THE RELATIVE EFFECTIVENESS OF OLEA EUROPEA SUBSP. AFRICANA AQUEOUS LEAF EXTRACT AND OF OLEA EUROPEA SUBSP. AFRICANA 6CH ON MILD TO MODERATE HYPERTENSION.

GARNET RONANDER

THE RELATIVE EFFECTIVENESS OF OLEA EUROPEA SUBSP. AFRICANA AQUEOUS LEAF EXTRACT AND OF OLEA EUROPEA SUBSP. AFRICANA 6CH ON MILD TO MODERATE HYPERTENSION.

BY

GARNET RONANDER

Dissertation submitted in partial compliance with the requirements for the Master's Degree in Technology: Homoeopathy in the Faculty of Health at the Technikon Natal.

I, Garnet Ronander do declare that this dissertation represents my own work in both conception and execution.

Signature of Student Date of Signature

APPROVED FOR FINAL SUBMISSION

Signature ~~of Supervisor~~ Date of Signature

Dr G Mc David M. Dip. Hom.

This dissertation is dedicated to my Grandfather and Grandmother, Mr and Mrs Reed who have been a continual inspiration to me through the years. Whose love and support have made possible everything that I have ever been able to achieve.

To them I owe everything.

To my loving wife Debbie Ronander, you are my future happiness.

ACKNOWLEDGEMENTS

I would like to thank all the patients that volunteered to take part in this study as well as the many people who helped me to reach as many people as this research required.

Dr Burger for his help and guidance.

Mr Nischen Govender for his help with completing the statistics.

Dr G. Mc David for his help in preparing the proposal and to it's final submission.

Mr Robert Bode who kindly allowed me to use his resources to complete this research.

The staff of Matushi for making themselves available as willing volunteers

The staff of The Silk Screen Company for making themselves available as willing volunteers

The staff of National Stevedores for making themselves available as willing volunteers

The staff of Clover Dairies for making themselves available as willing volunteers

ABSTRACT

This study investigated the effect of Olea europea subsp africana aqueous leaf extract (tincture), 6CH and placebo on mild to moderate hypertension, in order to determine whether any of these forms of Olea europea subsp africana are capable of producing a significant reduction in the systolic or diastolic blood pressures.

Patients were screened for mild to moderate hypertension and diagnosed after three successive measurements on three different visits. Once the patients were selected and agreed to take part in the study, they were randomly divided into one of three groups. Group 1 received 6CH Olea europea subsp africana, Group 2 received placebo and Group 3 received the Olea europea subsp africana tincture. The patients were instructed to take twenty drops three times a day for the duration of the research. The patient's blood pressures were recorded every three to four weeks and at each visit three readings were taken and the mean of these three readings was used for analysis.

The Kruskal-Wallis test showed no statistically significant difference between the three groups. The Friedman test however showed that all three groups had shown statistically significant improvement in the period of the research. The data was then analysed visually by means of bar charts using the mean levels of the systolic and diastolic readings of each visit. This showed that all the groups showed a decrease in mean blood pressure with Group 1 (6CH) having the greatest systolic drop of 11mmHg systolic and 5mmHg diastolic. Group 3 (tincture) had the second largest drop of 9mmHg systolic and 5mmHg diastolic where Group 2 (Placebo) showed the smallest drop with a 6mmHg drop in systolic and a 3mmHg drop in diastolic mean blood pressure.

Both Group 1 and Group 3 showed reductions in blood pressure classification, changing from mild hypertension to normal high hypertension and from normal high to normal blood pressure respectively. Group 2 did not show enough decrease in systolic and diastolic blood pressure levels to be reclassified. (Lane, 1997:116).

It was therefore concluded that, although not statistically different, Groups 1 and 3 showed more improvement than Group 2. Thus, both Olea europea subsp africana 6CH and tincture were found to be effective in decreasing the mean blood pressure over the period of the research, with the 6CH being slightly more effective (2 mmHg systolic) than the tincture.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER ONE

INTRODUCTION

1.1 Introduction

Good health into old age is one of life's most treasured assets. High blood pressure can silently do damage to our body's organs causing them to deteriorate and fail us as we reach the later stages of our life. It is because of it's largely asymptomatic nature that hypertension has been called the "Silent Killer" (Kenneth & Kiple, 1993:789).

This study investigated the usage and effectiveness of a locally occurring plant species, the African Wild Olive tree or Olea europea subsp. Africana in the treatment of hypertension. The aim of this study was to investigate the effect of an aqueous leaf extract (mother tincture) and a 6CH Homoeopathic potency of Olea europea subsp. Africana in a clinical trail with patients which are diagnosed with mild to moderate hypertension. In this way it was hoped to determine whether this substance is capable of producing a sustained decrease in blood pressure or not. Homoeopathic and Phytotheraputic (herbal) theory and practice proposes that Homoeopathic remedies in both the mother tincture (aqueous leaf extract) and the 6CH dilution are capable of such effects (Clarke 1992:63-64).

There is nothing new about the use of herbs to promote good health, recovery and general well being. Every culture throughout the world has at some point in time used plants as the basis for their medicine. Modern medicine has the use of herbs at its roots and until about fifty years ago. In fact, nearly all the entries in pharmacopoeias describing the manufacture of drugs indicated a herbal origin. (Hoffmann 1996:10).

Recently, many plants have been found to contain chemicals that have a direct action on the heart and circulatory system and have been shown in laboratory studies and clinical trails to decrease blood pressure. Some of these include Apium graveolens, Akkium sativum, Pueraria lobata and many others (Duke 1997: 308-312). Some of these plants are rich in a group of chemicals called cardiac glycosides (Hoffmann 1996:179). Cardiac glycosides are a group of chemicals which occur in certain plants and have a characteristic action on the contractile force of the cardiac muscle (Taylor 1988:708).

The leaf of the Olea europea subsp africana was used to obtain the mother tincture which was used as both a tincture and made up to the 6CH potency. The leaf of the Olea europea has many chemical constituents, a few of which have been shown to possess hypotensive properties. Oleuropein, which is found in the leaf, has been shown to be a hypotensive agent in the dog and rat (Bruneton, 1995:488-489). 3,4 – Dihydroxyphenylethanol, which arises from the hydrolysis of oleuropein, is also a calcium antagonist (Bruneton, 1995:488-489). Oleacein, which is another constituent of the leaf, inhibits the angiotensin-converting enzyme (ACE) and has a combined effect with oleuropein (Van Wyk, et al., 1997:180-181). These known constituents of the olive leaf along with the fact that no clinical trial can be found that tests the effectiveness of the Olea europea subsp africana tincture or 6CH in humans was the reason why it was considered for this clinical trial.

The Homoeopathic potency of 6CH Olea europea subsp africana was made up by a dilution process of serial dilution and succussion. In this process 1 part of solute to 99 parts of the solvent is used at each stage of succussion and this is known as the Hahnemannian method after the founder of Homoeopathy, Dr Samuel Hahneman. The solvent is purified water or alcohol unless the solvent is initially not soluble, in which case lactose is the solvent, and

serial dilution is by trituration until such a time as the solvent becomes soluble (usually after the 4th centesmal dilution). Succussion is a process of vigorous shaking with impact and occurs at every step of the dilution process. (Kayne 1997:49-50.)

This dilution process is known as "potentisation", because it conveys the sense that potentisation is an active process which is aimed at enhancing the substance's therapeutic effect (Gaier 1991:444). The process of serial dilution and succussion has another purpose and that is to decrease the potentially harmful effects of the crude substance (Hahneman 1996:67-70).

The 6CH centesimal dilution, which was utilised in this study, represents a deconcentration of 10^{-12} prepared according to the Hanhemanian methodology. The 6CH potency is used when frequent repetition and a physiological effect is required, when the medication is selected according to the pathology and is not specific to the mental and emotional symptoms (Hershoff 1996:58-59). The 6CH potency level can also be used up to three times a day (Lockie & Geddes 1992:25).

