_{2peak} exercise session to a no exercise control session, there were no differences for any of the outcome variables as a result of exercise in stage 1 hypertensive adults. The researchers speculated that the time of day for exercise (late afternoon between 17:00 and 19:00) and the nocturnal dipping status of the subjects might have been the reason for the lack of response to exercise treatment. The reduction would thus

be during the night time hours when nocturnal dipping is expected rather than during the day time hours when the daily stressors contribute to a higher BP.

6. Other

6.1 Post-exercise recovery

Different methods of recovery after exercise might also contribute to the magnitude or duration of PEH. Active recovery after exercise will maintain the functioning of the skeletal muscle pump, limiting venous pooling and therefore reduce PEH. Passive recovery on the other hand will cause venous pooling due to the increased vasodilation of the active muscles that are not matched by an increase in cardiac output during exercise, therefore increasing PEH (Halliwill, 2001).

The orthostatic position of recovery may also influence venous pooling and therefore influence the magnitude of PEH. In a seated or standing position, the major vasodilating vascular beds are below the level of the heart and therefore cause increased venous pooling in the active lower limb musculature. The orthostatic stress might therefore influence the results after an acute exercise bout. Recovery in a supine position will cause an increased venous return to the heart and will not cause such a large drop in BP (Halliwill, 2001). It has been shown that DBP will have a greater decrease if recovery takes place in the sitting position rather than in the supine position (MacDonald *et al*. 1999). It has been shown numerous times that a control session of quiet sitting or lying down is not sufficient to cause a sustained decline in BP (Forjaz *et al*., 2004; Touyz *et al*., 2004; Tsai *et al*., 2004; Quinn, 2000; MacDonald *et al*., 1999). It can thus be concluded that sitting alone is not sufficient to cause a reduction in BP, but that sitting after exercise would result in a greater PEH due to venous pooling as a result of the dilated vasculature in the active muscles and a cessation in the activity of the skeletal muscle pump.

7. Conclusion

According to Painter (2008) more research is needed to identify how regular exercise can benefit patients with chronic conditions. It is important to understand the

pathophysiology of a given condition to optimize interventions to specific physiological limitations and to implement regular exercise as an integral component of clinical treatment. If exercise is to be used as a non-pharmacological aid in the management of hypertension, more knowledge is required about the specific intensity, duration and modality of exercise to evoke optimal magnitude and duration of PEH and to decrease the risk for other diseases (Meredith-Jones *et* al., 2009; Jones *et al*., 2007).

F. METHODOLOGICAL ISSUES IN BP MONITORING

A problematic issue in most studies is the monitoring of BP. Clinical measures of BP seem to be less sensitive to detect significant changes due to terminal digit preference, observer bias and the white coat phenomenon (Pescatello & Kulikowich, 2001). It is well- known that respiration causes BP to fluctuate due to changes in intrathoracic pressure. Therefore numerous measures should be collected on different days before any diagnosis on hypertension could be made. It is known that it is more accurate to use beat by beat recordings to detect acute changes in BP, especially during and after exercise (MacDonald *et al*., 2001; MacDonald *et al*., 1999). Some researchers also failed to take the effect of diurnal variations on BP into account and only compared post exercise BP values to pre-exercise control values without controlling the time of day to initiate exercise (Jones *et al*., 2009; 2008b; MacDonald *et al*., 2001).

The ambulatory BP monitor is more sensitive and accurate in determining the magnitude and duration of PEH (Pescatello *et al*., 2004). There are some important methodological considerations when designing ambulatory BP monitoring studies. Pescatello and Kulikowich (2001) underlined some important considerations in the research of PEH, namely: a large sample size to detect the small changes in BP, a control session to compare baseline and pre-exercise BP values with post-exercise values, controlling confounding variables like medication used and the patients' compliance with the protocol. These could influence BP values and multiple BP assessments.

G. AMBULATORY BLOOD PRESSURE MONITORING

1. Background

The measurement of blood pressure is very difficult even when performed by experienced technicians. The golden standard for measuring blood pressure is via an intra-arterial catheter, but due to its invasive nature it is not routinely used. Manual or electronic non-invasive or indirect techniques to measure BP are preferred and the diagnosis and management of hypertension are based on these measurements. Clinic blood pressure does not accurately reflect a person's true BP, because of moment-to-moment variability of BP, variation in the time of day of measurement, observer bias and a phenomenon called white coat hypertension.

White coat hypertension is characterised by a change in the haemodynamics of the patient due to the situation or circumstance. This results in an elevated office BP while BP outside the office is normal. The BP reading can rise up to 23 / 18 mmHg in such a situation and has been found in 25% of patients who have been "wrongfully" diagnosed as being hypertensive (Verdecchia, 2000; O'Brien *et al*., 2000; Stanton & O'Brien, 1993). Ambulatory blood pressure (ABP) devices decrease the variability in measurements and incidence of white coat hypertension. It is thus a more accurate reflection of a person's true BP.

Automated twenty-four hour blood pressure monitoring was only developed about 30 years ago. It has grown to be a useful research and clinical tool (Stanton & O'Brien, 1993). In a clinical setting, ABP monitoring can be useful in the diagnosis of hypertension and to evaluate white coat hypertension. It can also be used to examine the efficacy of pharmacological treatment and investigate the effect of exercise treatment. It has been established that ABP monitoring is a more sensitive predictor of cardiovascular outcome than clinical BP measurement (O'Brien *et al*., 2000). Lehmkuhl *et al*. (2005) illustrated that ABP monitoring could be reproduced for both systolic and diastolic BP after non-exercise control sessions as well as after two exercise sessions at 50% VO_{2peak} in stage 1 hypertensive adults.

2. Methodological issues

Although the device for measuring 24 h blood pressure is called an ambulatory blood pressure monitor, it is not exactly ambulatory, because the device requires subjects to discontinue activity and be static for the duration of the measurement. The measures are therefore rather more intermittent than ambulatory. Achieving a good recording is dependent on the subject's acceptance of the device and compliance with the monitoring process. However, it still provides a clearer picture of a person's 24 h BP and circadian rhythms than office BP when compared to intra-arterial BP measures.

According to Wallace *et al.* (2005), ABP is more accurate, has high reproducibility, has less variability and has a higher correlation with end-organ damage than clinical BP. Furthermore, it was shown that ABP monitoring was reproducible when monitoring was initiated at the same time of day, but when it was initiated at opposite times of day, e.g. day vs. night, the measures were not reproducible. This finding seemed strange, because although BP follows a 24 h cycle, the outcome of the BP should not be affected as long as the entire 24 hours is measured. ABP monitoring may interfere with daily BP due to the production of a pressor response. Starting the measurements in the morning may contribute to the pressor response, because subjects who began their monitoring in the morning had higher blood pressure than those beginning in the evening. It is therefore important to consider design issues, such as the time of day, when monitoring ABP to ensure reproducible measures (Wallace *et al*., 2005).

3. Validation of ABP measuring devices

A number of 24 h ambulatory blood pressure measuring devices are available on the market, but they are very expensive. The manufacturers have to ensure that the device is accurate and that measures are repeatable. To decrease the reliance on producers, the monitors should be individually validated (O'Brien *et al*., 2000). A number of validation protocols have been published to ensure that independent assessments are accurate and that data from different automated devices can be compared.

The groups that have published such protocols are The Association for the Advancement of Medical Instrumentation (AAMI), the British Hypertension Society (BHS) and the European Society of Hypertension (ESH). Due to the different procedures and statistical criteria, it is recommended that all ambulatory blood pressure (ABP) measuring devices be validated by these protocols. The AAMI standard described a protocol where at least 85 participants had to be tested a minimum of three times with the mean difference not more than 5 mmHg with a standard deviation of 8 mmHg. This protocol has often been revised with the latest revision in 2002. The BHS protocol also requires 85 participants with three measurements per participant. This protocol specifies a grade for systolic and diastolic measurements on the basis of measurement errors within certain criteria. Devices with grades less than a "B" for both systolic and diastolic blood pressure are considered inaccurate and are not accepted. The devices being validated should meet the criteria and obtain high grades in both protocols before they are considered accurate (Stanton & O'Brien, 1993; Friedman *et al*., 2008).

The ESH published an international protocol in 2002 with the intention of providing a simplified protocol. This protocol requires a minimum of 33 participants with three measures from each participant. When this protocol was compared to those of the AAMI and the BHS, it lacked statistical power; it increased the risk of failure and was considered inaccurate (Friedman *et al*., 2008). In a validation study by O'Brien *et al*. (1993) it was shown that ABP monitors are more accurate in the low and mid BP ranges and less accurate in persons with a high BP (O'Brien *et al*., 1995).

H. CONCLUSION

Although there are numerous unresolved issues concerning PEH, one thing is certain. BP is affected by exercise. From the available literature it can be concluded that exercise is a valuable alternative therapy in lowering BP, especially in borderline or stage 1 hypertensive individuals. It can also aid the control in persons who have been diagnosed with hypertension and are already receiving pharmacological treatment. The positive effects of exercise cannot be denied. Exercise has a positive contribution to make towards public health care. Although exercise and a

healthy lifestyle require effort and self-discipline, research showed numerous times that it is worth a try.

CHAPTER FIVE

PROBLEM STATEMENT

A. SUMMARY OF THE LITERATURE

According to Painter (2008) more research is needed to identify how exercise can benefit patients with chronic conditions like hypertension. It has been established that both acute and long term exercise training on land decreases resting blood pressure and lowers average blood pressure for up to 22 hours after exercise (Brandão Rondon *et al*., 2002; Pescatello *et al*., 2001). Although exercise prescriptions for persons with hypertension are well established, there are still several unanswered questions. One of the questions is whether aquatic exercise could be an alternative exercise modality with similar health benefits to traditional land exercise.

It has been believed in the past that water exercise is only suitable for persons with a low initial work capacity and those who cannot participate in traditional fitness activities. Although it is true that water exercise is the preferred mode of exercise in persons with a high body fat percentage or with lower extremity injuries, water aerobics has been shown to meet the intensity guidelines set by the American College of Sports Medicine (2006) to produce health benefits such as improved cardiovascular and muscular endurance and a reduced risk for developing premature chronic disease (D'Acquisto *et al*., 2001; Eckerson & Anderson, 1992; Whitley & Schoene, 1987). Very little research has been done on the effects of water exercise on PEH as well as the long term effects of aquatic training in the control and management of hypertension. There is evidence in the available literature that water exercise is a good alternative to land exercise in the maintenance of cardiovascular fitness, muscular integrity and flexibility (Meredith-Jones *et al*., 2009; Barbosa *et al*., 2007). One of the main beneficial effects of water exercise is the decreased load on the joints of the lower body, especially in persons with lower extremity injuries, persons with a low exercise capacity, older as well as obese persons. Persons with chronic disease and who have been sedentary and overweight for an extended

period of time often have to start an exercise program. Thus water exercise should be ideal because of the lower metabolic demand at the same relative HR and the decreased compressive forces on the joints, reducing the risk for possible injury and over exertion. Water exercise is therefore a good alternative to initiate regular exercise in sedentary persons before one move on to more intense traditional land exercise.

Water immersion causes a change in the cardiovascular regulation when compared to land exercise. The hydrostatic pressure on the body during water immersion results in a redistribution of blood away from the extremities to the abdominal and thoracic cavities (Park *et al*., 1999). This increased thoracic blood volume results in increased central venous pressure leading to an increased pre-load. According to the Frank-Starling mechanism, this would result in a rise in stroke volume and therefore an augmented cardiac output (Cider *et al*., 2005; Park *et al*., 1999). Cardiac output significantly affects blood pressure because:

Blood pressure = Cardiac output x Total peripheral resistance

For this reason, water-based activities should influence the BP response to exercise. The extent of the change in BP response is unknown because the available literature reports equivocal results on this matter. BP has been shown to increase (Park *et al*., 1999), decrease (Šrámek *et al*., 2000; Darby & Yaekle, 2000) or to show no change (Cider *et al*., 2005; Hall *et al*., 1998; Fujisawa *et al*., 1995) when the subject is immersed in thermo-neutral water compared to when on land. It has been reported that water immersion causes decreased plasma concentrations of rennin, angiotensin II, aldosterone, norepinephrine and epinephrine, all of which regulate and affect BP (Cider *et al*., 2005). There is no evidence that water exercise is detrimental to the BP response, but there are no reports on the effect of aquatic exercise on BP in the 24 hours following exercise during free living conditions.

Ambulatory blood pressure monitoring is a popular tool to assess BP during day and night under free living conditions. Compared to clinical measures, ambulatory blood pressure monitoring has been shown to be more accurate, it has less variability and it minimizes the occurrence of the white coat phenomenon (Wallace *et al*., 2005;

O'Brien *et al*., 2000; Stanton & O'Brien, 1993). Most studies have used clinical measures for a few hours after exercise to measure the magnitude and duration of PEH (MacDonald, 2002; MacDonald *et al*., 1999; Forjaz *et al*., 1998; Cleroux *et al*., 1992). In these studies the participants were required to be seated or lay supine, while ABP monitoring allows the person to continue with activities of daily living. This ensures a more valid indication of the true magnitude and duration of blood pressure changes. Only a few studies have been conducted to determine the magnitude and /or duration of PEH with the use of ambulatory BP monitoring (Ciolac *et al*., 2008; Guidry *et al*., 2006; Quinn, 2000; Brownley *et* al., 1996; Reukert *et al*., 1996; Pescatello *et* al., 1991; Somers *et al*., 1991).

B. PROBLEMS WITH RESEARCH IN BLOOD PRESSURE

Due to differences in study design and other methodological issues, there is still no agreement on the clinical significance of an acute session of exercise in the management of hypertension. There is also no research on the benefits of water exercise compared to land exercise in the management of hypertension. Furthermore, there is no consensus on the mode, intensity, duration or modality of exercise to produce optimal reductions in blood pressure after exercise. If exercise is to be used as a non-pharmacological aid in the management of hypertension, it is essential to determine if the hypotensive effects of exercise are sustained under free living conditions and whether prolonged exercise attenuates or enhances this effect.

C. THE OBJECTIVE OF THE CURRENT STUDY

The primary aim of the study is to examine the magnitude and duration of post exercise hypotension during free living conditions in persons with mild to moderate hypertension after an acute exercise session.

Specific aims

1. To determine the magnitude and duration of post exercise hypotension during 24 hours of free living conditions after a session of land-based and water-based exercise.

2. To compare the effects of land-based and water-based exercise on the blood pressure response to exercise in persons with hypertension.

CHAPTER SIX

METHODOLOGY

A. STUDY DESIGN

This study used a controlled crossover, experimental design to determine the effect of water- and land-based exercise on the magnitude and duration of post exercise hypotension. Participants performed, in random order, one land-based and one water-based exercise session as well as a control session in which no exercise was involved. Outcome variables were measured after both exercise and control sessions.

B. SUBJECTS

Twenty-one men and women aged between 33 and 69 years volunteered to participate in the study. Subjects acted as their own control. Three sessions were performed with each participant, namely the control session, the land exercise and the water exercise. Participants were unaware of the order of the sessions until the commencement of the session.

Recruitment

Subjects were recruited through advertisements in the local press and through references from medical practitioners in the Stellenbosch and Somerset West areas. Forty-two subjects volunteered to participate in the study. All subjects underwent a screening procedure to identify those who met the inclusion criteria. The subjects were screened for co-morbidities to minimize external influences on the measured outcome variables and possible risks to participants. The co-morbidities were screened by means of cholesterol and glucose tests, anthropometry measures as well as a health questionnaire. Persons with more than two risk factors, according to the ACSM guidelines (ACSM, 2006), were asked to obtain medical clearance from their physicians before being allowed to participate in the study (Appendix A).

Of all the volunteers, 16 subjects could not be included in the study, because they did not comply with the inclusion criteria. A further three subjects were eliminated from the study due to an abnormal exercise response or an ST segment depression of more that 2mm during the exercise ECG and were referred for further evaluation by a cardiologist. One subject withdrew because of an orthopaedic work injury and another withdrew due to time constraints. Twenty-one participants (men and woman), with borderline hypertension (n = 2), stage I (n = 9) and stage 2 hypertension (n = 10) completed the study. The five participants that were taking anti-hypertensive medication had to obtain permission from their primary physicians to discontinue the use of their medication for at least two weeks prior to the testing. A two week wash-out period was provided for these patients where their blood pressure was monitored regularly before participation in the study began.

Inclusion and exclusion criteria

Participants were asymptomatic men and women between the ages of 33 and 69 years with known or suspected hypertension (> 135 / 85 mmHg). Subjects had no current musculo-skeletal, metabolic, endocrine, cardiovascular or end organ diseases. They were recreationally active, non-smokers and were not using medication that could affect blood pressure. Subjects were excluded if they had a BMI greater than 45 $kg.m^{-2}$, resting BP greater than 180 / 130 mmHg, took medication that could affect BP or had more than three risk factors according to the ACSM guideline for exercise testing and prescription (2006) (Appendix A).

1. Assumptions

In this study it was assumed that borderline hypertensive patients stage I and stage 2 hypertensive patients responded in a similar manner to exercise. It was assumed that men and woman of different ethnic groups responded in the same way to exercise. It was also assumed that participants were honest in reporting daily activities and the use of caffeine, tobacco and other medication in the activity diary and questionnaire.

2. Delimitations

Activity and stress levels of the participants may have differed during the day which may have influenced resting blood pressure. The normal daily activities and events of the participants could not be precisely controlled. The amount of sleep during the night may also have differed between participants. The discomfort of the BP halter might also have influenced the blood pressure and sleeping patterns of the participant. To minimize these delimitations, participants acted as their own control.

3. Limitations

Strength training can be very effective if the participants are familiar with the correct techniques. Mastering the correct techniques takes time. In this study participants underwent a familiarization session with the equipment and they were given clear instructions and demonstrations during the exercise session. Efforts were made to optimize the value of the land-based exercise.

C. EXPERIMENTAL DESIGN

1. Laboratory visits

The participants were required to visit the laboratory on a number of different occasions. The following is a summary of the separate visits:

Visit 1

The study protocol and aims of the study were explained to the volunteers. A health questionnaire (Appendix C) was completed and standardized screening tests were performed. The latter consisted of the following tests: glucose test, cholesterol test, waist circumference, hip circumference, body mass index, fat percentage, resting heart rate and blood pressure to ensure that all participants were borderline hypertensive (> 130 / 85 mmHg) or hypertensive (> 140 / 90 mmHg). Baseline blood pressure readings were determined from the average of three measurements taken in the seated position. Body composition was determined via Bio-electrical

Impedance Analysis (BIA). The subjects that passed the screening tests and complied with the inclusion criteria were invited to participate in the study.

Visit 2

The study protocol was again explained verbally and in writing. If the participants were still willing to participate, an informed consent form was explained to them and signed by the participants (Appendix D). If required after the initial screening session, the participants had to provide proof of medical clearance for exercise. Each participant's VO_{2peak} was determined on a treadmill using a Bruce protocol while a twelve lead ECG was performed. Afterwards the 10 repetition maximum (RM) muscle strength test was conducted for two muscle groups. This session also served as the familiarization opportunity to ensure correct technique when using strength training equipment. After the sessions subjects were fitted with the ambulatory blood pressure monitor to serve as a familiarization period with the monitor.

Visit 3, 5 and 7

The participants were asked to return to the laboratory within seven days after visit two. The participants were randomly assigned to performing a control session, a land-based exercise session or a water-based exercise session on the three days. The ambulatory blood pressure monitor was fitted and worn for 24 hours after each session. The three sessions were completed within 14 days.

Visit 4, 6 and 8

The participants were asked to return to the laboratory 24 hours after the control and exercise sessions to remove the blood pressure monitor.

2. Place of study

All laboratory tests were completed in the Exercise Physiology Laboratory at the Department of Sport Science at Stellenbosch University. This laboratory is fully equipped with all the apparatus needed for the project. The exercise sessions were

conducted at the Stellenbosch Biokinetics Center at the Department of Sport Science. Qualified medical doctors were on site for the duration of the testing and the exercise sessions. All tests were done at temperatures between 18 and 20°C.

After obtaining a resting ECG, subjects were cleared for exercise by the medical doctor. During and after the exercise ECG the risk profile of the patient was discussed with the doctor and a decision was taken whether the person was allowed to continue with the exercise sessions. Recommendations were made to decrease the cardiovascular disease factors for each patient during the exercise as well as specific lifestyle modifications that would be implemented.