Within the field of Homoeopathy and Phytotherapy this study adds to the studies already conducted on the effects of the Olea europea subsp africana and adds to the understanding of how it can be successfully applied in treating patients with mild to moderate hypertension.

1.2 Aim of the study

The aim of the study was to determine the effectiveness of Olea europea subsp africana aqueous leaf extract and 6CH in hypertension.

1.3 Statement of the objectives

The purpose of this placebo controlled study is to investigate the relative effectiveness of the Olea europea subsp africana aqueous leaf extract and Olea europea subsp africana 6CH on mild to moderate hypertension terms of objective clinical measurements.

1.4 Statement of the hypothesis

All hypotheses are stated in the null form.

1.4.1 The first hypothesis

It is hypothesised that the Olea europea subsp africana aqueous leaf extract will have no significant effect on hypertension in terms of its objective clinical measurements.

1.4.2. The second hypothesis

It is hypothesised that the Olea europea subsp africana 6CH will have no significant effect on hypertension in terms of its clinical measurements.

1.4.3. The third hypothesis

It is hypothesised that the placebo group receiving 20% ROH will have no significant effect on hypertension in terms of its objective clinical measurements.

1.5. The significance of the study

This study is significant in that it tests the effectiveness of a traditionally used South African subspecies of a well known and widely tested European plant. This is the first time that this leaf extract of our South African subspecies has been tested for its effects on mild to moderate hypertension.

1.6. The implications of the study

The positive results of this research project pave the way for future research as well as use of this locally occurring plant for the treatment of mild to moderate hypertension.

CHAPTER TWO

REVIEW OF THE RELATED LITERATURE

2.1 Definition and classification of hypertension

Arterial hypertension is defined as an elevation of systolic and/or diastolic blood pressure, and can be either primary (essential hypertension) or secondary hypertension (Berkow 1996:413). Hypertension can be further classified according to its severity:

Normal blood pressure systolic below 130 and diastolic below 85mmHg.

High normal blood pressure systolic 130-139 and diastolic 85-89mmHg.

Stage 1 (mild) systolic is between 140-159 and diastolic is 90-99mmHg.

Stage 2 (moderate) systolic is between 160-179 and diastolic is 100-109mmHg.

Stage 3 (severe) systolic is between 180-209 and diastolic is 110-119mmHg.

Stage 4 (very severe) Systolic is above 210 and systolic is above 120mmHg.

(Lane, 1997:116.)

Essential or primary hypertension is the most common form of hypertension (85-90% of the cases), in the other 5 – 10% hypertension is secondary to a primary disease, such as a renal, vascular or central nervous system (CNS) disorder (Berkow, 1996:415). Primary or essential hypertension is a silent but progressively lethal disease that may go undetected for one or two decades until serious end organ damage has ensued leading to debilitating complications (Kuteyi & Akinsola, 1998). These complications principally involve the CNS, the retina, the heart and the kidneys (Edwards, 1995:266). Systolic pressure in excess of

160mmHg also carries with it a 2.5 times greater probability of premature death than patients with a systolic pressure of about 140mmHg (Kenneth & Kiple, 1993:789).

2.2 Aetiology of hypertension

Primary or essential hypertension is of unknown aetiology. It is probable that there are many causes as a single cause would not explain it's diverse hemodynamic and pathophysiologic derangements (Berkow 1996:413). Many different actions or situations can normally raise blood pressure, for example stress or exercise. For this reason a diagnosis can only be made when a person has multiple high blood pressure readings over a period of time (Olendorf & Jeryan 1999:1523).

2.2.1 Heredity factors in hypertension

Heredity undoubtedly predisposes individuals to hypertension, although the exact mechanism is unclear. Isolated, perfused kidneys from Dahl salt sensitive rats do not excrete water or Na as rapidly as those from Dahl salt resistant rats, even before hypertension develops. Environmental factors (e.g., dietary Na), obesity and stress seem only to act in genetically susceptible individuals. (Berkow 1996:413).

2.3. Pathogenesis of hypertension

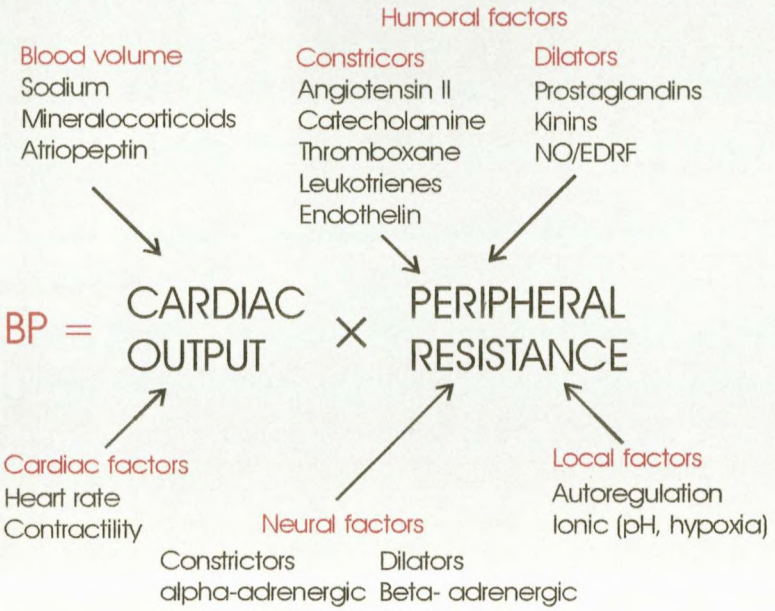


Figure 2-1 Blood pressure regulation. NO/EDRF denotes nitric oxide-endothelium-derived growth factor. (Kumar, et al. 1992:460).

Whatever the responsible pathogenic mechanisms, it must lead to either increased total peripheral resistance (TPR) by inducing vasoconstriction or to increased cardiac output (CO), or both: This is due to the fact that blood pressure equals flow (CO) multiplied by resistance. The sympathetic nervous system and the renin-angiotensin-aldosterone system have received the most attention by investigators of the pathophysiology of hypertension, since both can increase CO and TPR. (Berkow 1996:413).

2.3.1 Sympathetic nervous system

Indirect and direct measures of sympathetic function have clearly shown that sympathetic activation characterises essential hypertension (Lanfranchi 1998).

Maneuvers to stimulate the sympathetic system raise blood pressure, usually more in hypertensive or prehypertensive patients than in normotensives. Whether this hyperesponsiveness resides in the sympathetic nervous system itself, or in the myocardium and vascular smooth muscle that it innervates, is not known. A high resting pulse rate, which can be a manifestation of increased sympathetic nervous system activity, is a well known predictor of subsequent hypertension. Some, but by no means all, hypertensives have a higher than normal circulating plasma catecholamines at rest. (Berkow 1996:414).

2.3.2 The renin-angiotensin-aldosterone system

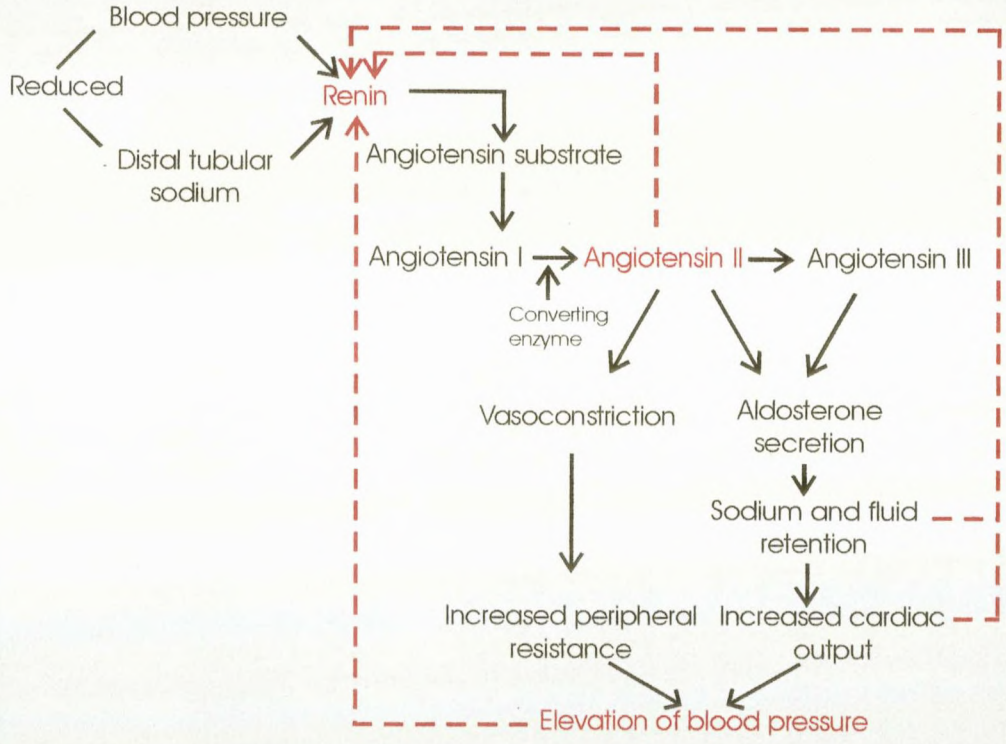


Figure 2-2. The role of renin-angiotensin system in regulation of blood pressure. Solid lines represent positive interactions; broken lines show negative interactions or feedback inhibition. (Kumar, et al. 1992:460).

The juxtaglomerular apparatus (JGA) is involved in volume and pressure regulation. Renin, a proteolytic enzyme formed in the granules of JGA cells, catalyses the conversion of

angiotensin (a plasma protein) to angiotensin I, a decapeptide. This is cleaved by a converting enzyme, mainly in the lung but also in the kidney and brain, to an octapeptide, angiotensin II, which is a potent vasoconstrictor and also stimulates release of aldosterone. (Berkow 1996:414).

Renin secretion is controlled by at least four mechanisms that are not mutually exclusive:

1. A renal vascular receptor that apparently responds to changes in tension in the afferent arteriolar wall.
2. A macular densa receptor that appears to detect changes in the delivery rate or concentration of NaCl in the distal tubule.
3. A negative feedback effect of circulating angiotensin on renin secretion.
4. The sympathetic nervous system which stimulates renin secretion via the renal nerve mediated by β receptors. (Berkow 1996:414).

2.3.3. The Mosaic Theory

The mosaic theory states that recruitment of multiple factors sustains elevated blood pressure even though an aberration of only one was initially responsible, e.g. the interaction between the sympathetic nervous system and the renin-agiotensin-aldosternone system. Sympathetic inervation of the JGA in the kidneys releases renin to stimulate angiotensin II which stimulates the autonomic centres in the brain to increase sympathetic discharge. Angiotensin also stimulates production of aldosterone, which leads to Na retention. An excess of intercellular Na enhances the reactivity of vascular smooth muscle to sympathetic stimulation. This is why it is so difficult to separate the relative roles of the renin-angiotensin-aldosterone system and the autonomic nervous system in the genesis of primary

hypertension. Other factors also become involved in hypertension and add to the cycle, increasing the hypertension. (Berkow 1996:414).

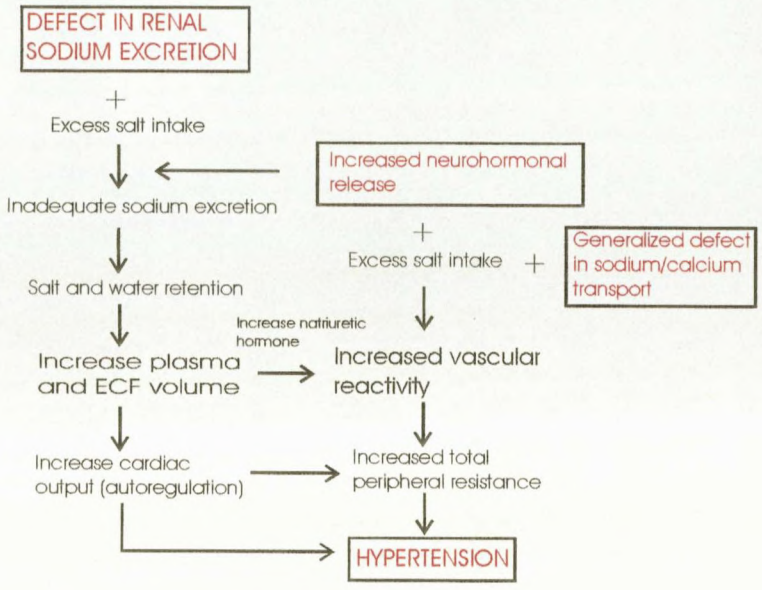


Figure 2-3 Hypothetical scheme for pathogenesis of essential hypertension, implicating genetic defects in renal sodium excretion, or in sodium/calcium transport, or causing increased neurohormonal release – coupled with excess output and increased cardiac output and increased total peripheral resistance contribute to the hypertension. (Kumar, et al. 1992:461).

2.3.4 Miscellaneous factors

Sometimes there is abnormal Na transport across the cell wall due to a defect in or inhibition of the Na-K pump (Na^+ - K^+ ATPase) or because of increased permeability to the Na^+. The net result is increased intracellular Na, which makes the cell more sensitive to sympathetic stimulation. Since Ca follows Na, it is postulated that the accumulation of intracellular Ca is responsible for the increased sensitivity. Na^+ - K^+ ATPase may also be responsible for

pumping norepinephrine back into the sympathetic neurones to inactivate it. Thus , inhibition of this mechanism could conceivably enhance the effect of norepinephrine. Defects in Na transport have been described in normotensive children of hypertensive parents. (Berkow 1996:415).

Hypertension results from the deficiency of a vasodilator substance rather than an excess of vasoconstrictor (e.g. Angiotensin or norepinephrine), (Berkow 1996:415).

Diet: Excessive dietary salt in conjunction with inadequate dietary potassium is a major contributor to hypertension. It has also been found that dietary deficiencies in calcium, magnesium, essential fatty acids and vitamin C all contribute to increased blood pressure and that increased consumption of sugar, caffeine and alcohol are associated with hypertension. (Murray & Pizzorno 1995:9-10).

Lifestyle: Smoking, obesity, stress and a sedentary lifestyle all contribute to the development of hypertension (Murray & Pizzorno 1995:9-10).

Environment: Cardiovascular diseases are influenced in each area by the unique physical environmental features of that environment especially the water supply and the presence or absence of ground metals and minerals (Wiesner 1992:88-90). Exposure to heavy metals such as lead and cadmium increase blood pressure (Murray & Pizzorno 1995:9-10).

2.3.5 Secondary Hypertension

Secondary hypertension is associated with bilateral renal parenchyma disease or with such potentially curable disorders as pheochomocytoma, Cushing's syndrome, primary

aldosteronism, hyperthyroidism, myxedema, coarctation of the aorta, renal vascular disease and unilateral renal disease. It may also be associated with the use of oral contraceptives and ingestion of alcohol. (Berkow 1996:415

2.4 Symptoms and signs

Primary hypertension is asymptomatic until complications develop. The symptoms and signs are non-specific and arise from the target organs; they are not pathognomic for hypertension since identical symptoms and signs can develop in normotensives. Dizziness, flushed face, headache, fatigue, epistaxis, and nervousness are not caused by uncomplicated hypertension. Complications include left ventricular failure, atherosclerotic heart disease, retinal haemorrhages, exudates, papilledema, and vascular accidents, cerebrovascular insufficiency with or without stroke and renal failure. Hypertensive encephalopathy due to severe hypertension and cerebral oedema is encountered only in hypertensive patients. (Berkow 1996:417).