3. Ethical aspects

The study protocol was approved by the Ethics Committee of Research Subcommittee A at the University of Stellenbosch (Reference number 126/2008). During the first and second visit the study protocol and informed consent form was explained to each subject. Participants were given the opportunity to read through the form and ask questions. The study did not involve any invasive procedures and subjects were informed that their participation was completely voluntary and that they could withdraw from the study at any time.

D. MEASUREMENTS AND TESTS

All participants completed a body composition screening, resting blood pressure reading, a maximal graded exercise test to exhaustion and maximal strength tests before participating in the exercise sessions.

1. Anthropometric measurements

Anthropometric measurements included stature, body mass, waist circumference, hip circumference and Bio-electrical Impedance Analysis (BIA) to assess percentage body fat. This ensured that the study population was homogenous in terms of fat percentage, BMI and hence cardiovascular risk factors.

Body Mass

Body mass was determined with a calibrated electronic scale (UWE BW – 150 freeweight, 1997 model, Brisbane, Australia) and recorded to the nearest 0.1 kilogram (kg). Subjects were asked to stand in the middle of the scale, distributing weight evenly on both legs and looking straight ahead. Subjects were barefoot and clothed in light-weight clothing.

Stature

Stature was measured with a sliding steel anthropometer (Siber-Hegner GPM, Switzerland). Measurements were taken to the nearest 0.1 centimetre (cm). The subjects stood barefoot with heels together and upper back, buttocks and heels against the wall. The head was placed in the Frankfurt plane. The Frankfurt plane is achieved by positioning the lower edge of the eye socket (Orbitale) in the same horizontal plane as the notch just above the tragus of the ear (Tragion). The measurement was taken from the inferior aspect of the feet to the vertex of the skull (the highest point on the skull). The height and weight were used to determine body mass index (BMI).

Circumferences

These were measured with the subject's body in the anatomical position according to the ISAK (International Standards for Advancement of Kinanthropometry) protocol (Marfell-Jones *et al*., 2006). Circumferences were measured with a spring-loaded, non-extensible anthropometric tape measure (*Rosscraft*, Canada). The measurements were taken to the nearest millimetre (mm) while the subject was standing upright with legs slightly apart and weight distributed evenly. The cross-hand technique was used for all measurements with the zero mark located more lateral than medial on the subject. The tape was held at right angles to the body segment and tension in the tape was held constant while minimizing gaps between the tape and the skin as well as minimizing indentations in the skin.

Waist circumference

The subjects were standing relaxed with the arms folded across the chest. The tape was placed around the waist at the narrowest point between the lower costal border and the top of the iliac crest, perpendicular to the long axis of the trunk. If there was no obvious narrowing in the waist, the measurement was taken at the midpoint between the lower costal border and the iliac crest. The subject breathed normally and the measurement was taken at the end of a normal expiration.

Hip circumference

The subjects were in a relaxed standing position with the arms folded across the chest, the feet together and the gluteal muscles relaxed. The measurement was taken at the level of the greatest posterior protuberance, perpendicular to the long axis of the trunk.

Bio-electrical Impedance Analysis (BIA)

The subject's lean and fat mass were measured with a portable body composition monitor (Bodystat 4.05® Quadscan 2007, Isle of Man). Lean mass comprises the bony skeleton, muscle mass, innards and entire water content of the body, while fat mass consists of adipose tissue. The BIA procedure involves sending a very small electric current through the body (800 µA at 50 kHz). This small electric current measures the resistance of the tissue. Lean tissue will provide less resistance than adipose tissue, because of its greater composition of water and electrolytes.

The participants lay quietly in a supine position with the arms 30° from the body with the limbs not touching each other or the center of the body. After cleaning the sites with an alcohol swab, two electrodes were placed on the dorsal side of the hand, one centimetre proximal to the knuckle of the middle finger and on the wrist between the head of the ulna and radius. The other two electrodes were placed on the dorsal side of the bare right foot between the lateral and medial malleoli as well as at the base of the toes between the hallux and the third phalange. The cables were then attached to the electrodes and the analyzer was switched on. The basic principle of the

method is that lean tissue conduct the electrical current due to the high contents of electrolyte containing water yielding a low impedance (resistance), whereas fat acts as an insulator and results in a high impedance to the high frequency current. The impedance measures reflect the degree of resistance to the flow of current in the body. Regression equations are derived from the impedance to determine fat free mass and total body water. The procedure took less than five minutes. Subjects were asked to void their bladders and to refrain from exercise and drinking diuretics like caffeine or alcohol for at least four hours before the tests.

2. Blood pressure and heart rate

Resting blood pressure and the subject's heart rate were measured after a five minute rest period. The average of three measurements was taken with an automated ambulatory air bladder-containing cuff (Ergoline Ergoscan 2008, Germany). All blood pressure measures were taken with the same automated device. This device measures BP via the oscillometric method and met the validation criteria of the International Protocol for Validation of Blood Pressure Measuring Devices in adults issued by the European Society of Hypertension (Langewitz & Tanner, 2009). Measurement duration is 30 – 45 seconds and deflation rate is between 3 to 5 mmHg per second. The device inflated to ± 160 mmHg for the first measurement and thereafter 25 mmHg above the last reading. The measurement range for systole was 60 – 260 mmHg, for diastole 40 – 220 mmHg and 35 – 240 $b.min^{-1}$ for heart rate.

The subjects were rested for five minutes in an upright position. Any clothing obstructing or occluding the circulation above the cuff site was removed. The cuff was placed on the non-dominant arm, 2.5 centimeters (cm) above the antecubital space, with the palm facing up and the air bladder directly over the brachial artery. Subjects had to sit quietly without speaking or moving while the recorder was started and measurement took place.

After a short rest period the blood pressure and heart rate were taken again in the same way as described above. If the two measures for either the systolic or diastolic blood pressures differed by more than five mmHg, a third measurement was taken to

determine the average of the three measures. If there was not a greater than five mmHg difference in the first two readings, the average of the first two readings was recorded as the resting blood pressure and heart rate.

3. Blood markers

Random (i.e. not necessarily fasting) blood glucose and cholesterol were measured at least two hours after the last meal. Blood glucose and cholesterol were measured as part of the screening procedure to determine the relative risk of cardiovascular disease in the subjects and did not serve a diagnostic purpose. Random blood glucose of 200 $mg.dl^{-1}$ or greater is recognized as a diagnostic criterion for diabetes. Although it is recognized that random blood glucose measures is not as accurate as fasting blood glucose or an oral glucose intolerance test, it is still useful as a screening tool for diabetes to encourage further diagnostic tests if necessary (Saudek *et al*., 2008). Similarly non-fasting total cholesterol test results can be used as a screening tool to provide information and guide decisions on primary health care (Craig *et al*., 2000). Nordestgaard *et al*. (2007) also reported that non-fasting lipid profiles can accurately predict the risk for atherosclerosis.

A resting blood sample was collected by means of a finger prick with a lancet device (Softclix ®, Boehringer Mannheim). Before the blood sample was collected, the subject was required to warm up his or her hands. The fingertip to be used was cleaned with an alcohol swab prior to the finger prick. On no account was the subject's finger compressed or squeezed to obtain blood, to prevent damaging blood cells and leakage of interstitial fluid into the blood sample. The first droplet of blood was wiped with a cotton swab to prevent contamination from the alcohol. The second and third blood droplet was used for the glucose and cholesterol measures respectively. A cotton swab was applied to the punctured finger to stop any additional bleeding.

Blood glucose: Blood glucose was measured with an automated blood glucose analyzer (HemoCue® Glucose 201^{+}, Ängelholm Sweden, 2001) and recorded in millimol per litre ($mmol.L^{-1}$). The HemoCue blood glucose analyzer has a SD of 0.3

mmol.L^{-1} and has been reported to be reliable and easy to use (Høi-Hansen *et al.*, 2004; Voss & Cembrowski, 1993).

Blood cholesterol: Cholesterol was measured with an automated total cholesterol analyzer (multiCare, Arezzo Italy, 2006). Total cholesterol was measured in mmol.L^{-1}. If the measure was above the critical accepted level, the subject was referred to a physician for a full blood lipid profile.

4. Maximum exercise capacity test

The results of this test were used to determine the exercise intensities for the aerobic training session. A progressive incremental exercise test to exhaustion was performed on the h/p/cosmos Saturn treadmill (Nussdorf-Traunstein, Germany), to determine maximal aerobic capacity (VO_{2peak}). The Saturn treadmill is interfaced with specialized computer software (*Cosmed Quark b^2* 2000. Italy). By using breath by breath analysis together with a telemetric heart rate monitor (POLAR®, Polar Electro Oy, Finland), the Quark b^2 software calculates and records exercise intensity and selected cardiorespiratory parameters continuously throughout each test. Oxygen saturation was also measured via a finger clamp connected to the Quark b^2 software during the exercise test. All exercise tests were performed under physician supervision.

The subjects were fitted on the treadmill with an adjustable safety harness. After the subjects warmed up for a minute at 2.7 km.h^{-1} and no incline, the face mask was fitted. The Bruce protocol was used to assess maximal effort. The test started at a speed of 2.7 km.h^{-1} and a gradient of 10%. At three minute intervals the incline and speed of the treadmill was increased until the subject reached exhaustion. Participants were also connected to a 12 lead electrocardiograph (ECG) (Mortara Instrument 2006, Milwaukee, USA), for the duration of the exercise test. This was to monitor cardiovascular changes during exercise and it enabled the physician to closely monitor the subjects and stop the exercise test if necessary.

The exercise test was terminated if any of the ACSM test termination criteria (Appendix B) were visible. Otherwise the test was terminated when the subject

reached exhaustion which was verified if the VO_2 did not increase by more than 150 ml per successive workload, a respiratory quotient (R) value equal or above 1.15 was reached, heart rate was more than 90% of the age-predicted maximal heart rate and the rating of perceived exertion (RPE) was above 19 on the 6 – 20 Borg scale. At the termination of the exercise test blood pressure was recorded with the automated device.

Throughout the test breath by breath gases were continuously recorded. Expired gas was sampled through the turbine flow meter and gas sampling line and analyzed by a cardio-pulmonary metabolic system (Cosmed *Quark* b^2, Rome. Italy). The gas analyzers were calibrated with atmospheric gas and known gas concentrations (16% O_2, 4% CO_2 balance N_2) and the turbine flow meter was calibrated with a three litre calibration syringe. Heart rate was measured through telemetry (POLAR®, Polar Electro Oy, Finland) interfaced with the metabolic system. Oxygen saturation (SpO_2) was also measured by the metabolic system for the duration of the exercise test. If the oxygen saturation dropped below 85%, the test was also terminated.

5. Muscle strength test

The purpose of the muscle strength test was to determine the intensity of the resistance exercise training session. A 10 repetition maximum test (10 RM test) was performed to determine the maximal muscle strength for each individual. A one repetition maximum test is not advised for untrained, inexperienced persons with a risk for cardiovascular disease. The subjects performed two exercises, one for upper body and one for lower body muscle groups. The exercises performed were the incline leg press and machine bench press. The subjects were informed about the test and familiarized with the equipment to ensure proper technique and to avoid the Valsalva manoeuvre and therefore a rise in blood pressure.

The subjects were instructed to warm up with five repetitions with light resistance before each exercise to ensure proper technique. After the warm up, a rest period of one minute was allowed. An initial load was estimated that allowed the subjects to complete three to five repetitions. The load was increased by five to 10% for the upper body exercise and 10% to 20% for the lower body exercise after the

completion of each stage. A two minute rest period was allowed after each set. During each set the participants were asked to estimate if he or she would be able to complete 10 repetitions. If so, the set was terminated and the weights increased until only ten or fewer repetitions could be completed. The maximum weight and number of repetitions were noted and used to calculate the person's one repetition max (1 RM). Blood pressure was monitored continuously during and directly after the strength testing with the same automated device. All testing was conducted by a qualified biokineticist.

6. Exercise sessions

All subjects completed two acute exercise sessions, namely a land-based exercise session and a water-based exercise session. All subjects also completed a control session of relaxed sitting. The order of the exercise and control sessions was randomly selected. The subjects were unaware of which session, exercise or control, they would be doing until the commencement of the session.

The water aerobics exercise

All participants performed an acute water aerobics exercise session consisting of a single exercise routine in a deep indoor pool of 25 m x 12.5 m. The temperature of the pool was regulated at 27 ℃ and the depth at the shallowest point in the pool was 2.1 m. The exercise session, presented by a qualified water aerobics instructor, consisted of aerobic exercises in the pool. The session lasted 50 minutes which included a warm-up and cool-down period. Heart rate was continuously recorded throughout the session with a Polar heart rate monitor (POLAR®, Polar Electro Oy, Finland). After the session the average and maximum heart rate during the session were noted. One person did not complete the water exercise session due to a fear of water and an inability to swim.

The combined aerobic and resistance land-based exercise

All participants completed a land-based exercise session which consisted of combined aerobic and resistance exercises. The aerobic part of the exercise session

consisted of 30 minutes of interrupted aerobic exercise between 60 and 80% VO_{2peak}, (RPE of 12 – 16). Exercise intensity was determined from the maximal exercise capacity test performed prior to the exercise session. The aerobic exercise consisted of 10 minutes of treadmill walking, 10 minutes of skiing on an elliptical trainer and 10 minutes on a stationary bike. Resistance and speed was adjusted to keep the HR in the prescribed zone.

The resistance part of the exercise session was done after the aerobic exercise and lasted about 25 minutes at an intensity of 65% of 1 RM (RPE of 12 – 16). The intensity of the exercises was determined from the results of the 10RM test done prior to the exercise session. The intensity of the rest of the exercises was obtained from prediction equations by the NCSA's Essentials of Personal Training (Earle & Baechle, 2003). The resistance program consisted of the following exercises: incline leg press, seated leg curl, leg extension, bench press, shoulder press, latissimus dorsi pulldown, seated row, triceps extension and bicep curl. Two sets of 10 repetitions were done for each exercise with a rest period of 30 seconds between each set and 90 seconds between each exercise. This program constituted a typical exercise session for hypertensive patients in a biokinetics practice.

After the exercise session participants performed an active session of recovery by means of stretches of the hamstrings, quadriceps and gluteal muscles as well as the lower back, upper back and chest. The session lasted 55 minutes which included a warm-up and cool-down period. Heart rate was continuously recorded throughout the session with a Polar heart rate monitor (POLAR®, Polar Electro Oy, Finland). After the session the average and maximum heart rates during the session were noted.

The control session

The control session consisted of 55 minutes of supervised, relaxed sitting in the laboratory where heart rate and blood pressure were monitored continuously.

7. Post exercise ambulatory blood pressure monitoring

After both exercise sessions and the control session, the participants were fitted with the ambulatory blood pressure monitor (Ergoline, Ergoscan 2008, Germany). Blood pressure monitoring was started immediately after the exercise or control session and lasted for 24 hours. Participants were given verbal and written instructions on how to operate the blood pressure monitor. The primary researcher's cell phone number was provided in case of emergency or questions regarding the monitor. The monitor was programmed to measure BP every 20 minutes during day time hours and every 90 minutes during night time hours. The recorder was set to measure day time hours between 06:00 and 22:00 and night time hours from 22:00 until 06:00.

The participants were also instructed to complete a daily activity diary for the 24 hours after exercise and control sessions (Appendix E). In the daily activity diary participants had to document what they ingested during the day as well as the activity where the majority of the time was spent during the morning, afternoon and evening. Time awake, bed time and wake-up times were also recorded. They were also instructed to document any unusual events during the day and to manually take a BP reading during that time. The participants were asked to keep daily activities as normal as possible on the three days of testing. After 24 hours the participants returned the BP monitors to the laboratory where the monitors were removed and the data downloaded onto a computer. The activity diary was also returned after 24 hours. Individual blood pressure measures were analyzed for missing or erroneous readings. Readings were smoothed if SBP was lower than DBP, SBP was >210mmHg or < 50mmHg, DBP was >140mmHg or <40 mmHg, if SBP deviated ± 40mmHg, DBP deviated ± 20 mmHg or HR deviated ± 30b.min^{-1}.

E. STATISTICAL ANALYSIS

Statistical analysis was performed with Microsoft Office Excel (Windows Vista 2007) and STATISTICA 8.0 (Statsoft, Inc. 2008, USA). Descriptive statistics are reported as means ($\bar{x}$) and standard deviation (±SD) unless otherwise specified. Repeated measures of analysis of variance (ANOVA) using a mixed model approach was performed to determine the magnitude and duration of PEH. The main group

(treatment) effect and time effect were independent factors with the interaction effect (time*treatment) as the dependant factor. Fischer's Least Significant Difference (LSD) Post Hoc tests were performed to identify significant differences among variables. The level of significance was set at $P < 0.05$ for all analyses.

CHAPTER SEVEN

RESULTS

A. DESCRIPTIVE CHARACTERSTICS

1. Subjects

The participants in this study were adults between the ages of 33 and 69 years, pre-hypertensive or hypertensive and recreationally active. The physical characteristics of the participants are summarized in Table 3. Seven participants discontinued the use of anti-hypertensive medication at least two weeks prior to the study. The other participants either never used anti-hypertensive medication or discontinued use at least 12 months before the study. There were some expected statistically significant differences in anthropometric characteristics between men and women, i.e. height, weight, lean body mass, body water and waist-to-hip ratio was greater for men than women and body fat percentage was greater for women ($P < 0.05$).

Most subjects had sedentary occupations where they spent most of their time sitting down during the day. Two subjects were retired, spending their day at home. Five persons reported standing and walking during working hours while none reported doing physical task during the work day. Participants were instructed to keep daily activities as similar as possible between different measurement days.

Table 4 depicts the outcomes of the screening tests of the men and women. Not all subjects had confirmed hypertension, but were included in the study if the average of two BP measures were above 130 mmHg (SBP) or 85 mmHg (DBP) after a rest period of five minutes on two separate occasions. There were no statistically significant differences between the resting SBP or DBP of men and women ($P > 0.05$) therefore the BP results for the exercise sessions were pooled. Resting HR of the men was 11.8 $b.min^{-1}$ lower than women at rest and the men reached 9.4 $ml.kg^{-1}.min^{-1}$ higher VO_{2peak} values ($P < 0.05$) during the maximal aerobic treadmill test.

Table 3: Physical characteristics of participants

	Men (n = 11)			Women (n = 10)		
Characteristic	**Mean**	**±**	**SD**	**Mean**	**±**	**SD**
Age (years)	50.1	±	10.58	53.6	±	9.85
Height (cm)	180.2	±	4.86*	163.8	±	6.30
Weight (kg)	96.6	±	11.09*	77.2	±	14.72
BMI ($kg.m^{-1}$)	29.1	±	3.27	28.9	±	6.15
Body Fat (%)	24.8	±	3.94#	38.9	±	6.74
Lean body mass (%)	75.2	±	3.94*	61.1	±	6.74
Body Water (L)	51.5	±	2.93*	35.5	±	3.80
Waist circumference (cm)	97.5	±	6.84	86.7	±	12.04
Hip circumference (cm)	107.4	±	4.98	108.1	±	11.66
Waist to hip ratio	0.9	±	0.05*	0.8	±	0.04

cm, centimetre; kg, kilogram; BMI, body mass index; $kg.m^{-2}$, kilogram per square meter; %, percentage; L, litre;

* Significantly greater than women, $P < 0.05$

Significantly less than women, $P < 0.05$

Table 4: Physiological characteristics of participants

	Men (n = 11)			Women (n = 10)		
Characteristic	**Mean**	**±**	**SD**	**Mean**	**±**	**SD**
Glucose ($mmol.L^{-1}$)	5.5	±	0.71	5.7	±	0.64
Cholesterol ($mmol.L^{-1}$)	4.5	±	0.96	4.0	±	1.32
Baseline heart rate ($b.min^{-1}$)	64.6	±	9.64	76.4	±	12.36
Resting Systolic BP (mmHg)	152.2	±	13.51	145.1	±	12.06
Resting Diastolic BP (mmHg)	102.5	±	12.35	94.1	±	10.69
HR_{max} ($b.min^{-1}$)	157.5	±	22.27	149.6	±	18.23
VO_{2peak} ($ml.kg^{-1}.min^{-1}$)	33.4	±	7.10*	24.0	±	4.69

$mmol.L^{-1}$, millimol per litre; $b.min^{-1}$, beats per minute; mmHg, millimetres mercury; HR, heart rate; VO_{2peak}, maximum aerobic capacity,

* Significantly greater than women, $P < 0.05$.

The maximal strength test results for men and women are presented in Table 5. The men obtained significantly greater strength scores on the maximal strength test for upper and lower body compared to the women ($P < 0.05$). The strength tests were conducted to estimate the resistance load during the resistance exercise program.