A fourth heart sound and broad notched P-wave abnormalities on the ECG are among the earliest signs of hypertensive heart disease. Echocardiography and X-ray evidence of left ventricular hypertrophy may occur later. Aortic dissection or leaking aneurysm of the aorta may be the first sign of untreated hypertension or may complicate untreated hypertension. Polyuria, nocturia, diminished renal concentrating ability, proteinuria, microhematuria, cylindruria and N^+ retention are late manifestations of arteriolar nephrosclerosis. (Berkow 1996:417).

2.5 Diagnosis

Diagnosis of primary hypertension depends on demonstrating that systolic and diastolic blood pressure are usually but not necessarily always higher than the normal, as well as excluding secondary causes. At least two blood pressure determinations should be made on three separate days before labelling a patient hypertensive. (Olendorf & Jeryan, 1999:1523). For patients in the low hypertensive range and especially patients with markedly labile blood pressures, more than this minimum number of determinations is desirable. The upper limit of normal blood pressure in adults is 140/90 mm Hg, being much lower for infants and children. A somewhat higher limit, especially for systolic pressure, is acceptable for patients 60 years and older. Sporadic higher levels for patients who have been resting for more than five minutes suggests an unusual liability of blood pressure which may precede sustained hypertension. (Berkow 1996:417). Office blood pressure is often higher than that obtained at home or by ambulatory monitoring. "Office" or "white coat" hypertension refers to blood pressure that is consistently elevated in the office but otherwise normal. In a recent study into the differences between white coat and sustained hypertensives there was found to be no significant differences in the cardiovascular risk or morbidity profile between the two. (Abasolo, et al. 1999).

The more severe the hypertension and the younger the patient, the more extensive evaluations should be. Rapid sequence IYU, renal scinitigraphy, chest x-ray, screening tests for pheochomocytoma and renin-sodium profiling are not necessarily routinely done. (Berkow 1996:417).

Pheochromocytoma secretes catecholamines, which besides elevating blood pressure, usually produces symptoms (various combinations of headaches, palpitations, trachycardia,

excessive perspiration, tremor and pallor) that should alert the physician to this possibility. (Berkow 1996:417).

Hypokalemia, not due to diuretics, should suggest primary aldosteronism. Proteinuria, cylinduria, or microheamaturia with or without N retention early in the course of hypertension is strong evidence of an underlying primary renal disease. Absent or markedly reduced and delayed femoral arterial pulsations in a hypertensive patient of less than thirty years old is presumptive evidence of coarctation of the aorta. Cushing's syndrome, collagen disease, toxaemia of pregnancy, acute porphyria, hyperthyroidism, myxedema, agromegally, and some CNS disorders must be also excluded, along with aldosteronism. (Berkow 1996:418).

2.6 Prognosis

Although there is no cure for hypertension, it can be well controlled with proper treatment. An untreated hypertensive patient is at great risk of developing disabling or fatal left ventricular failure, myocardial infarction, cerebral haemorrhage or infarction, or renal failure at an early age. (Olendorf & Jeryan 1999:1523).

Hypertension in the most important risk factor predisposing to stroke or renal failure at an early age. It is one of the three important risk factors predisposing to coronary athersclerosis. The higher the blood pressure and the more severe the changes, the worse is the prognosis. Fewer than 5% of patients with malignant hypertension characterised by papilledema and less than 10% of patients with changes to the fundus survive one year without treatment. Effective control of hypertension will prevent or forestall most complications and will prolong life in patients whose diastolic blood pressure is greater than 90mm Hg. Coronary heart disease is the most common cause of death among treated hypertensive patients. Systolic

blood pressure is a more important predictor of fatal and non-fatal cardiovascular events than diastolic blood pressure. (Berkow 1996:418).

Because anti-hypertensive medicines control high blood pressure and do not cure it, patients must continue taking the medications to maintain a reduced blood pressure and avoid complications (Olendorf & Jeryan 1999:1523).

2.7 Hypertension in the South African context

Hypertension affects some 6,5 million people in South Africa necessitating years of drug treatment (Randeree et al., 1995). This high prevalence of hypertension amongst all ethnic groups in South Africa with a concomitant increased risk for coronary heart disease and stroke is a major cause for concern (Smith et al., 1987). It has further been established that hypertension is more common in black people and also has a higher prevalence in the urban population (Kuteyi, 1998). Akinkugbe (1969) found that the incidence of hypertension was significantly higher in the urban African male than the rural African male in Western Nigeria.

In Durban the prevalence is higher in the urban Zulu as compared to the urban white and Indian populations (Seedat 1995). The incidence of hypertension was also found to be high in Rural Zululand where a prevalence of 32% was found (Ellis 1995:6-7). In the U.S.A. the prevalence of hypertension in black adults is estimated to be about 38% while in white adults it is about 29%. There is also subsequently a proportionately higher mortality rate among black adults (Berkow 1996:415). In South Africa, a high Hypertension associated mortality rate is also seen in the coloured population as well as the black population (Steyn 1990).

It has been found that diagnosed hypertension is often untreated or poorly treated particularly in developing countries as a result of the largely asymptomatic nature of the disease. Poverty, ignorance, high illiteracy rate and other socio-economic factors like accessibility of treatment and poor compliance also play a large role in the non-management of this disease (Kuteyi, 1998). Once started, the treatment will be for many years, if not for the rest of the patient's life, and intelligent co-operation in treatment is rare, particularly where there are no signs of complications (Arinkugbe, 1969). It was recently shown that there was inadequate detection, treatment and control of hypertension in a sample drawn from the coloured population in the Cape Peninsula (Smith et al., 1987). Even in USA, it is estimated that only 38% of the 58 million hypertensive patients are adequately controlled and that up to 20% are not aware they have hypertension (Berkow, 1996:415).

A study across seven different countries confirmed that although the increase in relative risk of death from coronary heart disease for a given increase in blood pressure was similar, the absolute risk at a given blood pressure varied considerably between the different countries (van der Hoogen, et al. 2000). The side effects of hypertension are especially significant in South Africa as it has been shown that Black hypertensives experience greater target organ damage (cardiovascular and renal) at any level of blood pressure than do non-blacks at similar levels (Naidoo et al., 1995). There is a frequent association of hypertension with metabolic abnormalities like insulin resistant diabetes mellitus, which has a high incidence in black hypertensives (Wing, et al. 1995). The hypertension related death rate especially related to cerebovascular events and hypertensive heart disease in black hypertensives is higher than their white counter parts. This could in part be due to a higher pressure load in Africans who show a more sustained blood pressure elevation throughout 24 hours with less nocturnal decline. Another factor putting the Africans at a greater risk is a greater degree of

left ventricular hypertrophy which is the strongest predictor of morbid cardiovascular events. (Sareli & Strugo. 1995).

All of these well documented clinical observations indicate that Black hypertensives constitute a high risk for target organ damage and therefore therapy must be aimed at not only lowering blood pressure but also conferring target organ protection (Sareli & Strugo. 1995).

2.8 Treatment and management of hypertension

There is no cure for primary hypertension, but therapy and lifestyle modifications can modify its course (Keryn & Lane 1997:117).

2.8.1 Non-pharmacologic therapy

Lifestyle modification is the first important step in the management of mild hypertension (stage 1) (Ellis 1995:23).

Simple non-medical intervention includes cessation of smoking, decreasing salt intake, eating fruit and fiber, losing weight, exercising, coping better with stress and decreasing alcohol consumption (Ellis 1995:23).

More severe dietary restrictions should be imposed to control diabetes mellitus, obesity or blood lipid abnormalities. Weight reduction to ideal levels, modest dietary Na restriction (< 2 gm/day of Na), curtailment of alcohol consumption to < 1 oz of ethanol daily and prudent exercise should be encouraged. (Berkow 1996:419). Werbach (1995:136-138) states that

overweight hypertensives that lose weight can achieve a substantial reduction in blood pressure.

It has been noted epidemiologically that vegetarians have lower blood pressures than the general population (Villiers & Serfontein 1989:134-135). Many features of this diet make it ideal; it has a high fibre content, high potassium and magnesium content as well a low content of fat and cholesterol. For this reason either a vegetarian diet or a diet low in animal protein is recommended and high fibre is recommended. (Werbach 1995:137).

If ones diet is deficient in substances whose deficiency has been linked to hypertension (e.g. Ca and Mg), supplementation of these substances can lower blood pressure (Balch, 1997:320-324).

2.8.2 Allopathic treatment of hypertension

If the hypertension continues to progress to moderate (stage 2) and is not controlled by nonpharmacologic therapy it becomes necessary to introduce drugs in a stepwise manner until the hypertension has been controlled. The goal of therapy should be to reduce blood pressure to normal (<140/90 mm Hg) or as nearly as possible that the patient and his cardiovascular system can tolerate (Berkow, 1996:419).

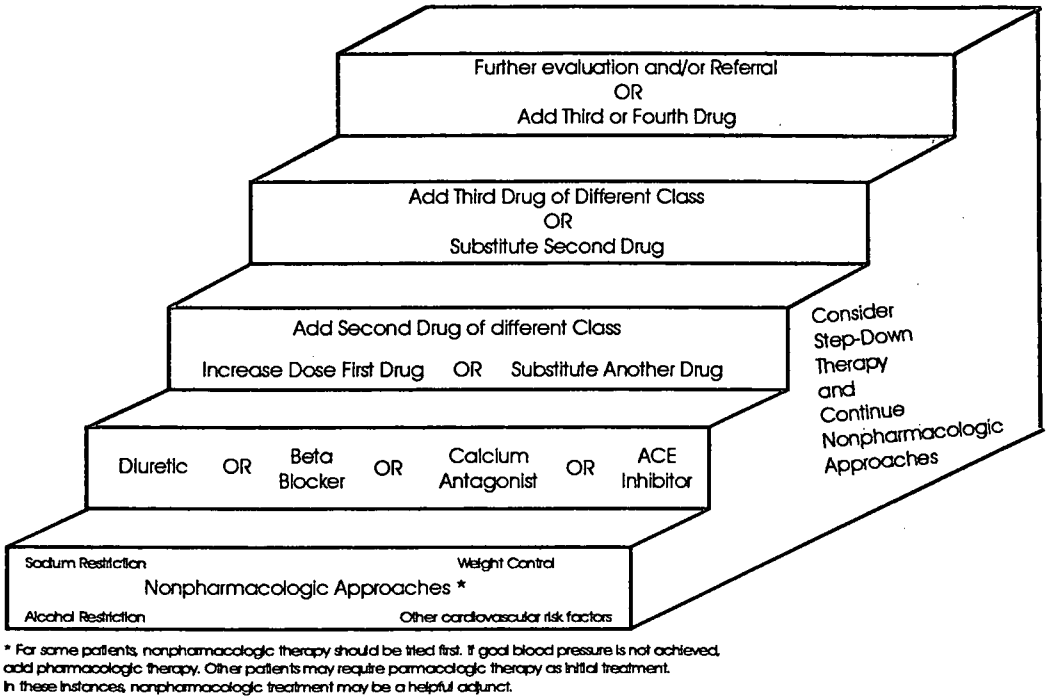


Figure 2-4 Individualized step care therapy for hypertension. (Seedat 1990).

A thiazide diuretic is introduced first in mild to moderate hypertension (Seedat 1990). The disadvantages of diuretics include sexual dysfunction in males and metabolic side effects. Thiazide diuretics can also precipitate diabetes or aggravate pre-existing diabetes whilst diuretic-induced hyperuricemia can also lead to clinical gout (Berkow 1996:422). Beta-blockers follow in an attempt to control hypertension (Seedat 1990). They can induce CNS side effects like sleep disturbance, fatigue, lethargy, sexual dysfunction in males and metabolic side-effects (Berkow 1996:423). At this moderate level of hypertension calcium (Ca) antagonists can also be used as a mono-therapy (Seedat 1990). They are potent peripheral vasodilators and reduce blood pressure by decreasing total peripheral resistance, but can cause headaches, dizziness, asthenia and flushing (Berkow 1996:425). Angiotensin converting enzyme (ACE) inhibitors are the last in the first step of treatment. These are

vasodilators that reduce blood pressure without inciting reflex trachycardia (Seedat 1990). ACE inhibitors can cause rashes, coughs, angioneurotic oedema and hyperkalemia (Berkow 1996:424).

As the hypertension becomes severe (stage 3) or very severe (stage 4) it is necessary to add to one of the drugs already mentioned another of a different class, or to increase the dose of the first drug. If this is still not sufficient to control the hypertension a third drug will be added and eventually a fourth drug in an attempt to control the blood pressure. (Seedat 1990).

Recently, the effectiveness of many of these traditional first line drug treatments is being questioned. Sareli and Strugo (1995) believe that the current recommendation to initiate therapy with a thiazide diuretic is unlikely to control blood pressure as a monotherapy or to confer optimal end organ protection. In a recent single drug trial, the mean conventional baseline blood pressure was 147/100mmHg. It was found that only 40% of patients receiving hyrochlorothiazide showed a reduction in diastolic blood pressure to below 95mmHg in one year. Similarly, in another trial a group of nineteen black South African hypertensives with a mean pre-treatment blood pressure of 152/99mmHg, showed only a modest reduction of the mean to 144/93mmHg. (Sarlei & Strugo, 1995.). Smith et al (1987) stated that control of only half of the patients on treatment is achievable which is considered a good result.

Allopathic drug treatment is essential in many cases of hypertension, especially in severe to very severe hypertension, but the many side effects of this treatment justify the search for a natural alternative that is safe and without side effects.

2.8.2 Homoeopathic treatment of Hypertension

Practitioners of Homoeopathy around the world have been treating patients for more than 200 years according to the principles of Homoeopathy laid down by the founder of Homoeopathy Dr Sammuel Hahnemann. The Homoeopathic method of potentizing medicines increases their healing potential. According to Dr Hahnemann (1996: 67-70), the smallest dose of medicine dynamised, in the appropriate case of disease, can be more curative by far than a large dose of the same medicine.

The Homoeopathic prescription for hypertension is best found when tailored to the individual constitutional make up of the patient (Casserley 1998). Homoeopathically many substances can be used to treat hypertension, the most frequently used ones include, Tabacum, Nux vomica, Veratrum viride, Ephedra, Secale, Cocculus, Digitalis, Natrum muriaticum, Barium, Plumbum, Adrenalin and Pituitrin (Gutman, 1995). Jouanny et al (1994: 225-228), also includes Sulphur, Lachesis, Aurum muriaticum, Ignatia, Strontium carbonicum, Glonoinum, Aconitum napellus, Kalium carbonicum, Natrum sulphuricum, Secale cornutum, Phosphorus and Arnica montana as the most frequently used Homoeopathic remedies. The Synthesis Homoeopathic Repertory itself lists 100 remedies under the heading Generals- Hypertension which have been shown to have a positive effect on Hypertension (Schroyens, 1995:1616). According to Jouanny et al. (1994:225) Homoeopathy treats hypertension effectively when it is moderate (stage 2) and not too advanced.