Table 5: Maximal strength characteristics of participants

	Men (n = 11)			Women (n = 10)		
Characteristic	**Mean**	**±**	**SD**	**Mean**	**±**	**SD**
1RM Leg press (kg)	238.2	±	57.76*	101.5	±	61.56
1RM Bench press (kg)	44.6	±	11.99*	14.4	±	8.26

RM, repetition maximum; kg, kilogram

* Significantly greater than women, $P < 0.05$.

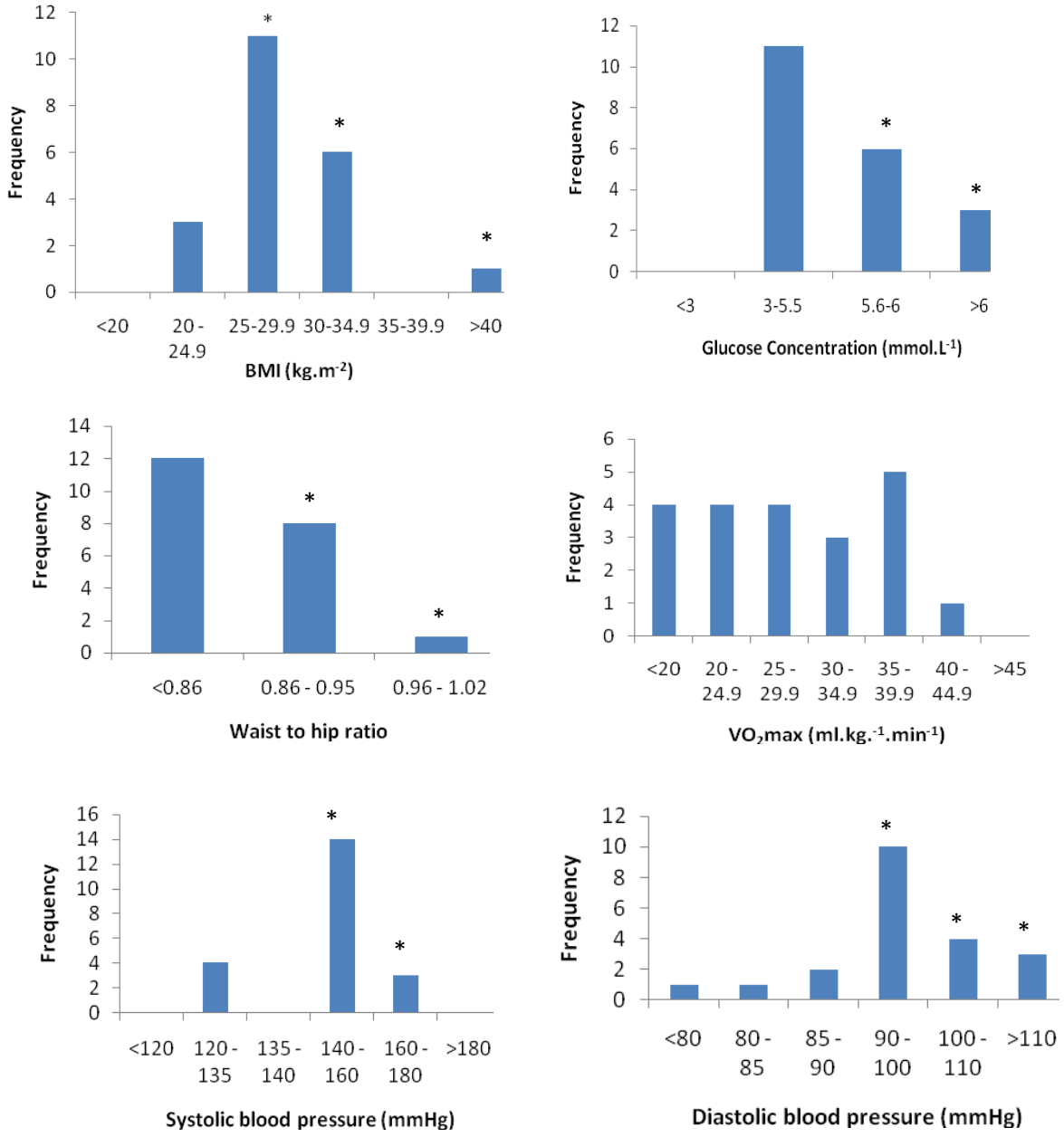
2. Classification of subjects

Figure 1 summarizes the risk profiles of the participants as determined during the risk screening session. The frequency histograms give an indication of relative health risks according to the classification of the American College of Sports Medicine (2006). In the classification for BMI, 11 subjects were classified as overweight (> 25 $kg.m^{-2}$), six as obese (> 30 $kg.m^{-2}$) and one as obese class 2 (> 40 $kg.m^{-2}$). Six subjects had glucose concentrations above 5.6 $mmol.L^{-1}$ and three persons had glucose concentrations above 6.0 $mmol.L^{-1}$. In the classification of waist-to-hip ratio, eight participants had a greater than 0.86 ratio and only one had a greater than 0.96 ratio. All the participants with elevated waist-to-hip ratio were men. Four participants displayed an aerobic capacity of less than 20 $ml.kg^{-1}.min^{-1}$ which is classified as a very low aerobic capacity. Based on SBP, 14 participants were classified as stage 1 hypertensive and three as stage 2 hypertensive. Based on DBP, 10 subjects presented stage 1 hypertension, four with stage 2 and 3 with stage 3 hypertension.

3. Exercise intervention

All subjects performed three experimental sessions. The control session consisted of 55 minutes of seated rest. The two exercise sessions (land-based and water-based exercise) were performed at an intensity between 70 – 85% of VO_{2peak}. The exercise sessions lasted between 45 and 60 minutes. All subjects completed the control session and land-based exercise session. One participant did not complete the water exercise session due to a fear of water. The average heart rate of the participants during exercise was 118 (± SD 11.3) $b.min^{-1}$ during land exercise and 119 (± SD 17.4) $b.min^{-1}$ during water exercise. There was no statistical difference

between the average heart rates achieved by men and women during land or water exercise.



* Above accepted normal values

Figure 1: Classification of participants according to BMI, glucose concentration, waist-to-hip ratio norms, maximal aerobic capacity as well as resting SBP and DBP.

B. OUTCOME VARIABLES

1. Twenty four hour BP and HR

Ambulatory SBP, DBP and HR were measured over 24 hours after the control session, the land-based exercise and the water-based exercise. Mean arterial pressure (MAP) was calculated from the measured SBP and DBP as follows: 1/3 (pulse pressure) + DBP, where pulse pressure is SBP – DBP ..... (7.1)

From the cessation of exercise until 22:00, was considered as daytime. Night time was from 22:00 until 06:00 the next morning. The next day, day 2, comprised the waking hours of the next day taken from 06:00 until the BP monitor was removed at the laboratory. On the graphs, time 0 hours indicates the end of the exercise session and the start of the 24 hour BP monitoring. Daytime is thus displayed as just after exercise until 15 - 16 hours after exercise and night time between 16h after exercise until 22h after exercise. Day 2 is indicated from 22h until 24h on the graph. The start and end of day and night time are indicated with arrows on the graph.

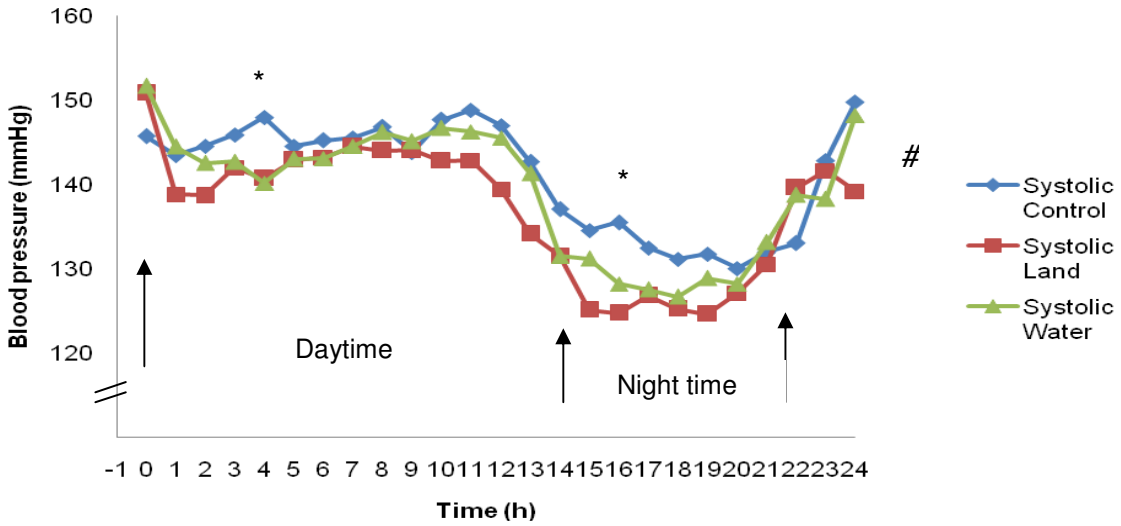
The statistical analysis showed that there were no significant differences between the BP responses in men and women (gender effect). The response to land and water exercise between men and women were not significantly different. Therefore the results were pooled for men and women in all the variables.

2. Systolic blood pressure

2.1 Changes in SBP over 24 hours

There was a significant time effect for SBP in all the groups over the 24 hour period ($P = 0.000001$). This can be explained by the diurnal variation, where night time SBP is lower than daytime SBP, as seen in Figure 2. There was no significant difference in SBP values at baseline for the three treatment groups (145 ± SD 13.49, 150 ± SD 16.59, 152 ± SD 12.42 mmHg for the control, land and water treatments; $P > 0.05$). The average SBP over 24 hours, including baseline, was 141.0 ± SD 6.6, 137.3 ± SD 7.7 and 138.8 ± SD 7.6 mmHg for the control, land and water exercise groups

respectively. Overall, the land exercise treatment resulted in a 3.6 mmHg lower average over 24 hours than the control treatment ($P = 0.04$). The average difference between the control and water treatment was 2.2 mmHg and for the land and water treatments it was 1.5 mmHg. These differences failed to reach statistical significance ($P > 0.05$).



Significant change over time, $P < 0.05$

* Significantly different between exercise groups compared to control group, $P < 0.05$

Figure 2: Average hourly ambulatory systolic blood pressure (SBP) after rest, land and water exercise.

Figure 2 illustrates that there was an acute drop in SBP during the first two hours after exercise in the land exercise treatment group. In the first hour after exercise, there was a 5.9 mmHg difference between land and control (land < control, $P = 0.09$) and a 0.3 mmHg difference between the water and control (water < control, $P > 0.05$). In the second hour after exercise the difference between the land and control was still 5.9 mmHg ($P = 0.06$), while the difference between water and control increased to 2.4 mmHg ($P > 0.05$).

At 3 – 4 hours after exercise, both the land and water exercise treatment groups had significantly lower BP (7.4 and 8.1 mmHg, respectively) than the control group ($P = 0.01$). For the rest of the daytime hours there was no significant difference between the SBP of the exercise groups compared to the control treatment at each time point, although the values for the control session were higher throughout the 24 hour

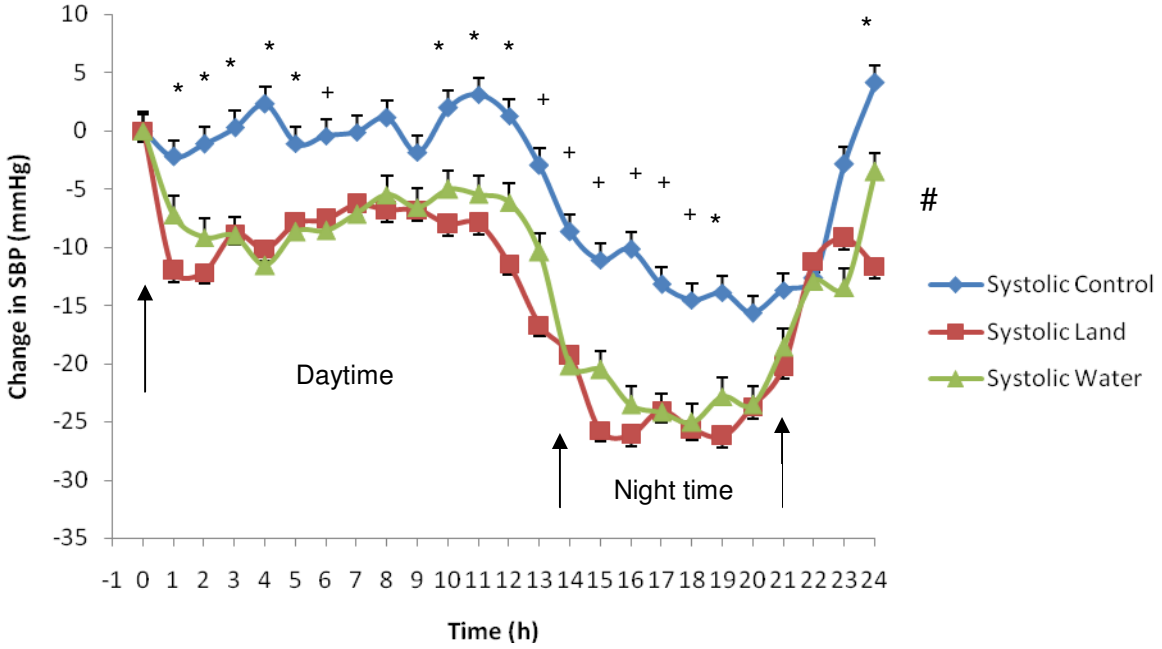
period. There was a trend that the SBP of only the land exercise treatment was lower than the control treatment for most of the night time hours. Between 15 and 16 hours after exercise, the SBP of both the land (12.1 mmHg, $P = 0.001$) and water (8.3 mmHg, $P = 0.04$) exercise treatment groups were significantly lower than the control treatment group. There was no significant difference between SBP values of the water exercise and land exercise treatments at any of the time points ($P > 0.05$).

Figure 3 illustrates the marginal difference between SBP values at each time point and the baseline value. There was a significant treatment effect in the average decrease in SBP from baseline (P = 0.005). The average decrease over 24 hours compared to baseline was 4.7 ± SD 6.65, 14.4 ± SD 7.34 and 12.9 ± SD 7.30 mmHg for the control, land exercise and water exercise treatments respectively. The decrease over 24 hours was significantly greater following the land and water exercise groups compared to the control session ($P = 0.002$ and $P = 0.009$ respectively). There was no significant difference in the average decrease between the land and water exercise treatments ($P > 0.05$).

The greatest reduction in SBP compared to baseline during the daytime following the land exercise was 12.7 mmHg. This occurred within the first two hours after exercise. The maximum reduction during night time was 26.6 mmHg. SBP was significantly decreased from baseline for the entire 24 hour period following land exercise ($P < 0.05$), except between 9 – 10 hours after exercise. The land-based exercise differed significantly from the control session from just after exercise until six hours after exercise and again from 9 – 19 hours and 23 – 24 hours after exercise. This time x treatment interaction effect showed a trend towards statistical significance ($P = 0.09$). The control treatment had a maximum reduction of 2.3 mmHg ($P > 0.05$) during daytime hours. During night time hours, the maximum reduction was 16.7 for the control group ($P = 0.00001$).

The greatest reduction after the water exercise during the day time was 11.3 mmHg. It occurred three to four hours after exercise. SBP stayed significantly lower for nine hours after exercise and was again significantly lower between 12 and 23 hours after exercise. During the night time hours the maximum reduction was 25.1 mmHg for the water exercise treatment. The water exercise resulted in significantly lower SBP

values than control treatment from one hour after exercise until four hours after exercise and again at 5 – 6 hours and 12 – 18 hours after exercise ($P < 0.05$). There was no significant difference in the decrease from baseline between the land and water treatments at any of the time points ($P > 0.05$).



* Significantly different between land exercise and control groups, $P < 0.05$

\+ Significantly different between both exercise groups and control group, $P < 0.05$

\# Significant average decrease over 24 h, $P < 0.05$

Figure 3: Absolute changes in SBP from baseline over 24 h. Time point 0 indicates the baseline value (resting), therefore the change in value from baseline equals zero. Time point 1 is the values within the first hour after exercise, starting immediately after exercise. It indicates the difference between the values within one hour after exercise and the resting baseline value.

2.2 SBP responses during day and night time

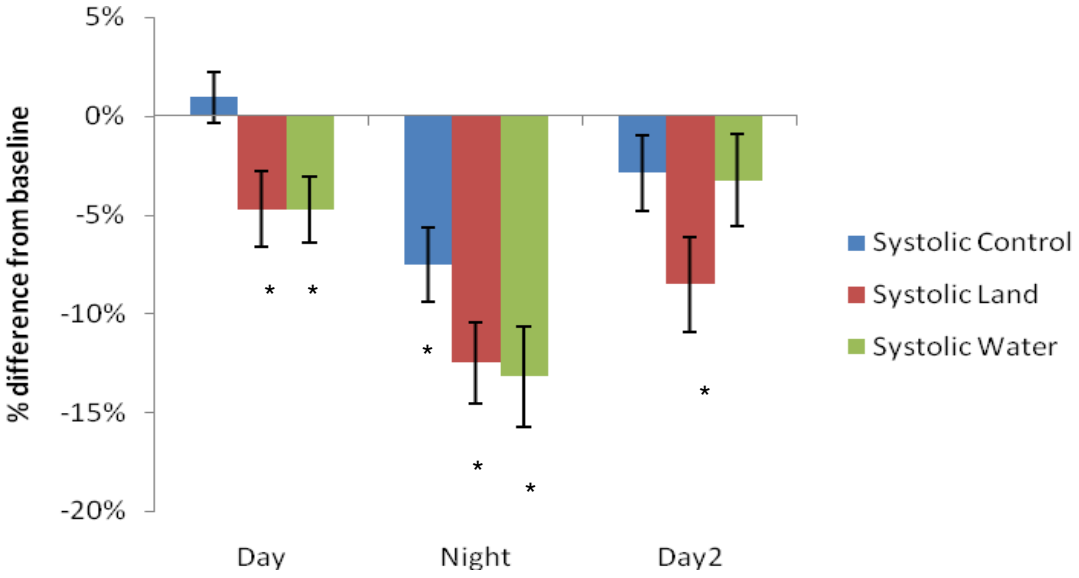
Table 6 depicts the average resting, daytime, night time and day 2 SBP. A significant time x treatment interaction was observed for the daytime and night time responses ($P = 0.01$). There was an increase of 1.4 ± SD 7.7 mmHg during the daytime after the control session ($P > 0.05$) while the two exercise groups responded with significantly lower values during daytime (land: 7.2 ± SD 13.08 mmHg, $P = 0.02$; water: 7.1 ± SD 11.63 mmHg, $P = 0.02$) (Figure 4).

All three groups showed significant decreases during night time hours compared to baseline (control: 10.9 ± SD 12.22 mmHg, $P = 0.0004$; land: 18.9 ± SD 15.21 mmHg, $P = 0.000001$; water: 20.0 ± SD 16.75 mmHg, $P = 0.00001$). Compared to baseline, the SBP at day 2 was 4.9 ± SD 12.21 mmHg lower for the control session, 12.9 ± SD 17.15 mmHg lower for the land exercise session and 4.1 ± SD 15.51 mmHg lower for the water exercise session. These differences were not statistically significant for the control or water exercise groups ($P > 0.05$), but were significantly lower at day 2 compared to baseline for the land exercise group ($P = 0.0004$).

Table 6: Average SBP after rest, land and water exercise for daytime, night time and next day hours.

	Control			Land			Water		
	Mean	**±**	**SD**	**Mean**	**±**	**SD**	**Mean**	**±**	**SD**
Rest	145.1	±	13.49	150.8	±	16.59	152.1	±	12.42
Day	146.5	±	11.65	143.6	±	10.36#	144.9	±	10.56#
Night	134.2	±	15.73#	131.9	±	11.69#	132.1	±	14.89#
Day2	141.0	±	13.77	137.9	±	11.07#	147.2	±	17.95

Significantly lower than baseline, $P < 0.05$



* Significantly different from baseline, $P < 0.05$

Figure 4: Percentage change in SBP during daytime, night time and the next day after rest, land and water exercise.

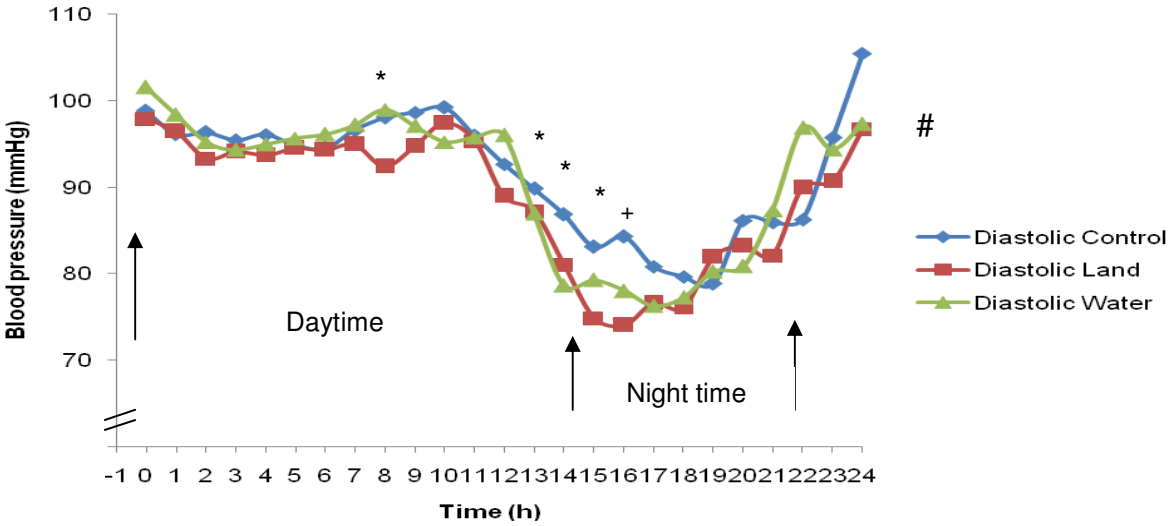
3. Diastolic blood pressure

3.1 Changes in DBP over 24 hours

Figure 5 illustrates a similar pattern for the change in DBP following all three treatments. There was, however, a significant time effect ($P < 0.000001$) for all three groups due to the nocturnal fall in DBP. There was no significant difference between the baseline DBP values of the three treatments (98.7 ± SD 13.51, 97.6 ± SD 12.94, and 101.6 ± SD 11.92 mmHg for the control, land and water treatments, $P > 0.05$).

The average DBP over 24 hours, including baseline, was 91.5 ± SD 7.12, 88.1 ± SD 7.80 and 90.1± SD 8.33 mmHg for the control, land and water exercise treatments respectively. Overall, the land exercise treatment resulted in a 2.7 mmHg lower average over 24 hours than the control treatment ($P = 0.04$). The average difference over 24 hours between the control session and the water exercise treatment was 1.3 mmHg and failed to reach significance ($P > 0.05$). Similarly there was no significant

difference in the average 24 h DBP of the land and water exercise treatment (1.3 mmHg, $P > 0.05$).



Significant change over time, $P < 0.05$

* Significantly different between land exercise and control group, $P < 0.05$

+ Significantly different between both exercise groups and control group, $P < 0.05$

Figure 5: Average hourly ambulatory diastolic blood pressure (DBP) after rest, land and water exercise.

Very few significant differences were observed between the control session and the exercise treatments at any specific time point. The only significant differences occurred between 7 – 8 hours after exercise where the DBP of the land exercise treatment was significantly lower than the control (5.2 mmHg) and the water exercise (5.4 mmHg) treatments (both, $P = 0.05$). For the rest of the daytime hours there was no significant difference between the DBP of the exercise groups compared to the control treatment.

During night time there was a statistically significant difference between the land exercise treatment and the control treatment between 12 and 16 hours after exercise (land < control, $P < 0.05$). Between 15 – 16 hours after exercise, the water exercise treatment also resulted in a significantly lower DBP than the control group ($P = 0.03$). There was no significant difference between DBP values of the water exercise and land exercise treatments during night time ($P > 0.05$).

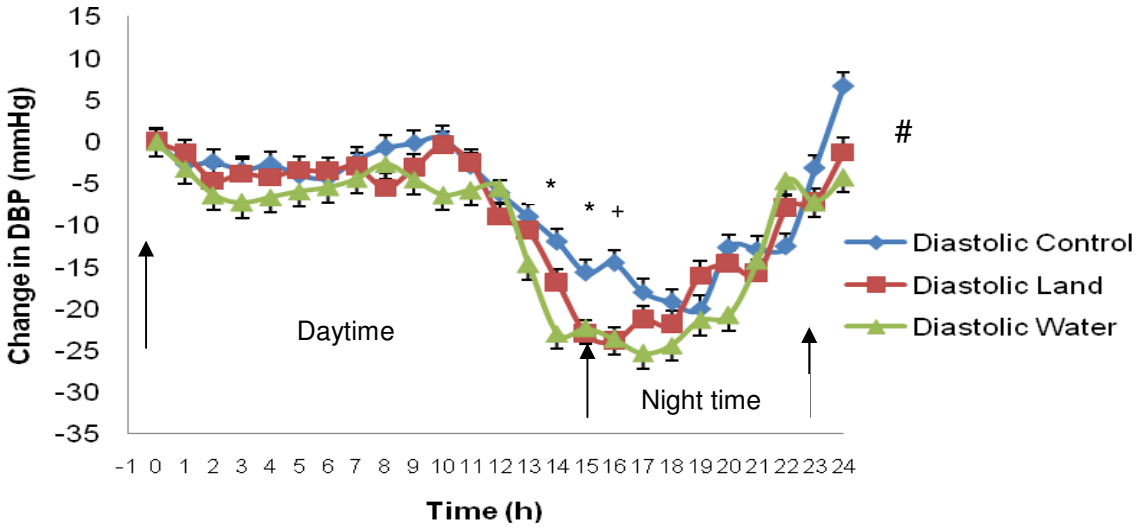
Figure 6 illustrates the difference between DBP and baseline at each time point over 24 hours. There was no significant treatment effect for DBP ($P > 0.05$) denoting that the average change in DBP over 24 hours did not differ significantly for the three groups. The average decrease over 24 hours compared to baseline was 7.1 ± SD 7.12, 9.0 ± SD 7.73 and 10.2 ± SD 8.19 mmHg for the control, land and water treatments respectively. Although the decrease over 24 hours following both land and water exercise was greater than the control session, it failed to reach significance ($P > 0.05$). There was also no significant difference in the average decrease from baseline between the land and water exercise treatments ($P > 0.05$).

The greatest reduction in the control group DBP was 5.7 mmHg during the daytime hours between 11 and 12 hours after exercise ($P = 0.04$) while maximal night time reduction was 19.4 mmHg ($P = 0.000001$). The decrease from baseline following the control session was only significant between 11 and 22 hours after exercise.

The greatest reduction in DBP after the land exercise compared to baseline during the daytime was 6.6 mmHg, occurring between 11 – 12 hours ($P = 0.01$). From one to four hours after exercise there was a strong trend towards significantly lower DBP values compared to baseline ($P = 0.07$). At 7 – 8 hours after exercise and again from 12 – 23 hours after exercise DBP was significantly reduced compared to baseline ($P < 0.05$). The maximal reduction during night time was 25.4 mmHg ($P = 0.000001$). Although the overall time x treatment interaction was not significant ($P > 0.05$), there were significantly lower DBP values at 15 – 16 hours after exercise compared to the control group ($P = 0.002$).

Following the water exercise, the maximal reduction occurred at two to three hours after exercise and was recorded at 6.9 mmHg ($P < 0.009$). During daytime, DBP was significantly reduced from baseline between one and four hours after exercise and again between 8 and 23 h after exercise ($P < 0.05$). During night time hours the greatest reduction following the water exercise treatment was 24.3 mmHg ($P = 0.000001$). The differences between the water and control treatment almost reached significance between 12 and 13 hours after exercise ($P = 0.05$) and was significantly greater than the control treatment between 13 and 16 hours after exercise ($P < 0.05$).

There was no significant difference in the reduction from baseline between the land and water exercise treatments at any time point ($P > 0.05$).



* Significantly different between water exercise and control group, $P < 0.05$

+ Significantly different between both exercise and control groups, $P < 0.05$

Significant change over time, $P < 0.05$

Figure 6: Absolute changes in DBP from baseline over 24 h. Time point 0 indicates the baseline value (resting), therefore the change in value form baseline equals zero. Time point 1 is the values within the first hour after exercise, starting immediately after exercise. It indicates the difference between the values within one hour after exercise and the resting baseline value.

3.2 DBP responses during day and night time

Table 7 summarizes the average values for the three treatment groups during rest, daytime, night time and day 2. There was no significant time x treatment interaction for the day or night time responses ($P > 0.05$). There was no significant decrease in the average daytime DBP when compared to baseline for the control or land treatments (control: 1.8 ± SD 8.64 mmHg, land: 1.9 ± SD 8.20 mmHg, $P > 0.05$). The water treatment, however, showed a strong trend towards significance with a 4.7 ± SD 9.29 mmHg decrease during daytime compared to baseline ($P = 0.08$).

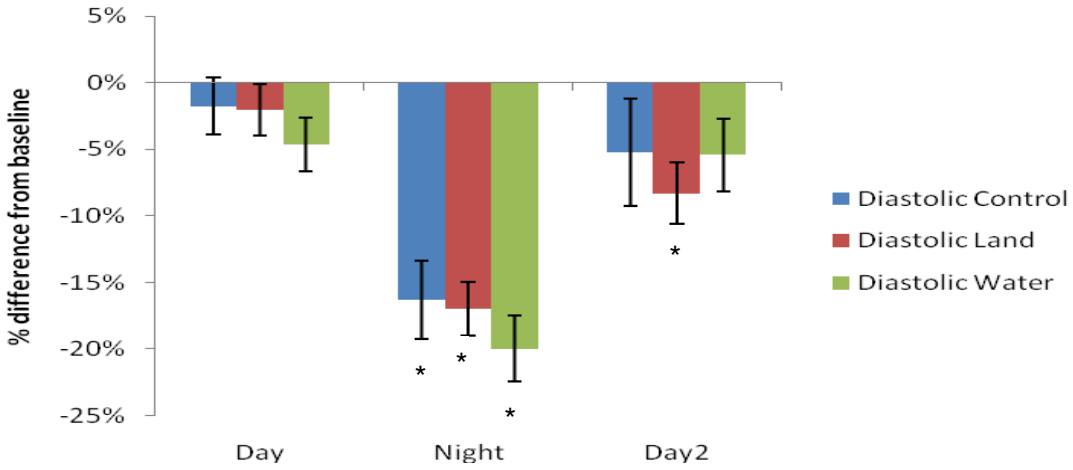
Figure 7 indicates that all three treatment groups showed significant reductions in DBP during night time hours compared to baseline (control: 16.1 ± SD 13.16 mmHg;

land: 16.5 ± SD 10.33 mmHg; water: 20.2 ± SD 12.20 mmHg, P = 0.000001). Compared to baseline, the DBP on day 2 was 5.2 ± SD 16.51 mmHg lower for the control session, 8.1 ± SD 10.76 mmHg lower for the land exercise session and 5.5 ± SD 11.99 mmHg lower after the water exercise session. These differences were not statistically significant for the control or water exercise groups, but were significantly lower at day 2 compared to baseline for the land exercise group (P = 0.01).

Table 7: Average DBP after rest, land and water exercise for daytime, night-time and next day hours.

	Control			Land			Water		
	Mean	**±**	**SD**	**Mean**	**±**	**SD**	**Mean**	**±**	**SD**
Rest	98.7	±	13.51	97.6	±	12.94	101.6	±	11.92
Day	96.9	±	9.22	95.7	±	9.24	96.9	±	9.61
Night	82.6	±	11.58#	81.1	±	10.17#	81.4	±	11.56#
Day2	93.5	±	14.63	89.5	±	10.57#*	96.1	±	12.39

Significantly lower than baseline, P < 0.05 * Significantly lower than water treatment, P < 0.05.



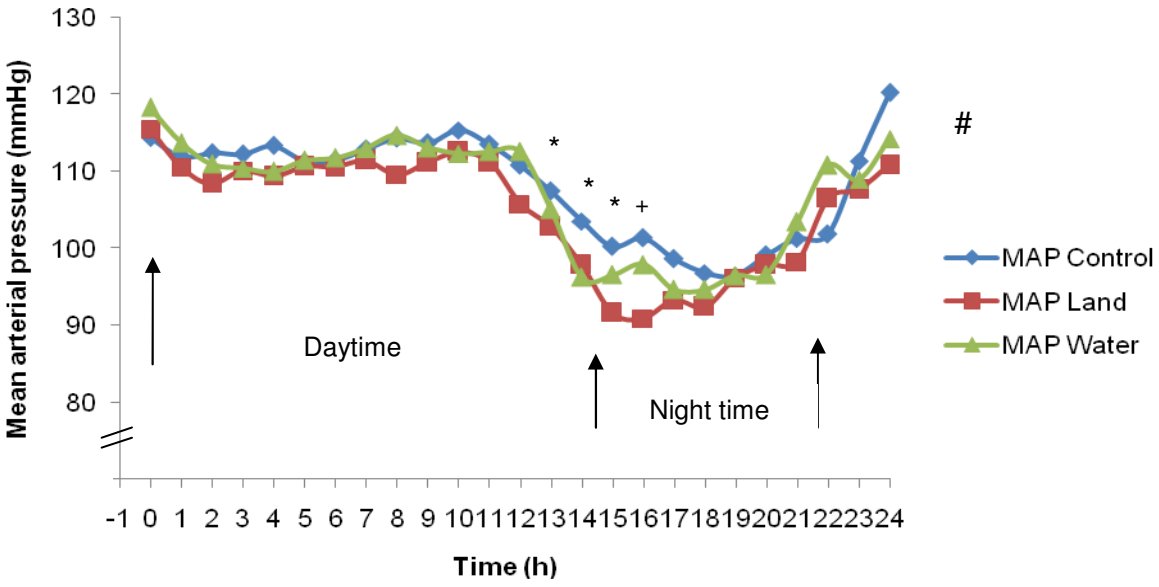
* Significantly different from baseline, P < 0.05

Figure 7: Percentage changes in DBP during daytime, night time and the next day after rest, land and water exercise.

4. Mean arterial pressure

4.1 Changes in MAP over 24 hours

There was a significant time effect ($P < 0.000001$), indicating a diurnal variation in MAP in all three groups (Figure 8). There was no significant difference between the MAP values at baseline for the three groups (114.2 ± SD 13.09, 114.6 ± SD 13.71, 118.5 ± SD 10.88 mmHg; $P > 0.05$). The average MAP over 24 hours, including baseline values for the control, land and water exercise treatments, was 107.9 ± SD 6.86, 104.9 ± SD 7.65 and 106.5 ± SD 7.63 mmHg, respectively. Although this treatment effect was not significant, it showed strong trends towards significance ($P = 0.09$). Overall, the land exercise resulted in a 3.0 mmHg lower average MAP over 24 hours than the control treatment ($P = 0.03$). The average difference between the water exercise treatment and the control session was 1.4 mmHg ($P = 0.30$). Between the land and water treatments it was 1.6 mmHg ($P > 0.05$).



Significant change over time, $P < 0.05$

* Significantly different between land exercise and control group, $P < 0.05$

* Significantly different between land and water treatments, $P < 0.05$

Figure 8: Average hourly ambulatory mean arterial pressure (MAP) after rest, land and water exercise.

Figure 8 illustrates that the MAP values during daytime were not significantly different between the treatment groups. However, during night time there was a statistically significant difference between the MAP of land-based exercise and control treatment between 12 and 16 hours after exercise (land < control; $P < 0.05$). There was also a statistically significant difference in the MAP between the land and water treatments at 15 – 16 hours after exercise (land < water; $P = 0.04$).

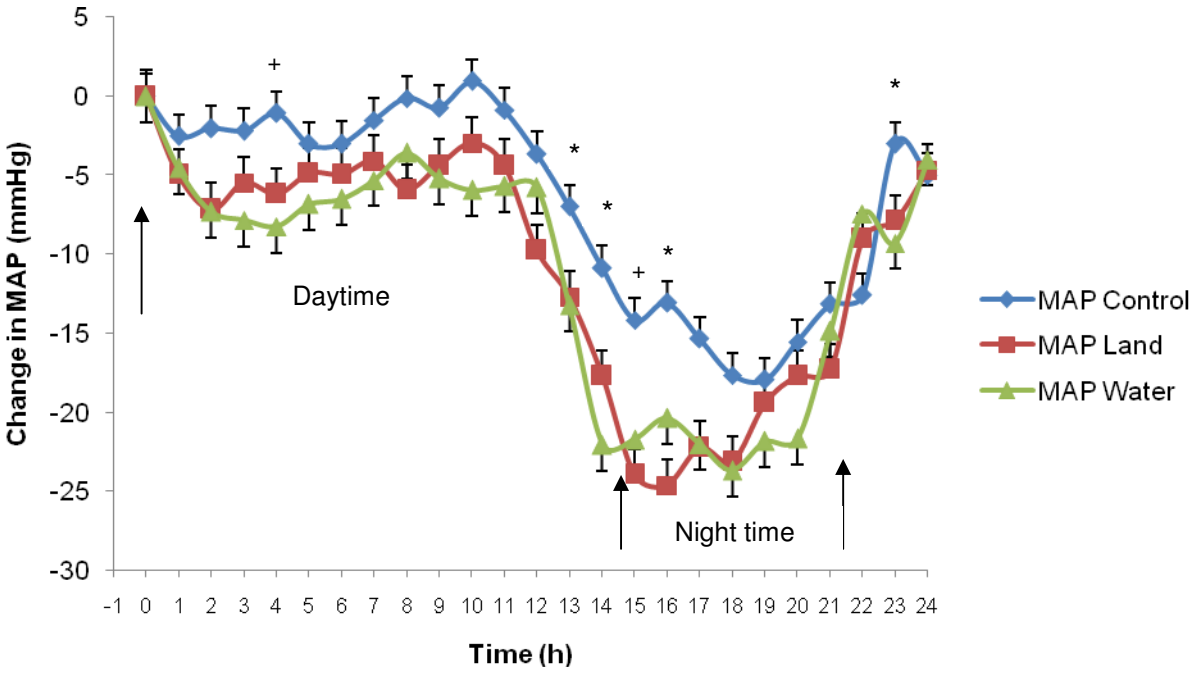
Figure 9 shows the marginal difference between MAP at each time point and the baseline value. The treatment effect for the decrease in MAP showed a strong trend towards significance ($P = 0.06$). The average decrease in MAP over 24 hours for the control, land and water exercise treatments were 6.3 ± SD 6.36, 10.4 ± SD 7.49 and 10.6 ± SD 7.32 mmHg. The decrease over 24 hours was significantly greater following the land and water exercise groups compared to the control session ($P = 0.04$). There was no significant difference in the average decrease between the land and water exercise treatments ($P > 0.05$).

The greatest reduction during daytime in the control treatment was 3.5 mmHg and occurred at 11 - 12 hours after exercise ($P > 0.05$). The greatest reduction during night time was 18.1 mmHg ($P = 0.000001$).

MAP was significantly reduced from baseline from just after exercise until 8 hours after exercise. It was again significantly reduced compared to baseline from 11 h after exercise until 23 hours after exercise. The maximum reduction in MAP following the land treatment during daytime was 7.7 mmHg and occurred between 11 and 12 hours after exercise ($P = 0.003$). The greatest reduction during night time was 25.6 mmHg ($P = 0.000001$). There was a strong tendency towards significantly greater reductions in the land exercise compared to the control session at one to two ($P = 0.07$) three to four hours ($P = 0.07$), seven to eight hours ($P = 0.07$), 14 hours to 15 hours ($P = 0.06$) and 16 hours to 17 hours ($P = 0.07$) after exercise. Between 12 and 14, 15 - 16 and 23 – 24 hours after exercise the land exercise caused significantly greater reductions in MAP compared to the control session ($P < 0.05$).

The maximum reduction following the water exercise treatment was 7.3 mmHg and occurred within three to four hours after exercise ($P = 0.004$). MAP was significantly

reduced compared to baseline from one hour after exercise for the whole 24 hours, except between 7 – 8 hours and 23 - 24 hours after exercise. During night time, the maximum reduction was 23.5 mmHg (P = 0.000001). The water exercise treatment resulted in significantly greater reductions in MAP than the control treatment between three to four hours and between 12 and 16 hours after exercise (P < 0.05). There were, however, no significant differences between the land and water exercise treatments at any time point (P > 0.05). Although the time x treatment interaction was not significant (P > 0.05), the exercise treatments showed greater reductions from baseline than the control treatment throughout the whole day until 22 hours after exercise.