For this study, it was decided to use the 6CH potency, because low potencies are related to the physical level, more to specific organs or systems without necessarily having to match the subtler internal hierarchy of processes, energies and biological intelligence (Hershoff 1996:314). The 6CH potency is capable of causing definite physiological response (Roy,

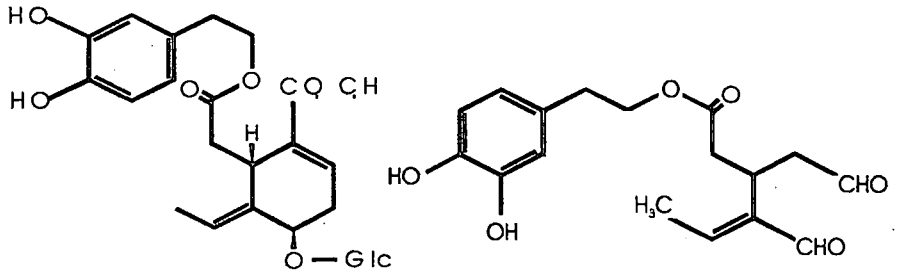
1994:66). For chronic conditions like hypertension it is advisable to take 6CH three times a day (Lockie & Geddes, 1992:25). Dr Lockie also recommends the taking of specific remedies for the heart and the circulatory system in 6CH potency (Lockie, 1998:197).

2.8.4. Phytotherapeutic treatment of hypertension

A survey was conducted in Morocco by Ziyyat et al. (1997), in order to determine the main medicinal plants used in folk medicine to treat hypertension. Six hundred and twenty six patients were surveyed and it was found that of the only eighteen species of plant that are used for the control of blood pressure, Olea europea being one of the most used.

Olea europea is found in areas of Spain, Italy, Greece, France, Algeria, Tunis, California, and South Australia (Denston, 1951:513). In Europe, Olea europea is widely used as a hypotensive, vasodilator and tonic (Hutchings et al., 1996:235). The oil of the Olea europea is also used medically for its nutrient, demulcent and mildly laxative qualities (Reynolds, 1989:1596).

It is the evergreen leaves of the Olea europea that are employed for the treatment of hypertension (Maurice, 1993:217). In 1996 a clinical assay of Olea europea aqueous leaf extract was carried out on two groups of patients suffering with mild to moderate essential hypertension. Of the patients in the study, twelve had had no previous treatment for their hypertension whereas the other eighteen patients were already taking anti-hypertensive drugs. The treatment was based on Olea europea aqueous leaf extract and was given four times a day during the three-month trial. For all patients there was a statistically significant decrease of blood pressure ($p<0.001$). (Cherif, et al., 1996.)



Oleuropeln Oleacein

Figure 2-5 Chemical structure of Oleuropein and Oleacein (Van Wyk et al. 1997:180-181).

According to Van Wyk et al. (1997:180-181) oleuropein, which is found in the Olive leaves, lowers blood pressure by increasing coronary flow and oleacein, another chemical found in the leaf, inhibits the angiotensin-converting enzyme (ACE). Oleuropein also has spasmolytic qualities and 3,4- dihydroxyphenylethanol, which arises from the hydrolysis of oleuropein, is a calcium antagonist (Bruneton, 1995: 489).The two compounds, oleuropein and oleacein, therefore work in combination to produce the hypotensive effect (Van Wyk, et al., 1997:180-181). These effects are especially important because both calcium antagonists and ACE inhibitors induce a rise in GFR, renal plasma flow and renal sodium excretion which help to reduce blood pressure and are preferred agent is diabetic nephropathy (Naidoo et al. 1999). Weiss, cited by Maurice (1993:217) recommends the olive leaf extract for labile and medium-severe hypertension in a daily dose of 100ml of the liquid infusion.

In 1994, Fehri et al. conducted a study to determine the possible toxic affects of Olea europea aqueous leaf extract after repeated administrations in rats. They found that at doses many times more than the recommended dose the extract produced increase of weight, hypotension, hypoglycaemia and hypouricemia in the treated animals (Fehri et al. 1994). But in humans, the olive leaf extract has been studied as a treatment for hypertension with no record of toxic or other side effects (Finch, 1998:26).

2.8.5 Olea europea subsp africana and it's traditional uses.

There have been studies, as mentioned above, conducted on the effect of Olea europea leaf extract in hypertension with favourable results. There has been no clinical trial conducted on the Olea europea subsp. africana, either in mother tincture or in Homoeopathic potency. The Olea europea subsp. africana is, according to many experts, the ancestor of the Olea europea (Hutchings et al., 1996:235). The Olea europea subsp. africana is a well-known plant on the African subcontinent and its common names include Wild olive, Swartolienhout and umNqumo (Zulu) (Moll, 1992:293). Isadululambazo, isi adlulambazo, umsityana (Zulu) (Hutchings et al., 1996:235). UmNquma (Xohza) (Pooley, 1993:412); Wilde-olyfboom, umHlwathi (Zulu), motlhware (Tswana), motlhware (N. Sotho) (Palmer and Pitman, 1961:302).

The wild olive, with it's dense, round crown and grey-green foliage, is a familiar sight in the Karroo, the North west Cape, Bechuanaland and the Free State (Palmer & Pitman, 1961:304). Olea europea subsp. africana is found in a variety of habitats, usually on rocky hillsides or on stream banks (Van Wyk, & Van Wyk, 1997:346). This tree is drought-resistant and frost tolerant and is also one of the few species of trees to be found almost through-out South Africa in open woodland, among rock and in mountain ravines (Palgrave, 1977:758). This tree is also widespread in other countries as well as in southern Mozambique, the western part of Namibia and fairly large areas of Zimbabwe (Van Wyk, 1993:116).

The bark and leaves are used medicinally for headaches, respiratory and renal ailments (Pooley, 1993:412). Africans drink an infusion of fresh bark to relieve colic. They also use an infusion of the leaves as a lotion for both humans and animals, while a decoction provides a gargle for sore throats (Palgrave, 1977:759) and act as diuretics and anti-diarrhoeals (Van

Wyk, et al., 1997:180). Infusions of the leaves are also widely used as eye lotions. The Zulu culture use grated root and scraped bark decoctions for urinary and bladder infections and for headaches. Masai take heartwood teas as strengthening blood cleansers and the Tswana use the juice of the ripe fruit to soften corns (Hutchings et al., 1996:235).

Despite the long traditional use of the wild olive by the people of Southern Africa as mentioned above for many conditions, the only reference to its use as a hypotensive is based on it's close relationship to the Olea europea. It is therefore necessary to investigate its effects on hypertension in a clinical trial. Although Olea europea is thought to be derived from subsp. africana, the chemical constituents and biological properties may differ and hence there may be differences in its medicinal actions (Hutchings et al., 1996:235).

CHAPTER THREE

RESEARCH MATERIALS AND METHODS

3.1 Introduction

The screening of patients and collection of data for this study took place at various venues according to the availability of patients. These included the Natal Technikon Homoeopathic Clinic, P&O Stevedores Clinic, Clover Dairies Clinic, Health Wise Homoeopathic Clinic, Matushi, Silk Screen Manufactures and Atlas Pharmacy.

3.2 Advertising

Advertisements were placed in the local media, around the Technikon Natal grounds, on the premises of Clover Dairies, Health Wise Health Shop, P&O Stevedores, Matushi Manufacturers, Silk Screen manufacturers and Atlas Pharmacy. Members of the public were invited for free blood pressure assessments in an effort to find suitable candidates for the study. Patients who satisfied the selection criteria were invited to participate in the study and educated as to the nature of the study.