Significant change over time, P <0.05

* Significantly different between both exercise treatments and control group, P < 0.05

+ Significantly different between water exercise and control group, P < 0.05

Figure 9: Absolute changes in MAP from baseline over 24 hours. Time point 0 indicates the baseline value (resting), therefore the change in value form baseline equals zero. Time point 1 is the value within the first hour after exercise, starting immediately after exercise. It indicates the difference between the values within one hour after exercise and the resting baseline values.

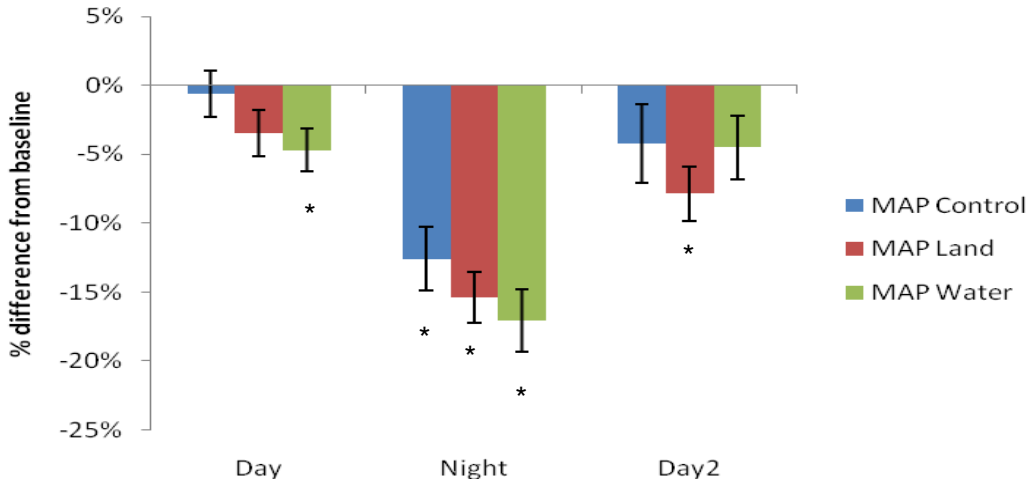
4.2 MAP responses during day and night time

Table 8 depicts the average MAP for day, night and day 2 for the control, land and water treatments. The control (0.7 ± SD 6.1 mmHg, $P > 0.05$) and land exercise (3.9 ± SD 7.51 mmHg, $P = 0.09$) treatments showed non-significant reductions in average daytime MAP compared to baseline. The water exercise was the only treatment that showed a significant reduction in daytime MAP compared to baseline (5.6 ± 6.37 mmHg, $P = 0.04$). All three groups showed significant reductions in night time MAP compared to baseline (control: 14.4 ± SD 8.17 mmHg; land: 17.6 ± SD 19.69 mmHg; water: 20.2 ± SD 7.26 mmHg, $P = 0.000001$). Compared to baseline, the MAP on day 2 was 4.9 ± SD 12.54 mmHg lower for the control session, 8.9 ± SD 10.04 mmHg lower for the land exercise session and 5.4 ± SD 7.62 mmHg lower for the water exercise session. These differences were not statistically significant for the control or water exercise groups, but were significantly lower compared to baseline for the land exercise group ($P = 0.002$).

Table 8: Average MAP after rest, land and water exercise for daytime, night time and next day hours.

	Control			Land			Water		
	Mean	**±**	**SD**	**Mean**	**±**	**SD**	**Mean**	**±**	**SD**
Rest	114.2	±	13.09	114.6	±	13.71	118.5	±	10.88
Day	113.5	±	9.77	110.7	±	8.55	112.9	±	9.54#
Night	99.8	±	12.36#	97.0	±	10.36#	98.3	±	12.22#
Day2	109.3	±	13.54	105.7	±	9.84#	113.1	±	12.82

Significantly lower than baseline, $P < 0.05$



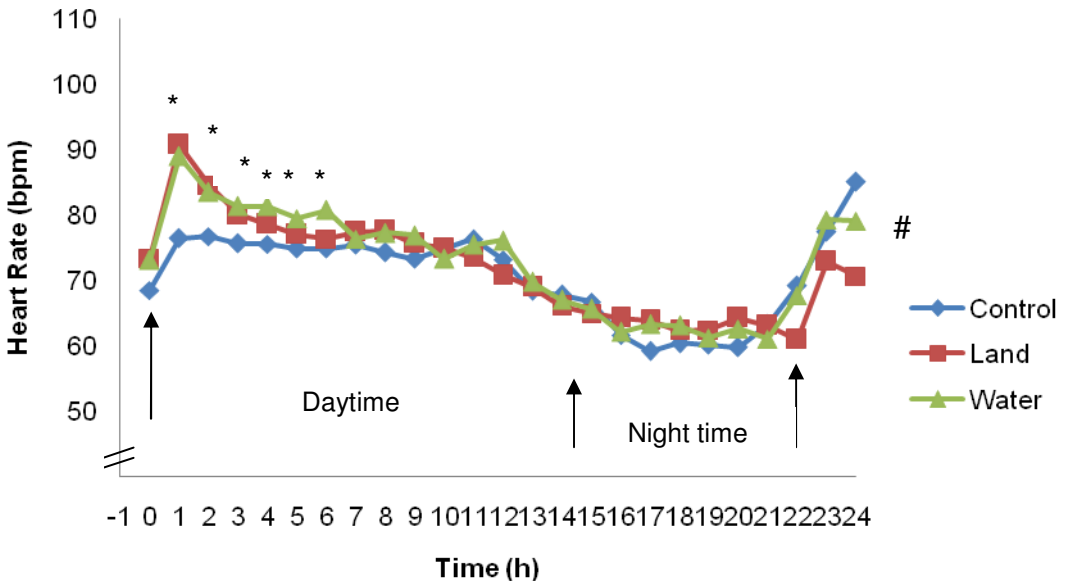
* Significantly different from baseline, $P < 0.05$

Figure 10: Percentage changes in MAP during daytime, night time and the next day after rest, land and water exercise.

5. Heart rate

5.1 Changes in HR over 24 hours

There was a significant time effect in HR for all the groups ($P = 0.000001$) over 24 hours (Figure 11). There was no significant difference in HR values at baseline for the three treatment groups (68 ± SD 10.54, 72.8 ± SD 11.48, 73.6 ± SD 16.99 b.min^{-1} for the control, land and water treatments, respectively; $P > 0.05$). The three treatments resulted in significantly different average HRs over 24 hours (treatment effect, P = 0.002). The average HR over 24 hours, including baseline, was 70.7 ± SD 6.92, 71.4 ± SD 7.70 and 74.6 ± SD 8.11 b.min^{-1} for the control, land and water exercise groups, respectively. Overall, the water exercise treatment resulted in a higher average HR compared to the control and land exercise (respectively 3.8 and 3.1 b.min^{-1}, $P < 0.05$). The difference between the average HR for the land and control groups was 1.3 b.min^{-1}, but failed to reach significance (land > control, $P > 0.05$).



Significant change over 24 h in different treatment groups, $P < 0.05$

* Significantly different between both exercise groups and control group, $P < 0.05$

* Significantly different between water exercise and control group, $P < 0.05$

Figure 11: Average hourly ambulatory heart rate (HR) after rest, land and water exercise.

Figure 11 shows that there was an acute increase in HR immediately after exercise following the land and water exercise treatments. In the first hours after exercise there was 14.4 b.min^{-1} difference between the land and control session (land > control, $P = 0.02$) and a 13.2 b.min^{-1} difference between the water and control (water > control, $P = 0.04$). In the second hour after exercise, the difference between land and control was 7.7 b.min^{-1} ($P = 0.002$) and between water and control it was 7.2 b.min^{-1} ($P = 0.06$). From three to six hours after exercise only the water exercise caused higher HR values compared to the control group ($P < 0.05$). There was a significant difference in HR between the land and water treatment groups at 5 – 6 hours after exercise with the water treatment resulting in a 5.3 b.min^{-1} higher HR than the land exercise ($P = 0.04$). For the rest of the day there were no significant differences in the HR between any of the groups.

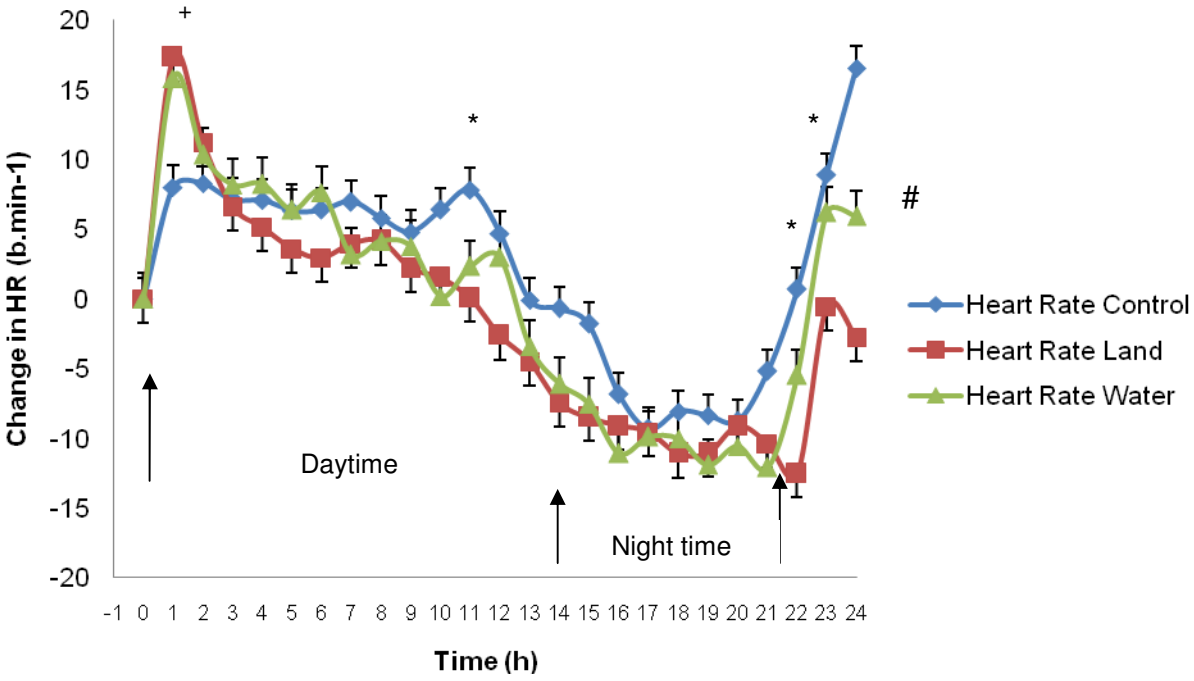
Figure 12 illustrates the difference between HR values at each time point compared to baseline. The control session resulted in an average increase in 24 hour HR of 2.0 ± SD 7.05 b.min^{-1} from baseline, while the land exercise and water exercise

treatment both showed reductions of 1.9 ± SD 7.86 and 0.2 ± SD 8.28 b.min^{-1} respectively, compared to baseline ($P > 0.05$).

There was a significant time x treatment interaction ($P = 0.009$) for HR of the three treatment groups. During the daytime hours HR never decreased below baseline values before 12 hours after exercise for any of the groups. The greatest increase in HR after the control session was 8.3 b.min^{-1} and it occurred between one and two hours after exercise ($P = 0.004$). HR stayed elevated for 11 hours after exercise. The maximum decrease during night time hours was 9.7 b.min^{-1}, ($P = 0.005$), and stayed lowered for most of the night time hours.

Following the land exercise, the greatest increase in HR was seen within the first hour after exercise (17.3 b.min^{-1}, $P = 0.000001$). HR stayed significantly elevated compared to baseline for three hours after exercise. During night time, after the land exercise, HR dropped below baseline values and was significantly lower than baseline between 13 and 22 hours after exercise. The greatest reduction was 11.0 b.min^{-1} ($P = 0.0006$). Compared to the control treatment, the land exercise treatment showed significantly greater increases in HR in the first hour after exercise and then significantly greater decreases in HR at 10 – 11 hours and between 21 and 23 hours after exercise ($P < 0.05$).

The largest increase in HR after the water exercise was 15.6 b.min^{-1} in the first hour after exercise ($P = 0.000001$). HR stayed elevated for six hours after exercise compared to baseline. During night time there was a reduction in HR below baseline. The maximum reduction in HR was 12.2 b.min^{-1} ($P = 00002$). It was significantly lower than baseline between 14 and 21 hours after exercise. The water exercise treatment resulted in significantly greater increases in HR compared to the control group in the first hour after exercise ($P = 0.04$). For the rest of the day, the increases in HR did not significantly differ between the control and water treatments ($P > 0.05$). There was no significant difference in the HR changes between the land and water exercise treatments at any time point ($P > 0.05$).



Significant change over time in all group, P <0.05

+ Significantly different between both exercise treatment and control groups, P < 0.05

* Significantly different between land exercise and control groups, P < 0.05

Figure 12: Absolute changes in HR from baseline over 24 h. Time point 0 indicates the baseline value (resting), therefore the change in value from baseline equals zero. Time point 1 is the value within the first hour after exercise, starting immediately after exercise. It indicates the difference between the values within one hour after exercise and the resting baseline values.

5.2 HR changes during day and night time

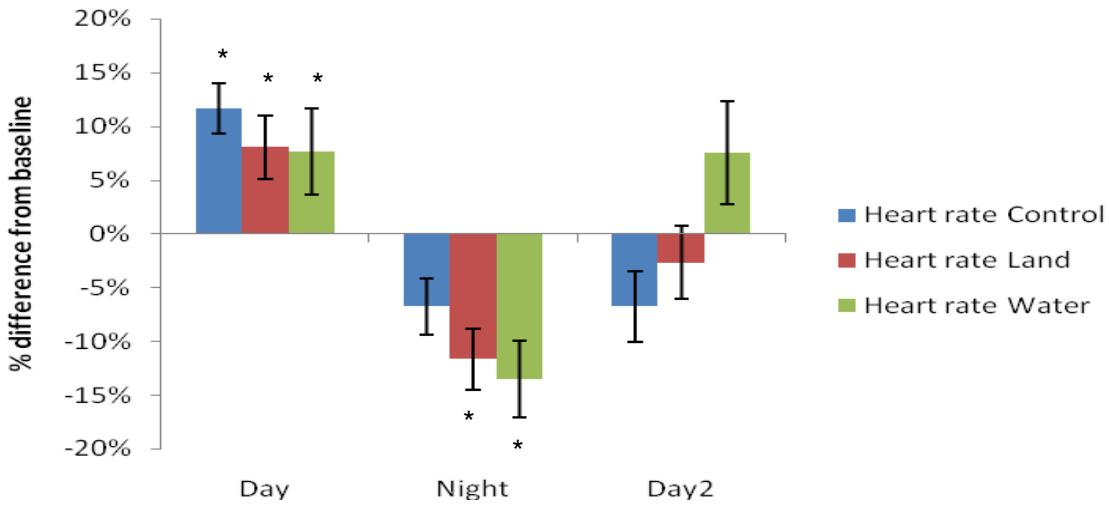
Table 9 illustrates the average resting, daytime, night time and day 2 HR. There was an increase in HR during the daytime following all three treatment groups (control: 8.0 ± SD 6.73 b.min^{-1}, P = 0.003 land: 5.9 ± SD 9.83 b.min^{-1}, P = 0.03; water: 5.6 ± SD 5.13 b.min^{-1}, P = 0.04; Figure 13). During night time the control group showed a non-significant decrease in HR compared to baseline of 4.6 ± SD 8.91 b.min^{-1} (P = 0.09), while the land and water exercise treatment groups showed significant reductions in night time HR compared to baseline (land: 8.5 ± SD 9.63 b.min^{-1} , P = 0.001; water 9.9 ± SD 8.5 b.min^{-1}, P = 0.0006). Compared to baseline, the HR for the control and land exercise treatments were lower on day 2 (control: 4.6 ± SD 10.56 b.min^{-1}, P = 0.07; land: 2.0 ± SD 9.67 b.min^{-1}, P > 0.05). HR was still elevated

on day 2 after water exercise compared to baseline (5.6 ± SD 19.30 b.min^{-1}, P = 0.08).

Table 9: Average HR after rest, land and water exercise for daytime, night time and next day hours.

	Control			Land			Water		
	Mean	**±**	**SD**	**Mean**	**±**	**SD**	**Mean**	**±**	**SD**
Rest	68.5	±	10.54	72.8	±	11.48	73.6	±	16.99
Day	76.5	±	8.51*	78.7	±	9.06*	79.2	±	9.48*
Night	63.9	±	9.76	64.3	±	8.27#	63.7	±	7.92#
Day2	63.9	±	13.09	70.8	±	13.48	79.2	±	17.45

Significantly lower than baseline, $P < 0.05$ * Significantly higher than baseline, $P < 0.05$



* Significantly different from baseline, $P < 0.05$

Figure 13: Percentage changes in HR during daytime, night time and the next day after rest, land and water exercise.

6. Relationship between cardiovascular risk factors and changes in BP

Table 10 shows the correlation between cardiovascular risk factors and the average decrease in SBP and DBP. There was no significant correlation ($P > 0.05$) between the individual cardiovascular risk factors (BMI, body fat percentage, waist-to-hip ratio and cardiovascular fitness) and the average SBP changes after exercise. The

resting SBP and DBP were, however, the only risk factor that had a slight negative correlation with the average decreases in SBP or DBP ($P < 0.05$).

Table 10 (a): The relationship between the average change in SBP after the control, land and water sessions and the different predictors of cardiovascular disease obtained during the health screening.

Variable	Correlation coefficient (r)		
	Control	Land	Water
Delta SBP vs. VO_{2peak}	0.05	0.12	0.13
Delta SBP vs. resting SBP	-0.11	-0.41	-0.42
Delta SBP vs. BMI	0.15	0.02	0.00
Delta SBP vs. waist to hip ratio	-0.31	-0.18	-0.09

Table 10 (b): The relationship between the average change in DBP after the control, land and water sessions and resting DBP as a predictor of cardiovascular disease obtained during the health screening.

Variable	Correlation coefficient (r)		
	Control	Land	Water
Delta DBP vs. resting DBP	-0.50	-0.55	-0.31

SBP, systolic blood pressure; VO_{2peak}, maximum aerobic capacity; DBP, diastolic blood pressure

After the control session more than half of the participants (52%) showed an increase in SBP during the daytime hours compared to the baseline value. However, following the land exercise only four participants (19%) showed increases in SBP during the daytime hours, and after the water exercise only three persons (15%) did not show a decrease in SBP compared to baseline. Following the control, land and water exercise treatment, 8, 7 and 6 individuals respectively showed increases in DBP compared to baseline. Twelve out of 21 patients (57%) showed reductions in MAP during daytime following the control session. Six subjects had an increase in MAP after land exercise and four subjects had increases in MAP following the water exercise. Only one person did not respond positively after either the land or water exercise in terms of changes in SBP, DBP and MAP.

Figure 14 and 15 illustrate the relationship between the average change in SBP during work day hours (3 – 11 hours after exercise) on the land and water exercise day compared to the average work day SBP values on the control day. There was a moderate positive correlation between control day SBP and the decreases in SBP after exercise during work day hours (land: r = 0.45, and water: r = 0.47 respectively, $P < 0.05$). 57% of the participants had lower SBP during the workday after the land exercise, while 45% had lower SBP after the water exercise.

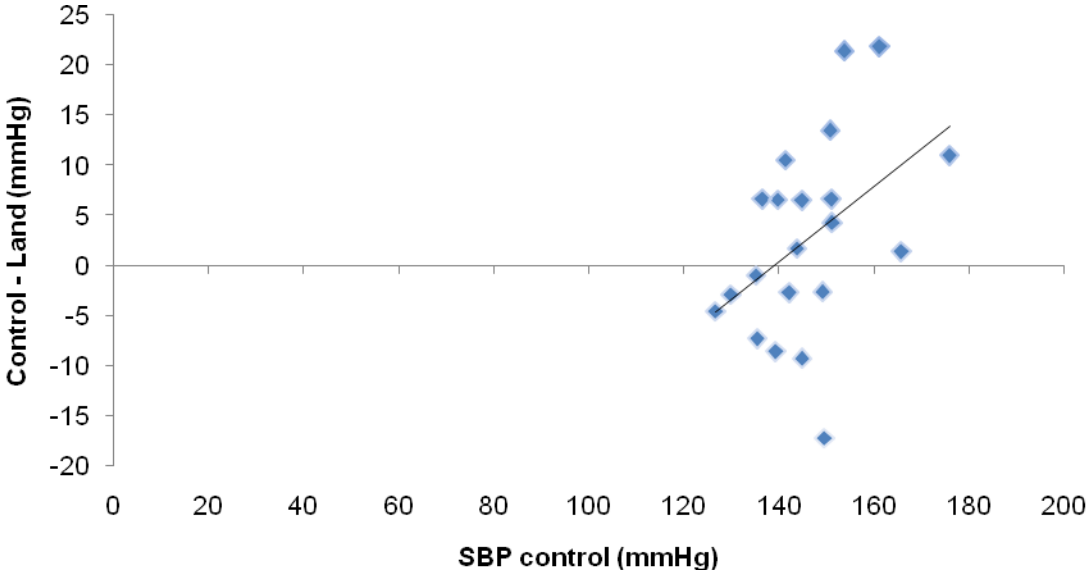


Figure 14: The relationship between the work day hours SBP on the control day and the change in SBP after land exercise (r = 0.45, $P < 0.05$)

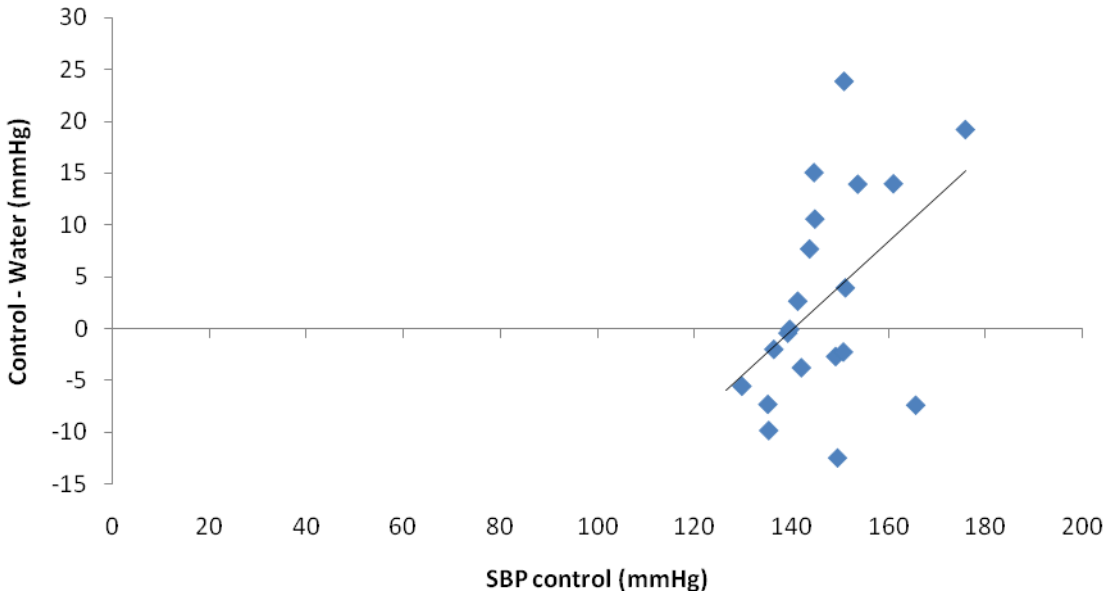


Figure 15: The relationship between the work day hours SBP on the control day and the change in SBP after water exercise (r = 0.47, $P < 0.05$)

CHAPTER EIGHT

DISCUSSION

A. INTRODUCTION

The present study examined the influence of an acute land and water exercise session on the magnitude and duration of post exercise hypotension. Exercise is regularly prescribed as a means of lifestyle modification to prevent and manage hypertension, and therefore this study will not only contribute to the broad knowledge base of this field, but also have important practical relevance.

The main findings of the current study are that both land and water exercise treatments resulted in a significant reduction in SBP during the daytime hours, compared to the control treatment. During the night time hours, the exercise treatment groups still showed greater reductions in SBP than the control group. Although the magnitude of the reduction did not differ between the treatments, the land exercise caused a longer duration of SBP reductions. The average 24 h DBP was significantly lower in the land exercise group compared to the control group. There was no significant decrease in DBP during daytime hours, but all three groups showed significant reductions during night time. The HR after exercise increased significantly compared to the control treatment for two hours, but returned to near baseline values during the day time. The water treatment group showed elevated HR compared to baseline for a longer duration after the exercise than the land exercise group. The results suggest that both a combined session of aerobic and resistance exercise of moderate intensity, as well as moderate intensity water based exercise is effective in reducing BP after an acute exercise session.

Hypertension is one of the most prevalent chronic disorders worldwide and is a major risk factor for cardiovascular disease (Chobanian *et al*., 2003). If hypertension is left untreated it can result in kidney failure, heart failure and damage to the eyes, thus reducing quality of life. Hypertension in the early stages is best treated with lifestyle

modifications, because unlike medication, it not only lowers BP, but it also addresses some of the underlying causes of hypertension.

B. DESCRIPTIVE CHARACTERISTICS

SBP increases with advancing age (Pescatello *et al*., 2004), however, age is not the only risk factor for high BP. Steyn *et al*. (2008) reported that the main predictor for hypertension is an urbanised lifestyle, with a high stress job, inactivity and an unhealthy diet. Thus, elevated BP can become a problem at any age and younger persons exposed to high BP for a long period of time is alarming because of the destructive effect of high BP on the blood vessels. Therefore individuals at risk for hypertension should be identified at a young age to initiate lifestyle changes to prevent hypertension and to decrease the necessity of pharmacological treatment.

In this study the study sample was representative of hypertensive adults between the ages of 30 and 70 years with no other confirmed cardiovascular disease. Most of the subjects were sedentary or recreationally active, overweight and had a low aerobic capacity, typical of an urbanised society. All subjects were classified as either pre-hypertensive or hypertensive. All subjects underwent a familiarization session with the ABP monitor to get used to the monitor and to identify persons with white coat hypertension.

One of the main predictors of PEH is the resting BP, where persons with higher resting BP show greater PEH responses (Queiroz *et al*., 2009; Pescatello *et al*., 2004; 2001; Fisher, 2001). In this study there was a moderate correlation between resting BP and the average decrease in BP (land: $r = 0.41$; water: $r = 0.42$), with the persons with the highest resting BP, showing the greatest reductions in BP after exercise. When comparing the exercise interventions to the control session during the work day hours a significant correlation was found between changes in the work day SBP after land ($r = 0.46$) and water ($r = 0.47$) exercise and control day SBP. This shows that the persons with the higher SBP showed greater decreases after the exercise during the work day hours. The results also suggest the person with high BP would benefit greatly from exercising in the morning, because it causes lower SBP during the stressful work hours, and thus have a cardio protective effect when

needed the most. 57% of the participants had lower SBP during the workday after the land exercise, while 45% had lower SBP after the water exercise. However, the resting SBP of the group varied within a narrow range (124 – 169 mmHg), which may explain the low correlation. The other cardiovascular risk factors, i.e. BMI, aerobic capacity and waist-to-hip ratio, were not significant predictors of PEH (r = -0.18, 0.02, 0.12). Again, because the study sample small and varied within a narrow range with regards to cardiovascular risk factors it is unlikely to see a significant relationship. If the study sample also included normotensive and more stage 2 and 3 hypertensive individuals, one would likely find better correlations. However, examining the predictors of the PEH response was beyond the scope of this study.

Numerous studies have been done to investigate PEH after an acute session of aerobic exercise, in normotensive and hypertensive individuals. Some studies also investigated the effect of resistance exercise on PEH, but the results on the combination of aerobic and resistance exercise are limited (Meredith-Jones *et al*., 2009; Colado *et al*., 2009; Marzolini, 2008; Rezk *et al*., 2006). It has been documented that the PEH response is mainly associated with aerobic exercise, but that resistance exercise, either on its own or in conjunction with aerobic exercise also has a positive effect on BP (Umpierre & Stein, 2007; Cornelissen & Fagard, 2005; Pescatello, 2004; MacDonald *et al*., 1999). Resistance exercise is also associated with increased muscle mass, therefore increased metabolic rate and improvement in the ability to perform tasks of daily living. The importance of resistance exercise in a regular training program can therefore not be ignored. In the rehabilitation setting, persons with high BP would typically perform a combination of aerobic and resistance exercise, therefore this combination was also selected for this study to determine the hypotensive capabilities of such an exercise session. Furthermore, to date there are no studies in which the effect of water based exercise on the PEH were investigated. This is important because water exercise is an excellent alternative medium for exercise, especially in those individuals with a low exercise capacity, older individuals, obese persons and individuals with injuries of the lower extremities. Therefore, the PEH capabilities of water exercise were investigated and compared to traditional land based exercise.

Following the land exercise, no adverse effects were reported in any of the participants. Two subjects reported headaches after the water based exercise and of these two, one subject was severely lethargic and tired compared to after the land exercise. However, no adverse changes in BP were noted in these individuals. These symptoms could be the result of other factors, such as the high humidity and the strong smell of chlorine in the indoor swimming pool area.

C. OUTCOME VARIABLES

1. Systolic blood pressure

Isolated systolic hypertension has been regarded less dangerous than elevated diastolic BP, but in recent years the importance of isolated systolic hypertension as a cardiovascular risk factor has received more attention (Kaplan, 2000). It has been reported that the risk for cardiovascular events are higher with isolated systolic hypertension, than with isolated diastolic hypertension. It is well known that systolic BP increases throughout adult life, thus the risk for developing systolic hypertension increases with advancing age, but hypertension in older individuals is not unavoidable (Pescatello *et al*., 2004; Kannel, 1999). Furthermore, a large difference between systolic and diastolic BP (pulse pressure) is a major determinant of cardiovascular disease, because an increased pulse pressure reflects on aortic and other large vessel stiffening, leading to less compliant vasculature. Thus the lowering of elevated SBP is essential and will decrease the rate of strokes, heart attacks and heart failure, specifically in older adults (Kaplan, 2000; Kannel, 1999). This is therefore the reason why the potential post exercise hypotensive effects of exercise are so important, as it provides an effective strategy to acutely lower BP on a daily basis, without the use of medication.

1.1 Changes in SBP over 24 hours

In this study the average BP, including baseline, over 24 hours was significantly lower (3.6 mmHg) following the land exercise compared to the control treatment. Although the water exercise also resulted in a lower average 24 h BP (2.2 mmHg) compared to the control, the difference was not significant. There was no difference

in the average BP between the land and water exercise treatments. The average 24 h BP for the control treatment was 141 mmHg, staying within the classification for stage 1 hypertension. The average 24 h SBP for the land and water exercise were 137.3 and 138.8 mmHg respectively, both moving down to the pre-hypertensive class. Ciolac *et al*. (2008) also reported significantly lower average SBP (2.9 mmHg) after 40 minutes of moderate intensity aerobic exercise (60% HRR) compared to the control group in pharmacologically treated hypertensive patients. This reduction was significant to decrease BP into normotensive ranges for 20% of the participants that showed marked hypertension, even though they were pharmacologically treated. This shows that exercise can augment the response to drug treatment.

In the first hour after exercise it seemed that the land exercise resulted in an immediate reduction in SBP which gradually levelled off during the rest of the day. This finding is similar to previous research in which an immediate decrease in SBP at the cessation of exercise was evident (Smelker *et al*., 2004; Quinn, 2000; Rueckert *et al*., 1996: Pescatello *et al*., 1991; Wilcox *et al*., 1982). The water exercise, on the other hand, showed a delayed response, by slowly decreasing after exercise and reaching the lowest point only three to four hours after exercise. This could possibly be explained by the hemodynamic effects of the water. Water immersion results in an increased resting SV due to the hydrostatic effect of the water. This can bring about an increased CO and possibly, increased SBP at rest (Cider *et al*., 2005; Šrámek *et al*., 2000; Park *et al*., 1999). It has been reported that at the same relative intensity, water exercise produces a higher RPE and peak SBP than land exercise (Park *et al*., 1999; Hall *et al*., 1998). Thus, the possible greater rise in CO and SBP during water immersion exercise might have attenuated the immediate drop in SBP after exercise.

Another reason for the difference in response between the land and water exercise session could be explained by a difference in exercise intensity. Although both exercise sessions were performed at similar average HR (117 vs. 119 $b.min^{-1}$), it is debatable whether the relative intensity during the water exercise was similar to that of the land exercise session. Eckerson & Anderson (1992) reported that HR is not a good indication of exercise intensity during water exercise, thus the land exercise might have been more intense than the water exercise. According to Darby & Yaekle (2000) one has to exercise at a much higher HR (7 – 13 $b.min^{-1}$) in water to maintain

the same VO_2 than on land exercise. In this study such a high HR was not sustainable in untrained individuals who were not familiar with water exercise. The individuals self selected their intensity during the water exercise to maintain a moderate intensity for the duration of the session. Even if the HR during the water exercise was adjusted by 10 $b.min^{-1}$, it would still fall within the range of the prescribed intensity of exercise (60 – 80% VO_{2peak}). Smelker *et al*. (2004) reported that more intense exercise showed an immediate and greater PEH response, while lower intensity exercise showed a delayed response in PEH. At 120 minutes, there was no difference in the magnitude of the SBP reduction between the intense and low intensity exercise. This could also clarify the difference in response between the land and water exercise in this study in the first four hours after exercise.

In the current study the average SBP reduction over 24 hours compared to baseline was the greatest following land exercise (14.4 mmHg), but it was not significantly greater than the reduction following the water exercise (12.9 mmHg). Both treatment groups showed significantly larger reductions than the control group (4.7 mmHg). In hypertensive individuals PEH of up to 20 mmHg have been observed in studies using auscultatory BP monitoring. However, studies using ambulatory BP monitoring do not show such large PEH responses and it must be remembered that these measures are more reliable and sensitive than auscultatory measures. Numerous variables that could affect the magnitude of PEH have been investigated, but differences in study design and population studied make it difficult to compare the magnitude of PEH.

One of the main predisposing factors of a large PEH is baseline BP. Persons with higher resting SBP usually show the greatest PEH (Pescatello *et al*., 2004, Kenney & Seals, 1993; Somers *et al*., 1991). Another variable that is believed to influence PEH is exercise intensity. There is no certainty regarding the effect of exercise intensity on the magnitude of PEH. Some researchers have shown that there is no difference in the magnitude of PEH between light and moderate intensity exercise (MacDonald *et al*., 1999; Forjaz *et al*., 1998; Pescatello *et al*., 1991). Other researchers, however, have reported that higher intensity exercise resulted in greater decreases in BP after exercise (Guidry *et al*., 2006; Smelker *et al*., 2004; Forjaz *et al*., 2004; Quinn, 2000). Exercise intensity influences the hemodynamic response during exercise therefore it is believed that exercise intensity will also affect the post exercise haemodynamics.

However, most of these studies only measured PEH for a short period of time under controlled conditions; therefore the results cannot be compared to the magnitude of the average PEH over 24 hours. In this study there was no statistically significant difference in the magnitude of SBP reduction following the land and water exercise. Although it is suspected that the land exercise was performed at a higher intensity, this difference in intensity was probably not large enough to cause significant differences in the magnitude of PEH.

In the current study SBP was significantly reduced from baseline for the entire daytime and night time period, except between 9 – 10 h after land exercise. This reduction was significantly greater than those of the control group immediately after exercise for six hours and again between 9 and 19 hours and 23 – 24 h after exercise. The water exercise resulted in significant differences from baseline for nine hours during daytime and again for the entire night time. Compared to the control treatment the reduction following water exercise was significantly greater between one and four hours after exercise. The water exercise also showed a greater hypotensive effect than the control treatment during the night time hours (12 – 18 h). Thus the PEH response lasted for 24 hours after land exercise and 9 hours after water exercise during free living conditions, where subjects continued with a normal work day. The literature reports that duration of PEH appears to be highly variable, with reports of duration of 1h to as long as 24 h (Forjaz, *et al*., 2004; Brandão Rondon *et al*., 2002; Pescatello *et al*., 1991). Relatively few studies have measured PEH for the full 24 h period following exercise, thus results are limited to short laboratory based monitoring periods.

MacDonald *et al*. (2001) reported a 16 mmHg reduction in SBP for 70 minutes after exercise where post-exercise activities were controlled. The researchers speculated that moderate intensity aerobic exercise has the potential of sustained PEH during activities of daily living. Most researchers that measured PEH for a sustained period of time reported significant reductions compared to baseline or compared to a control group after dynamic aerobic exercise, lasting 20 – 60 minutes at low to moderate intensity exercise (40 – 75% VO_{2peak} or 60 – 70% HR_{max}). Pescatello *et al*. (1991) showed a 5 mmHg reduction in SBP compared to baseline and the control group, which was sustained for 8.7 h after exercise. Quinn *et al*. (2000) also found a

significant reduction of 10 mmHg for 12 h after exercise relative to the control session. Brandão Rondon *et al*. (2002) reported lower average SBP compared to the control, which was sustained for 22 h after exercise. Park *et al*. (2006) showed that SBP was reduced after 40 minutes of continuous aerobic exercise for 7 h (5.6 mmHg) and for 11 h (5.4 mmHg) after intermittent exercise.

Unlike the current study, Brownley *et al*. (1996) and Guidry *et al*. (2006) showed an increase in SBP from baseline after exercise, but this increase was significantly smaller in magnitude than in the control group. Brownley *et al*. (1996) showed lower SBP (6.5 mmHg) than the control group that lasted for 5 h after 20 minutes of moderate intensity aerobic exercise , while Guidry *et al*. (2006) reported a 5.6 mmHg lower SBP than the control group after light and moderate intensity aerobic exercise for 9 hours.

Other researchers have found no sustained reductions in SBP or PEH response. Somers *et al*. (1991), Rueckert *et al*. (1996) and Forjaz *et al*. (2004) showed significantly reduced SBP after exercise during BP measures in the laboratory, but when the subjects continued with daily activities, there was no sustained PEH. Somers *et al*. (1991) used a maximal incremental exercise test, which might have been of too high intensity to cause a significant sustained reduction in SBP. The possible reason for the lack of results in all three studies is that the baseline and laboratory measures after exercise were measured by auscultation, while the home BP were measured via an automated or ambulatory BP monitor. Thus the ambulatory values could not be compared to a baseline value and was only compared to values on a no exercise day. The lack of positive results could also not be attributed to exercise intensity, because two of the studies used moderate intensity aerobic exercise for 45 minutes, which is similar to other research that did show a sustained reduction in SBP.

Limited studies are available on the effect of resistance exercise on PEH, but some researchers have shown statistically significant systolic hypotension after a session of resistance training. Boroujerdi *et al*. (2009) showed a 9.6 mmHg lower SBP compared to baseline. MacDonald *et al.* (1999) compared resistance and aerobic exercise and showed that the maximal reduction in SBP was 20 mmHg compared to

baseline after the resistance training. Fisher (2001) also showed a 2.3 mmHg lower SBP than the control treatment. All three studies showed that PEH does occur after resistance exercise, similar to the current study. However, all these studies only measured the recovery SBP for 60 minutes post exercise and could not give an estimation of the duration of PEH after resistance exercise. Only one study measured the effect of resistance exercise on the duration of PEH. Queiroz *et al*. (2009) reported reductions in SBP for 60 minutes under controlled laboratory conditions, but once the subjects left the laboratory the reduction in SBP was diminished. The lack of results is not necessarily due to the modality of exercise, it can also be ascribed to the fact that this study used a normotensive population and that different BP monitors were used for the baseline and ambulatory periods. Sustained reductions might have been observed in a hypertensive population. Thus, the current study is one of the first to show that combining aerobic and resistance exercise result in a significant magnitude and duration of PEH. Resistance exercise is thus effective in causing PEH that can be sustained during activities of daily living.

1.2 SBP responses during day and night time

SBP decreased significantly during daytime in both exercise groups, while the control treatment showed an increase compared to baseline. This shows that morning exercise, irrespective of the modality, causes significant reductions in SBP during the daytime hours, when it is most needed during the stressors of a workday. However, the existence of a circadian rhythm has been reported, but the extent of variation during daytime is still uncertain (Jones *et al*., 2009; 2008b; Mancia *et al*., 1983, Watson *et al*., 1980). In this study the decreases in daytime BP could not be attributed to BP variability alone, because both exercise groups showed significantly greater reductions than the control treatment. Jones *et al*. (2009) showed that morning exercise is associated with small or no reductions in SBP, while PEH was more pronounced after exercise in the afternoon. This was not the case for the present study that showed significant reductions during day- and night time after morning exercise. If the morning surge of BP is associated with cardiovascular events, it is even more important to find a way to decrease the BP in the morning and daytime hours. If exercise is done in the late afternoon, PEH occurs when there is

already a nocturnal fall in BP. Furthermore the PEH would have worn off the next morning when BP again shows an excessive rise.