3.3. Selection criteria

3.3.1 Inclusion criteria:

Patients where included into the study if they had a blood pressure reading which put them into the classification of mild (stage 1) to moderate hypertension (stage 2), at three

successive measurements on three different occasions. Male and female patients between the ages of 20-60 years old were accepted into the study. Patients were advised to make no changes to their life-style or eating habits, and were not to start with any other medications while participating in this clinical trial.

3.3.2. Exclusion criteria:

Patients who were on any other type of drug treatment were excluded. Pregnant patients, patients suffering from congestive cardiac failure, drug induced hypertension, grade two or three heart block, active hepatic or renal disease, obstructive airway disease, diabetes mellitus, peripheral vascular disease and severe obesity were excluded.

Forty nine patients were accepted into the trial and were divided into three groups: group 1 was treated with Olea europea subsp africana 6CH, group 2 received a placebo of 20% Alcohol and group 3 was treated with Olea europea subsp africana aqueous leaf extract. The first treatment group received ten drops of Olea europea subsp. africana 6CH three times a day for 3 months. The second (placebo) group received ten drops of 20% Alcohol three times a day for three months. The third treatment group received ten drops of Olea europea subsp. africana tincture three times a day for 3 months treatment

The patients were assigned to the three groups randomly by an independent party, making the study a double blind placebo controlled study.

All the patients signed an informed consent form at the start of the trial. The patients were monitored at three to four weekly intervals. The monitoring consisted at the first consultation of an initial full medical case history and physical examination and the patients were

requested to keep their lifestyle consistent and not to make any changes. At the end of the first, second and third month the patient's blood pressure levels were monitored and recorded to evaluate any changes that may have taken place. The patients were advised on the taking of the medication at the start of the trial and educated on the importance of taking the medication constantly for the period of the research.

If at any time the patient's blood pressure rose more than 10 mmHg from their starting level or they started experiencing any adverse symptoms of hypertension, it was felt that it is detrimental to their health to continue with the study and they were referred for treatment.

3.4. Preparation of Medication

3.4.1 Olea europea subsp africana aqueous leaf extract

Olea europea subsp africana mother tincture was prepared by the Parceval Pharmaceutical company according to the clearly defined principles in the German Homoeopathic Pharmacopoeia (British Homoeopathic Association 1985:20-21). This, however, was slightly adjusted as there is no monograph for Olea europea subsp. africana and no record of it being used in this specific form. The concentration used was 0.4g of fresh plant material / ml in 30% m/m ethanol. According to Van Wyk et al. (1997:180-181) decoctions are often used and it was therefore decided to imitate the traditional preparation and decocted the extract for 30 min at 100 degrees Celsius under reflux. (Feiter, 1999.)

3.4.2 Olea europea subsp africana 6CH

Olea europea subsp. africana 6CH was prepared by a W. Last Pharmaceutical company in accordance with the clearly defined principles in the German Homoeopathic Pharmacopoeia (British Homoeopathic Association 1985:20-21).

3.5. Experimental method of blood pressure measurement.

The blood pressure was measured according to the method described by the American Heart Foundation (1967). As postural changes of blood pressure can occur, the patients were seated comfortably with their feet supported and the arm at the level of the heart. Systolic and diastolic blood pressure was measured on the right arm. The phase IV Korotkoff sounds were used to measure the systolic blood pressure and the disappearance of the sound will be used to indicate diastolic pressure. The blood pressure was measured three times with a five minute interval between measurements. These levels were recorded and the mean average calculated. (Gallery 1997).

3.6 Method of randomisation of the three test groups

Sixty pieces of paper where cut up by an independent party, the extra eleven allowed for patients who did not complete the treatment or dropped out of the study. Twenty had placebo written on them, twenty had tincture written on them and twenty had 6CH written on them. These pieces of paper where placed in a bowl and mixed up. The independent party then selected at random pieces of paper out of the bowl and the paper's contents recorded on a numbered piece of paper in the order that they where selected. This was then the order

in which they where given to the patents. The number for each bottle was recorded and the patient received the same numbered bottle for the duration of the research period.

3.7. Statistical method of data analyses.

There were three independent groups of 16, 16 and 17 patients each. As the sample size per group is small non-parametric statistical methods were used. There were 2 variables of study: Systolic and diastolic blood pressure levels. There where 4 readings for each variable of study.

3.7.1 Procedure 1: Comparison between 3 or more independent (unpaired) groups.

Groups 1,2 and 3 were compared to each other with regards to the two variables of study; the Kruskal-Wallis non-parametric Analysis of Variance (ANOVA) method was used. In each test, the null hypothesis states that there was no significant difference between groups means being compared with each other. The alternative hypothesis states that there will be a significant difference. (Fisher 1993:447).

3.7.1.1 Decision rule:

The null hypothesis is rejected at the α level of significance if $p <= \alpha/2$ where p is the observed significance level or P-value. Otherwise, the null hypothesis is accepted at the same level.

3.7.2 Procedure 2: Comparison between three or more related (paired) samples.

The Friedman's ANOVA method was used to compare related samples from groups 1, 2 and 3. In each test, the null hypothesis states that there will be no significant difference between the group means being compared, at the α level of significance. The alternative hypothesis states that at least two of the group means will differ significantly. (Fisher 1993:430.)

3.7.2.1 Decision rule:

The null hypothesis is rejected at the α level of significance if $p <= \alpha/2$ where p is the observed significance level of P-value. Otherwise, the null hypothesis will be accepted at the same level.

3.7.3 Procedure 3: Medians, averages and variances for each variable of study

These results were needed for power analysis and the construction of bar charts

3.7.4 Procedure 4: Comparison using bar charts

Selected visual summaries of analytical findings were given by use of barcharts to compare groups 1, 2 and 3 with respect to selected variables of interest. Median readings were used to make bar charts.

Statistical package SPSS was used for data entry and analysis.

CHAPTER FOUR

RESULTS

4.1 The criteria governing the admissibility of the data

For each visit three blood pressure readings where recorded with a short break in between each measurement. The mean of these three readings was used for statistical analysis.

4.2 Statistical analysis of the results

4.2.1 Kruskal-Wallis test

This test compares the three groups with each other. Refer to 3.5.1 for procedure.

	Group	N	Mean Rank
Sys 1	1.00	16	26.16
	2.00	16	26.38
	3.00	17	22.62
	Total	49	
Dia 1	1.00	16	22.44
	2.00	16	26.34
	3.00	17	26.15
	Total	49	
Sys 2	1.00	16	25.31
	2.00	16	27.56
	3.00	17	22.29
	Total	49	
Dia 2	1.00	16	22.44
	2.00	16	27.94
	3.00	17	24.65
	Total	49	
Sys 3	1.00	16	24.69
	2.00	16	27.81
	3.00	17	22.65
	Total	49	
Dia 3	1.00	16	21.75
	2.00	16	27.28
	3.00	17	25.91
	Total	49	
Sys 4	1.00	16	24.44
	2.00	16	29.06
	3.00	17	21.71
	Total	49	
Dia 4	1.00	16	23.34
	2.00	16	26.94
	3.00	17	24.74
	Total	49	

Table 4.1 Kruskal-Wallis Test Ranks

Test Statistics[a,b]

	Sys 1	Dia 1	Sys 2	Dia 2	Sys 3	Dia 3	Sys 4	Dia 4
Chi-Square	0.727	0.769	1.134	1.203	1.090	1.308	2.225	0.516
df	2	2	2	2	2	2	2	2
Asymp.sig. (P)	0.695	0.681	6.810	0.548	0.580	0.520	0.329	0.773

a. Kruskal-Wallis Test

b. Grouping Variable: group

Table 4.2 Kruskal-Wallis Test Statistics

4.2.2 Explanation of the results of the Kruskal-Wallis test

Ho: $\mu_1 = \mu_2 = \mu_3$

Ha: There is a significant difference between two or more of the groups

Decision Rule:

At the $\alpha = 0.05$ level of significance,

1. Reject the Ho if the p-value (Asymp. Sig.) < $^{\alpha}/_{2}$ (=0.025)
2. Accept Ha if p-value> α

Sys 1: p-value = 0.695 therefore accept Ho

Dia 1: p-value = 0.681 therefore accept Ho

Sys 2: p-value = 0.567 therefore accept Ho

Dia 2: p-value = 0.548 therefore accept Ho

Sys 3: p-value = 0.580 therefore accept Ho

Dia 3: p-value = 0.520 therefore accept Ho

The Kruskal-Wallis test therefore shows that there is no statistically significant difference between the three groups on any of the three systolic or diastolic readings.