Brown *et al*. (2002) reported that, due to genetic differences, only 75% of patients will respond positively to exercise, i.e. experience lower BP with exercise. In this study, after the control treatment, most participants showed an increase in SBP during the daytime hours, compared to the baseline value. Following the land exercise only four participants (19%) showed an increase in SBP during the daytime hours, while the rest responded with marked decreases compared to baseline. After the water exercise only three persons (15%) did not show a decrease in SBP compared to baseline. Interestingly, only one man did not respond positively to either the water or the land exercise treatment. The possible reasons could be that he was truly a non-responder; also he could not have reported his daily activities or medication use accurately. Another possible reason is that his daily stressors could have diminished the hypotensive effects, because he did report high psychological stress due to work pressure.

During night time all the groups showed significant decreases in SBP. Nocturnal falls is well documented to range between 10% – 25% from daytime to night time (Lurbe & Redon, 2000; Verdecchia, 2000; Stanton & O'Brien, 1993; O'Brien *et al*., 1993). This was also noted in the current study where the control group showed an 8% decrease from baseline, while both exercise treatments resulted in a 13% decrease from baseline. These findings are similar to those reported by Brandão Rondon *et al*. (2002), however, Ciolac *et al*. (2008) reported no significant reduction in daytime SBP, but only found significantly lower night time SBP after exercise in pharmacologically treated hypertensive patients.

On day 2, SBP was still lower than baseline values after the land exercise treatment (13 mmHg lower), while the differences for the water and control treatments were much less (± 5 mmHg). However, very few measurements were taken between 23 and 24 hours and this may affect the reliability of these results. Only two other studies reported sustained PEH after exercise. Brandão Rondon *et al*. (2002) reported reductions for 22 h after exercise and Ciolac *et al*. (2008) reported 24 h of sustained PEH. These studies, however, only reported average reductions over the

monitoring period and did not comment specifically on the next day, after rising. Most other research reported that the PEH is diminished after 12 hours (Park *et al*., 2006; Quinn *et al*., 2000; Pescatello *et al*., 1991).

It can be concluded that the land exercise treatment resulted in a longer duration of PEH compared to the water exercise. One possible reason for the longer duration of PEH is that the land exercise was performed at a higher intensity than the water exercise. Other studies showed that doing aerobic exercise at a higher percentage of VO_{2max} causes a greater change in magnitude and duration of the hypotensive effect, especially during the evening and sleeping hours (Boroujerdi *et al*., 2009; Forjaz *et al*., 2004; MacDonald, 2002). Quinn (2000) also showed that after exercise at 70% VO_{2max} the duration of systolic hypotension was 13 h, compared to the 4 hours following exercise at 50% VO_{2max}. In a training study by Marceau *et al*. (1993), it was also shown that higher intensity exercise lowered BP during day and night time whereas lower intensity exercise, only lowered BP during the day time.

Another possible reason to suspect a different intensity between land and water exercise treatment is the differences in muscle mass involved during water and land exercise. Dynamic exercise on land might have used larger and more muscle groups than during water exercise, where postural muscles are less active due to the buoyancy of the water. MacDonald *et al*. (2000) reported that there is a difference in the PEH response after arm vs. leg exercise, resulting in greater peripheral vasodilation seen after exercise involving larger muscle mass. The researchers concluded that exercise involving larger muscle mass resulted in a longer duration of PEH. This may therefore be a further explanation why the land exercise resulted in a longer PEH response than the water exercise.

However, even though participants trained at the same HR and used different muscle groups in the water and on land, the water exercise still produced a similar magnitude of PEH. Although the duration of PEH was not as long following water exercise compared to the land exercise, the water exercise still proves to be a good alternative mode of exercise to induce PEH in middle aged, hypertensive individuals.

It is not clear why some studies did not find a sustained PEH response after exercise. Since the volume of exercise was similar in most studies, it is highly unlikely that exercise intensity or duration could explain such large dissimilarities. Ciolac *et al*. (2008) reported that in the studies where no PEH was reported, the exercise intensity was either maximal (Somers *et al*., 1991) or too low (Quinn, 2000). The low intensity exercise results in PEH of a very short duration, while maximal exercise might cause a different hemodynamic, neural or hormonal response during exercise compared to moderate intensity exercise. It can be deduced that moderate intensity exercise, lasting longer than 30 minutes is sufficient in reducing BP after exercise. It is inconclusive whether the lack of positive results in other studies are due to different study designs, study populations, inadequate sample size, inaccurate BP measures or that the participants were non-responders. One possible reason for the differences could be methodological issues, such as using different BP monitoring devices for baseline and ambulatory measures (Rueckert *et al*., 1996). Another methodological difference could be that the pre exercise monitoring period in this study was not longer than 10 minutes, whereas other studies reported more than 20 minutes in a seated or supine position before the start of exercise. This may have resulted in lower baseline values and thus not such large differences as seen in this study. However, in this study the control group spent an hour in the seated position while BP was being monitored. The average SBP over this hour did not differ significantly from baseline values, thus did not confound the results. Another possible reason is that the observer in this study was not blinded to the exercise sessions. An automated BP monitor was used for BP measures throughout the study, thus the measurements could not be influenced by observer bias.

The possible reasons for the positive results obtained in the current study are that the intensity was high enough to cause a significant magnitude of PEH that could be sustained during daytime, and the same BP measuring device was used for baseline and ambulatory measures. The study also included a control session, thus the values obtained on an exercise day could be compared to a non-exercise day. Lastly the sample was large enough to detect small changes. Another possible reason is that the majority of participants were responders to exercise and that their baseline BP was high enough to cause significant PEH.

2. Diastolic blood pressure

Elevated DBP has always been selected as the primary measure of hypertension status, but the association between cardiovascular disease and DBP is not as clear as the association with elevated SBP. Elevated DBP is mostly diagnosed in the younger population, because DBP rise in the early years of life, reaching a peak between 55 and 60 years, where after it decreases (Kannel, 1999). Although elevated DBP does not seem to be as important as systolic hypertension, it cannot be left untreated.

2.1 Changes in DBP over 24 hours

The DBP responded in a similar manner for the three treatment groups throughout the 24 hour monitoring period. According to the baseline BP subjects were classified as hypertensive (> 90 mmHg) on all three occasions. The average DBP following the land exercise was significantly lower (2.7 mmHg) than after the control treatment. The water exercise treatment also resulted in lower average 24 h DBP (1.3 mmHg) than the control, but this difference failed to reach significance. Following the land exercise, DBP decreased below the hypertensive threshold, while following the water and control treatments stayed above 90 mmHg. Ciolac *et al*. (2008) also reported similar lower average DBP (2 mmHg), following 40 minutes of moderate intensity aerobic exercise (60% HRR) compared to the control group in pharmacologically treated hypertensive patients.

The average 24 h decrease from baseline was 7.1 mmHg for the control, 9.0 mmHg for the land exercise and 10.2 mmHg following the water exercise treatment. Both exercise treatments resulted in larger average differences than the control group, but unlike the SBP, the differences were not statistically significantly more than the control group.

The land exercise showed strong trends towards significant decreases from baseline during the early hours of the day, but only reached significance at 7 – 8 hours after exercise. The water exercise treatment caused a significant reduction in DBP compared to baseline for four hours after exercise and again between 8 – 14 h after

exercise. The reductions from baseline were not significantly greater following either exercise session compared to the control session. During night time the land exercise showed significantly greater reductions than the control group at 15 – 16 hours after exercise and water exercise treatments showed significantly greater reductions than the control session between 13 and 16 hours after exercise.

Similar to the result of the current study, Guidry *et al*. (2006), Smelker *et al*. (2004) and Park *et al*. (2008) also reported non-significant decreases in DBP following the light to moderate aerobic exercise. On the other hand, Somers *et al*. (1991) and Rueckert *et al*. (1996) showed significant decreases in DBP in the first hour after exercise, but it returned towards baseline values within one hour, when the subjects left the laboratory. MacDonald *et al*. (2001) reported a significant decrease in DBP of 5 mmHg after aerobic exercise that was sustained during 70 minutes of simulated activities of daily living. The researchers concluded that moderate intensity exercise is sufficient to cause PEH that has the potential of being sustained during activities of daily living. In contrast to the current study, Pescatello *et al*. (1991), Brownley *et al*. (1996), Brandão Rondon *et al*. (2002) and Quinn *et al*. (2000) reported statistically significant decreases in DBP that lasted between five and 22 hours after aerobic exercise. The reason for the discrepancy between these results, and those of the current study could not be explained by exercise variables, because similar exercise intensities and duration of exercise were used in some of the studies that showed a reduction in DBP. The only explanation is that the resistance exercise performed in the current study might have influenced the DBP response compared to the aerobic exercise in the studies that did show positive results.

Fisher (2001) and MacDonald *et al.* (1999) also showed significant reductions in SBP, but no decrease in DBP after resistance exercise. Queiroz *et al*. (2009) found significant reductions in DBP for 60 minutes under controlled laboratory conditions. When the subjects returned home, DBP during daytime was not significantly different from baseline but during night time DBP decreased below baseline values, similar to the current study. In the DBP measures following resistance exercise, Boroujerdi *et al*. (2009) also reported that DBP values returned to baseline within 30 minutes post exercise. The lack of a lower DBP following resistance exercise could possibly be explained by the observations of Rezk *et al*. (2006) who have shown that resistance exercise does not cause a reduction in TPR, but rather a reduction in CO that is

mediated by a reduction in SV. Consequently the reduction in CO will result in the significant reductions observed in SBP but the lack of a decreased TPR will result in an unchanged DBP after a resistance exercise session. In the current study, if the aerobic exercise was divided into two 15 minute bouts at the start and end of the exercise session, with the resistance exercises in between, it might have caused a more sustained decrease in TPR.

The recovery position after exercise might also influence the PEH response. In most studies, subjects remained seated after exercise for the entire recovery and measurement period. The fact that the participants in this study were seated for only 5 minutes after the exercise, (only time to attach the BP monitor) could explain the different results. The vasodilation and blood pooling during seated rest could be diminished due to resuming daily activities after finishing the exercise session. However, it is unlikely that in a real life situation people are going to sit or lie down straight after exercise and remain in that position for an hour. It is therefore important to understand what happens to BP after exercise, when continuing daily activities.

2.2 DBP responses during day and night

None of the groups showed significant decreases in DBP during the daytime hours. The land and control groups showed a less than 2 mmHg reduction in DBP, while the water treatment showed a 4.7 mmHg decrease. Although the decrease after water exercise failed to reach significance, such a large decrease in DBP is still clinically important. A reduction of 5 mmHg does not always reach statistical significance (due to small sample size and the large inter-individual variation), but a 3 mmHg reduction in BP has been reported to reduce mortality substantially, causing a 8% reduction in the occurrence of stroke and a 5% reduction in coronary artery disease (Touyz *et al.*, 2004).

During night time all three treatment groups showed a significant drop in DBP. Similar to the SBP this could be attributed to nocturnal falls in BP, rather than the effect of the exercise. The water exercise treatment showed a 20% decrease from baseline, while the control treatment showed a 16% decrease and the land treatment a 17% decrease compared to baseline. Although the reduction was greater following

the water treatment compared to baseline during night time, this difference was not significantly greater compared to the control or land exercise groups. Queiroz *et al*. (2009) also reported a significant reduction in both the exercise and control group during night time after resistance exercise, in the absence of a reduction during daytime. This can be explained by the nocturnal decrease in DBP, as seen in SBP.

The next day both the control and water exercise groups had a 5 mmHg lower DBP compared to baseline. The land exercise group, however, still showed an 8 mmHg reduction compared to baseline and this was significantly lower than resting values. This shows that although the land exercise did not result in major decreases in DBP, the small reduction was sustained throughout the 24 h monitoring period. However, very few measurements were recorded at 22 – 24 h, thus the reliability of this result is questionable.

In the current study water exercise resulted in greater decreases in DBP during the day and night time. The reason for this larger decrease might be the result of greater vasodilation in the periphery due to the water immersion, compared to land exercise. During water immersion in a thermoneutral temperature, it is suggested that peripheral vasodilation is augmented to compensate for the increase in SV and thus CO (Park *et al*., 1999). It is possible that the water treatment caused greater, sustained vasodilation to compensate for the rise in CO during water immersion, and thus decreased the TPR. Furthermore, the sequence of exercises in the land exercise session might have contributed to the change in DBP. The participants started with 30 minutes of aerobic exercise, followed by resistance exercise. Resistance exercise causes mechanical compression of the vasculature, attenuating the vasodilation that occurred during the aerobic exercise (McArdle *et al*., 2001). Therefore, the subjects may have ended the land exercise with a greater degree of TPR, than what they started with. Another possible explanation is that the water exercise was done at a lower intensity than the land exercise treatment, as explained in the SBP differences as well. Guidry *et al*. (2006) also showed that the reduction in DBP following longer duration low intensity exercise was greater and lasted longer compared to short duration intense exercise.

When analysing the DBP for each individual, it was observed that following the control treatment, 8 of the 21 individuals (38%) showed increases in DBP during the day, while seven (33%) and six (30%) individuals did not respond positively to the land and water exercise, respectively. Two of the individuals did not respond to either the land or the water treatment, and one of these individuals was the same person that did not respond with a lower SBP either.

The reason for the low response in DBP after both treatments is unknown. It is possible that the mechanism responsible for PEH does not affect the aetiology of the DBP, but rather addresses the causes of the increased SBP. The absence of a hypotensive effect during the daytime hours suggests that the mechanism underlying PEH is not merely a sustained reduction in TPR. This indicates that central factors and CO might mediate the reduction in SBP, because if vasodilation was maintained after exercise, a definite decrease in DBP would have been observed, because decreased TPR is associated with a reduction in DBP (McArdle *et al*., 2001). Another possible reason for the lack of statistically significant reductions in DBP is that the sample size was too small. The changes in DBP are very sensitive and are not very large, thus to notice a significant difference a large sample size would be needed. The lack of significant decreases in DBP is not necessarily problematic, because a larger pulse pressure is associated with greater risk for cardiovascular disease. Therefore, if exercise only lowers SBP, as seen in this study, the pulse pressure would be reduced even if DBP do not change. Thus, the cardiovascular risk would be lower irrespective of the change in DBP.

3. Mean arterial pressure

Mean arterial pressure gives an indication of the average pressure that is exerted by the blood on the walls of the arteries during the entire cardiac cycle, and thus reflects on both the SBP and DBP. Combined hypertension, where both systolic and diastolic BP are elevated, occurs predominantly in middle aged individuals and carries the greatest risk for cardiovascular disease. However with ageing, this usually progresses to isolated systolic hypertension, because DBP decreases with age (Kannel, 1999).

3.1 Changes in MAP over 24 hours

The land exercise resulted in a 3 mmHg lower average MAP over 24 h than the control treatment. Although the water exercise treatment resulted in a 1.4 mmHg lower average 24 h MAP than the control group, this difference was not statistically significant. These results are mirrored in the changes in SBP, where only the land exercise group showed a statistically significant lower average pressure than the control group. The difference in the average 24 h MAP between the land and water exercise groups were not statistically significant.

Both exercise treatments showed significantly greater decreases from baseline over 24 hours compared to the control treatment, with no difference between the two exercise treatments. After the land exercise MAP was significantly reduced from baseline values for 8 hours after exercise. It was again significantly lower than baseline between 11 and 23 h after exercise. Compared to the control group the land exercise resulted in greater decreases in MAP for most of the daytime hours, but was only statistically significant between 12 and 16 hours after exercise. The water exercise resulted in a significant reduction compared to baseline from one hour after exercise until 23 h after exercise, except between 7 – 8 h after exercise. The water exercise also showed greater reduction than the control group, reaching statistical significance between 3 and 4 hours and from 12 – 16 h after exercise.

The results of the current study are supported by Guidry *et al*. (2006), Brandão Rondon *et al*. (2002), Brownley *et al*. (1996) and Pescatello *et al*. (1991) that also showed significantly lower MAP that lasted between 5 h and 22 h after light to moderate intensity aerobic exercise. Following resistance exercise MacDonald *et al.* (1999) also reported lower MAP (7 mmHg) compared to baseline values, but this reduction was not sustained during activities of daily living. Very few studies have reported the effect of exercise on MAP, probably because it is a function of SBP and DBP and not a separate entity. However, those that have reported on MAP or mean BP, showed a positive effect of exercise in the reductions in MAP.

3.2 MAP responses during day and night time

During daytime the water exercise was the only treatment to cause significantly lower MAP (5.6 mmHg) compared to baseline. The land exercise resulted in a 4 mmHg reduction compared to baseline. Although this reduction was not statistically significant, it is clinically important. The control group showed no decrease in MAP during day time. Twelve out of 21 patients (57%) showed reductions in MAP during daytime following control treatment. Six subjects (29%) did not respond positively after the land exercise and four subjects (20%) showed increases in MAP following the water exercise. Again only one person did not respond to either the land or water exercise treatment and it was the same subject that did not respond in terms of SBP and DBP either. Guidry *et al*. (2006) and Brownley *et al*. (1996) also showed that the light to moderate intensity aerobic exercise resulted in greater decreases from baseline during daytime than the control group. Thus the lower intensity of the water exercise might have mediated the greater reductions in MAP. Queiroz *et al*., (2009) showed that resistance exercise, however, resulted in an increase in mean BP compare to baseline. Thus it seems that the mechanical compression during the resistance exercise blunts the reduction in MAP during daytime due to increased TPR, similar to the results seen in the current study, where the water exercise resulted in larger decreases in MAP than the land exercise session.

During night time, all three groups showed reductions compared to baseline (12 – 17%). This could be ascribed to the diurnal variation seen in the SBP and DBP as well. It was again evident that following either exercise treatment the reduction during night time was more pronounced than what it would usually be (control). In contrast to the current study, Brownley *et al*. (1996) showed no effect of exercise on the MAP during sleeping hours, but they also did not show a sustained reduction in SBP and DBP during night time hours either, which could explain the lack of a sustained reduction in MAP. Again the differences in results cannot be explained by the variation in exercise variables, because most of the studies implemented moderate intensity aerobic exercise between 20 and 60 minutes, which has lead to mixed results.

During the next day hours, the land exercise was again the only group to show statistically significant lower MAP compared to baseline. After land exercise MAP

was still 9 mmHg lower on day 2, while the difference after the control and water treatments were 5 and 5.4 mmHg, respectively.

4. Heart rate

4.1 Changes in HR over 24 hours

The average 24 h HR was higher following the land and water exercise treatments, compared to the average HR of the control treatment. This was expected as HR increased during aerobic exercise, but it gradually returned to values similar to the control group during the day. The average 24 h HR following the water exercise was higher than the control and land exercise treatments respectively. The reason for the higher average HR following water exercise is unknown, since most researchers reported lower HR during water immersion compared to on land (Barbosa *et al*., 2007; Darby & Yaekle, 2000). The lower HR during water exercise is a result of the increased SV that is mediated by the redistribution of blood away from the extremities to the abdominal and thoracic cavities due to the hydrostatic pressure of the water (Park *et al*., 1999). In the current study the average HR during the land and water exercise treatments was similar, thus no differences in post exercise HR is expected. However, the effects of water exercise on the HR response after exercise is not known, thus it is possible that the changes in the cardiovascular regulation after water exercise can result in a reflex tachycardia. Very few researchers commented on 24 h HR as opposed to BP.

Most researchers only measured HR in studies evaluating the short term hemodynamic response after exercise, which only lasted for one to two hours after exercise. These studies reported that HR returned to pre-exercise values within one to three hours after exercise (MacDonald *et al*., 1999; Forjaz *et al*., 1998; Cleroux *et al*., 1992). In this study, both the land and water exercise resulted in significantly higher HR than the control group for the first two hours after exercise. Although the HR stayed elevated in the water exercise group for another four hours, this was not statistically significant compared to the control group. Forjaz *et al*. (2004), however, showed that after aerobic exercise (75% VO_{2peak}), HR stayed elevated during the whole 24 h monitoring period compared to less intense exercise sessions.

4.2 HR changes during day and night time

All three groups showed significant increases in HR during the daytime hours compared to baseline. This is to be expected as activities of daily living also produced higher HR than during seated rest, as seen during the baseline period. Just like BP, HR shows variability during the day that is influenced by activities of daily living and stress (Parati *et al*., 1996; Mancia *et al*., 1983, Watson *et al*., 1980), but unlike BP one expects HR to increase during the day while busy at work or with daily tasks. Thus the results of the study are not unusual, because one does not expect a low HR during the day, as seen with the PEH response. However, the fact that BP stayed reduced during working hours, in spite of a normal or higher HR during the day, shows that a reduced TPR during the day contribute to the reduction in BP. This advantage of exercise is very important, because it provides a period of reduced load on the heart.

During night time only the land and water exercise treatments showed significant decreases in HR compared to baseline. The decrease from baseline for the control group was 6% and both the land and water exercise was 10.9%. The average HR during night time was similar for the three groups, but the baseline HR was slightly lower for the control group, leading to a non-significant reduction. Brownley *et al*. (1996) showed similar results, reporting no significant differences in HR between the control and exercise groups during the day or night time.

At day 2 there was no significant difference from baseline HR in any of the treatment groups. The water exercise displayed an average HR above baseline, while the control and land exercise treatments still showed HR below baseline. The reason for this difference is unknown. This increased HR might explain the lack of significant results on day 2 seen in the SBP of the water exercise treatment as well.

D. CONCLUSION

No research has been conducted on the effect of water exercise as opposed to land exercise on PEH. The primary finding of this study is that both land and water exercise result in sustained PEH during the working day. Water exercise caused a

significant reduction in SBP for nine hours of the day. It also caused significantly greater reductions in SBP than the control treatment during night time. It has been reported that hypertension seldom occurs in isolated cases, but that it rather occurs in clusters with other chronic conditions like obesity and diabetes. Some of these individuals are not able to perform traditional exercise on land, thus water exercise would be a good alternative medium for exercise by reducing the joint stress and risk for injuries. The water aerobic session in this study was a typical aqua aerobics class that community dwellers would attend at a wellness facility, making it a safe alternative exercise modality for individuals with hypertension.

Most studies reporting sustained PEH only utilized aerobic exercise. However, in the typical rehabilitation session aerobic and resistance exercise will be combined due to the added beneficial effect of resistance training on the muscular integrity. Thus it was important to characterize the hypotensive capabilities of such a session in order to determine whether the goals of rehabilitation will be reached when employing combined aerobic and resistance exercise. The finding that this combination of exercise caused a sustained PEH for 24 hours after exercise is therefore hugely encouraging. In both the water and land exercise sessions, the BP was also monitored under free living conditions. Thus this study gives an indication of the BP response after an acute exercise session during a normal work day, and emphasizes the importance of daily exercise, preferably in the morning, in the treatment and control of hypertension.

This is the first study to show that the magnitude of the PEH response is similar for land and water exercise. Although the land exercise resulted in a longer duration of PEH, the water exercise also resulted in a sustained PEH during daytime hours. If water exercise was done at a higher intensity, it might have resulted in the same duration of PEH than the land exercise. This suggests that either the mechanism responsible for PEH is different for water and land exercise or that the possible difference in intensity of exercise between the land and water treatments resulted in the differences in the magnitude and duration of PEH. If this is the case, then it can be suggested that a higher intensity exercise results in a longer duration PEH.

Most people are in search of an immediate cure for high BP, but due to the significant correlation between BP and cardiovascular disease, the importance of lowering BP should be emphasized. Exercise is an easily administrable, inexpensive and save option in the management of hypertension. Exercise will not only decrease BP, it will also address some of the causes of hypertension and reduce other risk factors of cardiovascular disease. Thus exercise is a valid alternative in the treatment and control of hypertension. The PEH provides a period of reduced load on the heart. In this study it was shown that after land and water exercise this period is sustainable during the working hours of the day.

E. STUDY LIMITATIONS AND FUTURE STUDIES

A limitation of this study was that the intensity of the water exercise program was not monitored and controlled. It has been shown that HR is not a good predictor of exercise intensity during water exercise, due to the hemodynamic alterations with water immersion. During the land exercise treatment the main researcher conducted the exercise sessions and guided the intensity of the exercise session, to ensure that participants exercise between 60 and 80% VO_{2peak}. During the water exercise, participants were not instructed to keep their HR at a certain interval. They were merely told to work at a moderate intensity that could be maintained for 45 minutes. HR was recorded during the water exercise session and did not differ significantly from the average and maximal HR recorded during the land based exercise. Because HR should be adjusted when exercising in the water, the intensity of exercise during the water and land based exercise might have not been the same. However, this did not influence the outcomes of the study, because both the land and water exercise resulted in significant decreases in SBP and MAP that lasted during the working hours of the day. The duration of the hypotensive response was longer following the land exercise, thus in future research the intensity of the water exercise can be adjusted and controlled during the session to ensure similar oxygen consumption than land exercise. Furthermore, it should be examined whether higher intensity water exercise results in a similar duration of PEH than land exercise.

Another limitation of this study was that very few data points were collected on the next day (22 – 24 h after exercise). This could have influenced the reliability of the

results for this time point. After the land exercise, BP was still reduced at 24 h. It is possible that the PEH still persists after 24 h; therefore the true duration of PEH is still unknown. Future studies should consider continuing BP measures for 48 h after exercise. During night time BP was measured every 90 minutes, which possibly could have affected the reliability of these data. However, if measurements are made more frequently during night time, it may significantly interfere with the individual's sleep pattern and the night time data would also not be reliable.

In the current study the subjects completed an activity diary to monitor daily activities, but the activity level of the person is not known exactly. Although daily activities could potentially influence the results, the individuals in this study did not report any outstanding event that could have significantly affected the outcomes of this study. In any case, the purpose of the study was to evaluate the post-exercise BP response during daily activities and not during controlled laboratory conditions which would have little practical value. To an extent, the effects of daily activities were controlled by measuring BP on a no exercise day as well, making this study more realistic because it shows that PEH is sustained during a normal work day, irrespective of daily activities and stress. However, in future studies a pedometer could be attached during the 24 h follow up period to measure activity level during the day and bring the activity level into account when determining PEH.

No results are available on the hemodynamic changes following water based exercise, thus future studies should monitor the hemodynamic changes following water and land exercise. Most studies only measured the haemodynamics for 1 – 2 h after exercise, therefore no data beyond this point exists and further research is warranted. Further research is also needed to investigate the different mechanisms responsible for PEH following land and water exercise.

In theory repeated bouts of acute exercise can lead to accumulative benefits affecting the cardiovascular system. One of the main questions regarding the reduction in BP after long term training is whether the reduction is the result of the exercise *per se*, or whether it is the accumulative effect of several acute bouts of exercise. Future studies need to examine whether PEH after an acute exercise bout

is similar before and after a period of exercise training, or if the PEH will level off after exercise training.

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APPENDIX A

ACSM RISK SCTRATIFICATION: Coronary artery disease risk factor thresholds

Positive risk factor	**Defining criteria**
1. Family history	Myocardial infarction, coronary revascularization, or sudden death before 55 years of age in father or other male first-degree relative (i.e., brother or son), or before 65 years of age in mother or other female first-degree relative (i.e., sister or daughter)
2. Cigarette smoking	Current cigarette smoker or those who quit within the previous 6 months
3. Hypertension	Systolic blood pressure of ≥140 mm Hg or diastolic ≥90 mm Hg, confirmed by measurements on at least 2 separate occasions, or on antihypertensive medication.
4. Dyslipidemia	Total serum cholesterol of >200 mg/dl (5.2 mmol/L) or high-density lipoprotein cholesterol of <35 mg/dL (0.9 mmol/L), or on lipid-lowering medication. If low-density lipoprotein cholesterol is available, use >130 mg/dL (3.4 mmol/L) rather than total cholesterol of >200 mg/dL.
5. Impaired fasting glucose	Fasting blood glucose of ≥110 mg/dL (6.1 mmol/L) confirmed by measurements on at least 2 separate occasions
6. Obesity	Body Mass Index of ≥30 kg/m^2, or waist girth of >100 cm (≈ 39.4 inches).
7. Sedentary lifestyle	Persons not participating in a regular exercise program or meeting the minimal physical activity recommendations from the U.S. Surgeon Generals' Report.
Negative risk factor	**Defining criteria**
1. High-serum HDL cholesterol	>60 mg/dL (1.6 mmol/L)

(Taken form: ACSM's guidelines for exercise testing and prescription, 2006:22)

APPENDIX B

Test termination criteria

- Onset of angina or angina-like symptoms
- Significant drop (20 mm Hg) in systolic blood pressure or a failure of the systolic blood pressure to rise with an increase in exercise intensity
- Excessive rise in blood pressure: systolic pressure >260 mm Hg or diastolic pressure > 115 mm Hg
- Signs of poor perfusion: light -headedness, confusion, ataxia, pallor, cyanosis, nausea, or cold and clammy skin
- Failure of heart rate to increase with increased exercise intensity
- Noticeable change in heart rhythm
- Subject requests to stop
- Physical or verbal manifestations of severe fatigue
- Failure of the testing equipment

(Taken form: ACSM's guidelines for exercise testing and prescription, 2006:78)

APPENDIX C

PRE-PARTICIPATION INFORMATION AND SCREENING FORM

Name & Surname: _____ ID no:_____

Gender: M / F Age:_____Ethnic group:_____

Occupation:_____ Tel no:_____email:_____

Please answer the following questions

Do you have any of the following diagnosed diseases (only mark if positive)

Cardiovascular disease – cardiac, peripheral, vascular or cerebrovascular disease	
Pulmonary disease – chronic obstructive pulmonary disease, asthma, interstitial lung disease or cystic fibrosis	
Metabolic disease – Diabetes mellitus (type I or II), thyroid disorders or liver disease	

Do you have any of the following coronary artery risk factors? (Only mark if positive)

Positive risk factor	
1. Family history of cardiac disease. Do you have a close relative who has had a heart attack or sudden death before age 55 (father or brother) or age 65 (mother or sister)?	
2. Cigarette smoking or stopped smoking in last 6 months	
3. Hypertension (High blood pressure)	
4. Dyslipidemia (High cholesterol)	
5. Impaired fasting glucose	
6. Obesity	
7. Sedentary lifestyle (less than 30 minutes a day 3 days per week)	
Negative risk factor	
1. High-serum HDL cholesterol	

Do you have signs and symptoms of any of the following?

Pain, discomfort (or other anginal equivalent) in the chest, neck, jaw, arms, or other	
Shortness of breath at rest or with mild exertion	
Dizziness or syncope	
Orthopnea (breathing discomfort when not in an upright position) or paroxysmal nocturnal dyspnea (interrupted breathing at night)	
Ankle oedema (swelling)	
Palpitations (abnormal rapid beating of the heart) or tachycardia (rapid heart beat)	
Intermittent claudication (cramping pain and weakness in legs, especially calves,	

during walking due to inadequate blood supply to muscles)	
Known heart murmur (atypical heart sound indicating a structural or functional abnormality)	
Unusual fatigue or shortness of breath with usual activities	

Please answer the following questions to the best of your knowledge:

Are you aware through your own experience, a doctor's advice, or any other physical reason that would prohibit you from engaging in physical activity?	
Do you have a bone or joint problem that could be aggravated by engaging in physical fitness testing?	
Are you currently experiencing or have you recently experienced any muscle or joint pain?	
Did you experience any of the following problems / conditions recently: severe and chronic headaches, abdominal problems, gout, anaemia, hernia, head injury or epilepsy?	
Have you been pregnant recently?	
Have you recently been ill or injured? Please specify if yes.	

Do you take medication for any of the following?

Heart condition		Depression	
Blood pressure		Pain killers	
Cholesterol		Thyroid	
Insulin or other medicine for diabetes		Allergies	
Hormones		Weight loss	
Asthma		Arthritis	
Anxiety			
Any other medication. Please specify			

Please report your physical activity participation in the past six months

Did you participate in any physical activity?	
How many times per week (days per week)?	
How long was each exercise session (minutes)?	
Type of activity e.g. swimming, walking, resistance etc.	
Intensity of exercise (light / moderate / intense)	

Resting blood pressure

1		2		3		average	

Resting heart rate (bpm)	
Height (m)	
Weight (kg)	
BMI ($kg.m^{-2}$)	
Waist circumference (cm)	
Hip circumference (cm)	
Waist to hip ratio	
Fating blood glucose (mmol/L)	
Cholesterol reading (mmol/L)	

APPENDIX D

STELLENBOSCH UNIVERSITY
CONSENT TO PARTICIPATE IN RESEARCH

The magnitude and duration of post exercise hypotension after land and water exercise

You are asked to participate in a research study conducted by Aletta Esterhuyse, Hons Sport Science (Biokinetics) from the Sport Science Department at Stellenbosch University. The results obtained in the study will be made available to the study leader and will be used for a master's thesis. It can also be published in scientific journals. You were selected as a possible participant in this study because only individuals with mild to moderate hypertension, which are not participating in formal activities, qualify for this studied.

1. PURPOSE OF THE STUDY

The primary aim of the study is to examine the magnitude and duration of post exercise hypotension during free living conditions in persons with mild to moderate hypertension. The study also compares the effects of land based training to water based training on the blood pressure response to exercise.

2. PROCEDURES

If you volunteer to participate in this study, we would ask you to participate in the following:

2.1 The first visit is a screening procedure of about 30 minutes to ensure that all participants conform to the set criteria. The following will be done: body composition by determining height, weight, waist circumference, hip circumference and body fat percentage and three resting blood pressure measurements. All testing and follow up sessions will be done at the Sport Physiology Laboratory at Coetzenburg, Stellenbosch.
2.2 The second visit is a baseline test of 2 hours, at least 7 days prior to the exercise session. The following will be done: a maximal incremental aerobic exercise test where expired gasses will be collected, to determine maximal aerobic capacity. Participants will also be connected to a 12 lead ECG to monitor the cardiovascular changes during exercise. Ten repetition maximum tests will also be conducted to determine upper and lower body strength.
2.3 During the third visit you will do a sub-maximal exercise session, consisting of water aerobic exercises. After the exercise an ambulatory blood pressure monitor will be fitted to monitor blood pressure for 24 hours, as you proceed with daily activities. You will also keep an activity diary for 24 hours of you daily activities.
2.4 The fourth visit is a follow up visit after 24 hours, where only blood pressure will be measured and will only take 10 minutes.
2.5 During one of these sessions you will do a control session of 60 minutes of seated rest while blood pressure and heart rate will be continuously monitored. After this session blood pressure will also be monitored for 24 hours.
2.6 The sixth visit is a follow up visit after 24 hours, where only blood pressure will be measured and will only take 10 minutes.
2.7 During the seventh visit you will do a sub-maximal exercise session, consisting of 30 minutes of aerobic exercise between 60 – 80% of my VO_{2peak} and 30 minutes of resistance exercise at 65% of 1RM. An ambulatory blood pressure monitor will be fitted to monitor blood pressure for 24 hours, as you proceed with daily activities. You will also keep an activity diary for 24 hours of you daily activities.
2.8 The eight visit is a follow up visit after 24 hours, where only blood pressure will be measured and will only take 10 minutes.
2.9 During the ninth visit to the laboratory you will do a control session of 60 minutes of seated rest while blood pressure and heart rate will be continuously monitored. After this session blood pressure will also be monitored for 24 hours

2.10 The tenth visit is again a follow up visit after 24 hours, where only blood pressure will be measured and will only take 10 minutes.
2.11 You will do your best to be available for follow up testing.

3. POTENTIAL RISKS AND DISCOMFORTS

It is possible that you may experience adverse effects during or after the exercise testing or exercise training session. The symptoms include the following: dizziness, nausea, fainting, abnormally high or low blood pressure, heart beat disorders (too slow, too rapid or irregular), shortness of breath or bronchoconstriction. None of the testing or exercise sessions involve invasive procedures, and all are standardized tests. All testing will be done under supervision of a medical doctor (see protocol for details) and all possible steps will be taken to ensure a safe environment. All staff that will be involved in this project is qualified in CPR and basic life support, and emergency equipment is available on the premises of the laboratory. ER24 will also be on standby should any emergency situation arise.

If you experience any of the adverse symptoms described above, you can stop the exercise tests or training session at any time. The tests will be terminated if an ST segment depression or elevation of more than 1mm occurs on an ECG monitor. If any of the equipment fails, the tests will be terminated.

4. POTENTIAL BENEFITS TO SUBJECTS AND/OR TO SOCIETY

You will learn important information about your current health status and possible risk factors could be identified that could prevent future cardiovascular disease, especially for older participants. The exercise might also decrease the hypertensive patient's blood pressure and be advantageous to your health. You may also have the opportunity to participate in training sessions, supervised by a Biokineticist, which may inspire you to commit to long term regular training.

The treatment of hypertension is expensive, with high costs to both the individual and the health care system. Exercise is a highly cost effective alternative in the treatment of hypertension; however, one should then make sure that the best possible exercise is prescribed in order to obtain the best possible results. This will not only ensure that the condition is treated effectively, but it will also contribute to better exercise adherence by patients when they become aware of its positive effects. Furthermore, this study is one of only á few studies to test the magnitude and duration of the post-exercise hypotensive response under free-living conditions and therefore determine the true clinical significance of exercise in the treatment of hypertension.

5. PAYMENT FOR PARTICIPATION

No participant will be remunerated for participating in the study.

6. RESPONSIBILIETS

For the duration that the participant is in possession of the ambulatory blood pressure monitor, the participant will responsible for the monitor and for any damage that occurred to the monitor during that time.

7. CONFIDENTIALITY

Any information that is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission or as required by law. Confidentiality will be maintained by means of assigning a number to your data in order to keep personal information confidential. Data will be stored on a computer, which is password protected. All hard copies of testing data will be stored in a locked cabinet in the Sport Physiology Laboratory. Only the personnel from the Sport Physiology Laboratory and the study leader will have access to the data.

The data can be made available to the participant in the form of a standardized laboratory report. No data will be revealed to other parties, it will only be published in a master's thesis or in scientific journals. No raw data will be published, it will be reported as means of a groups.

8. PARTICIPATION AND WITHDRAWAL

You can choose whether to be in this study or not. If you volunteer to be in this study, you may withdraw at any time without consequences of any kind. The investigator may withdraw you from this research if circumstances arise which warrant doing so. If you fail to comply with the inclusion criteria or if you are not available for re-testing you may be withdrawn from the study. If any health risks arise that could endanger your life, you might be withdrawn from the study.

9. IDENTIFICATION OF INVESTIGATORS

If you have any questions or concerns about the research, please feel free to contact:
Aletta Esterhuyse (021) 808 2818 or Prof E. Terblanche (021) 808 4915
Sport Science Department, Coetzenburg, STELLENBOSCH.

10. RIGHTS OF RESEARCH SUBJECTS

You may withdraw your consent at any time and discontinue participation without penalty. You are not waiving any legal claims, rights or remedies because of your participation in this research study. If you have questions regarding your rights as a research subject, contact Ms Maryke Hunter-Husselman (mh3@sun.ac.za; 021 8084623) at the Division Research Development.

SIGNATURE OF RESEARCH SUBJECT OR LEGAL REPRESENTATIVE

The information above was described to me by Aletta Esterhuyse in Afrikaans and / or English and I am in command of this language. I was given the opportunity to ask questions and these questions were answered to my satisfaction.

I hereby consent voluntarily to participate in this study. I have been given a copy of this form.

_____

Name of Subject/Participant

_____ _____

Signature of Participant **Date**

SIGNATURE OF INVESTIGATOR

I declare that I explained the information given in this document to _____.

He / she was encouraged and given ample time to ask me any questions. This conversation was conducted in Afrikaans and / or English.

_____ _____

Signature of Investigator **Date**

_____ _____

Signature of Witness **Date**

APPENDIX E

Activity diary

Wake-up time:_____ Hours Sleep:_____ Bed time:_____

Time of day	Food consumed	Liquids consumed	Physical activity		Significant events
Morning 06:00 – 12:00		Coffee / Tea:	Do you spent most of your time:		
		Water:	Sitting		
		Fizzy drinks:	Standing		
		Other:	Standing and walking		
			Doing physical tasks		
Afternoon 12:00 – 18:00		Coffee / Tea:	Do you spent most of your time:		
		Water:	Sitting		
		Fizzy drinks:	Standing		
		Other:	Standing and walking		
			Doing physical tasks		
Night 18:00 – 22:00		Coffee / Tea:	Do you spent most of your time:		
		Water:	Sitting		
		Fizzy drinks:	Standing		
		Other:	Standing and walking		
			Doing physical tasks		

Comments:_____