4.2.3 Friedman test

This test compares the readings within each of the three groups. Refer to 3.5.2 for procedure.

Non-Parametric Tests Group 1 Systolic Readings

Descriptive statistics

	N	Mean	Std Deviation	Minimum	Maximum	Percentiles		
						25th	50th (Median)	75th
Sys 1_1	16	145.3542	13.9196	130.00	169.33	132.1667	141.6667	157.6667
Sys 1_2	16	140.2500	13.3308	122.67	164.67	129.6667	139.3333	150.0000
Sys 1_3	16	134.5000	14.6454	109.33	170.00	124.1667	134.0000	144.3333
Sys 1_4	16	133.9167	11.2134	111.33	150.00	126.3333	132.6667	143.6667

Friedman Test

Ranks

	Mean Rank
Sys 1_1	3.72
Sys 1_2	2.81
Sys 1_3	1.63
Sys 1_4	1.84

Test Statistics

N	16
Chi-Square	27.902
Df	3
Asymp. Sig.	0.000

a. Friedman Test

Table 4.3:Friedman test Group 1, systolic readings

Non-Parametric Tests Group 1 Diastolic Readings

Descriptive statistics

	N	Mean	Std Deviation	Minimum	Maximum	Percentiles		
						25th	50th (Median)	75th
Dia 1_1	16	91.9167	9.4685	70.67	109.33	87.3333	89.6667	100.1667
Dia 1_2	16	89.4583	6.2880	78.67	100.67	84.0000	89.6667	94.6667
Dia 1_3	16	86.1250	6.1257	74.00	96.00	80.0000	88.0000	90.6667
Dia 1_4	16	86.7917	6.6320	74.67	96.00	79.5000	89.0000	90.6667

Friedman Test

Ranks

	Mean Rank
Dia 1_1	3.22
Dia 1_2	3.03
Dia 1_3	1.94
Dia 1_4	1.81

Test Statistics

N	16
Chi-Square	16.26
Df	3
Asymp. Sig.	0.001

a. Friedman Test

Table 4.4: Friedman test Group 1, diastolic readings.

Non-Parametric Tests Group 2 Systolic Readings

Descriptive statistics

	N	Mean	Std Deviation	Minimum	Maximum	Percentiles		
						25th	50th (Median)	75th
Sys 2_1	16	142.7083	7.0067	133.33	160.00	137.8333	141.0000	146.0000
Sys 2_2	16	141.0417	6.9834	130.00	156.00	136.8333	139.3333	146.8333
Sys 2_3	16	135.5417	7.4584	120.67	150.67	130.6667	138.0000	141.0000
Sys 2_4	16	136.9167	6.5631	120.67	148.67	131.8333	138.0000	141.1667

Friedman Test

Ranks

	Mean Rank
Sys 2_1	3.5
Sys 2_2	2.91
Sys 2_3	1.88
Sys 2_4	1.72

Test Statistics

N	16
Chi-Square	21.604
df	3
Asymp. Sig.	0.000

a. Friedman Test

Table 4.5: Friedman test Group 2, systolic readings.

Non-Parametric Tests Group 2 Diastolic Readings

Descriptive statistics

	N	Mean	Std Deviation	Minimum	Maximum	Percentiles		
						25th	50th (Median)	75th
Dia 2_1	16	93.2917	6.4749	80.67	102.67	89.3333	95.3333	99.1667
Dia 2_2	16	92.7917	7.0521	78.67	104.00	88.3333	91.3333	99.8333
Dia 2_3	16	89.5417	7.0205	76.67	100.67	85.6667	88.6667	94.5000
Dia 2_4	16	90.2500	7.5851	76.67	102.00	86.6667	89.0000	97.3333

Friedman Test

Ranks

	Mean Rank
Dia 2_1	3.16
Dia 2_2	2.94
Dia 2_3	1.91
Dia 2_4	2.00

Test Statistics

N	16
Chi-Square	12.54
df	3
Asymp. Sig.	0.006

a. Friedman Test

Table 4.6: Friedman test Group 2, diastolic readings.

Non-Parametric Tests Group 3 Systolic Readings

Descriptive statistics

	N	Mean	Std Deviation	Minimum	Maximum	Percentiles		
						25th	50th (Median)	75th
Sys 3_1	17	142.2745	11.8628	130.67	172.00	132.6667	139.3333	148.0000
Sys 3_2	17	137.3725	14.8408	116.00	163.33	124.6667	135.3333	147.6667
Sys 3_3	17	132.5490	13.4582	114.00	167.33	120.0000	134.0000	140.0000
Sys 3_4	17	133.0980	12.4498	115.33	166.00	121.0000	133.3333	139.0000

Friedman Test

Ranks

	Mean Rank
Sys 3_1	3.56
Sys 3_2	2.47
Sys 3_3	2.15
Sys 3_4	1.82

Test Statistics

N	17
Chi-Square	18.018
df	3
Asymp. Sig.	0.000

a. Friedman Test

Table 4.7: Friedman test Group 3, systolic readings.

Non-Parametric Tests Group 3 Diastolic Readings

Descriptive statistics

	N	Mean	Std Deviation	Minimum	Maximum	Percentiles		
						25th	50th (Median)	75th
Dia 3_1	17	94.1961	11.3352	74.00	118.67	89.3333	93.3333	100.3333
Dia 3_2	17	91.6078	13.1847	68.67	114.67	81.0000	90.0000	102.0000
Dia 3_3	17	88.9804	10.1629	68.00	111.33	81.3333	89.3333	95.0000
Dia 3_4	17	88.5098	10.3857	76.67	109.33	80.0000	86.6667	98.3333

Friedman Test

Ranks

	Mean Rank
Dia 3_1	3.29
Dia 3_2	2.94
Dia 3_3	2.15
Dia 3_4	1.62

Test Statistics

N	17
Chi-Square	18.164
df	3
Asymp. Sig.	0.000

a. Friedman Test

Table 4.8: Friedman test Group 3, diastolic readings.

4.2.4 Explanation of the results of the Friedman test.

Ho: $\mu_1 = \mu_2 = \mu_3$

Ha: There is a significant difference between two or more of the groups

Decision Rule:

At the $\alpha = 0.05$ level of significance,

1. Reject the Ho if the p-value (Asymp. Sig.) < $^{\alpha}/_{2}$ (=0.025)
2. Accept Ha if p-value> α

Group 1 systolic reading: p-value = 0.000 therefore reject Ho accept Ha

Group 1 diastolic reading: p-value = 0.001 therefore reject Ho and accept Ha

Group 2 systolic reading: p-value = 0.000 therefore reject Ho accept Ha

Group 2 diastolic reading: p-value = 0.006 therefore reject Ho and accept Ha

Group 3 systolic reading: p-value = 0.000 therefore reject Ho accept Ha

Group 3 diastolic reading: p-value = 0.000 therefore reject Ho and accept Ha

These results show that there is a statistically significant change within all of the three groups during the study period.

4.2.5. Comparison using bar charts

Selected visual summaries of the analytical findings are given by use of bar charts to compare groups 1, 2 and 3 with respect to the variables of interest. Median readings have been used to make bar charts. Refer to 3.5.4.

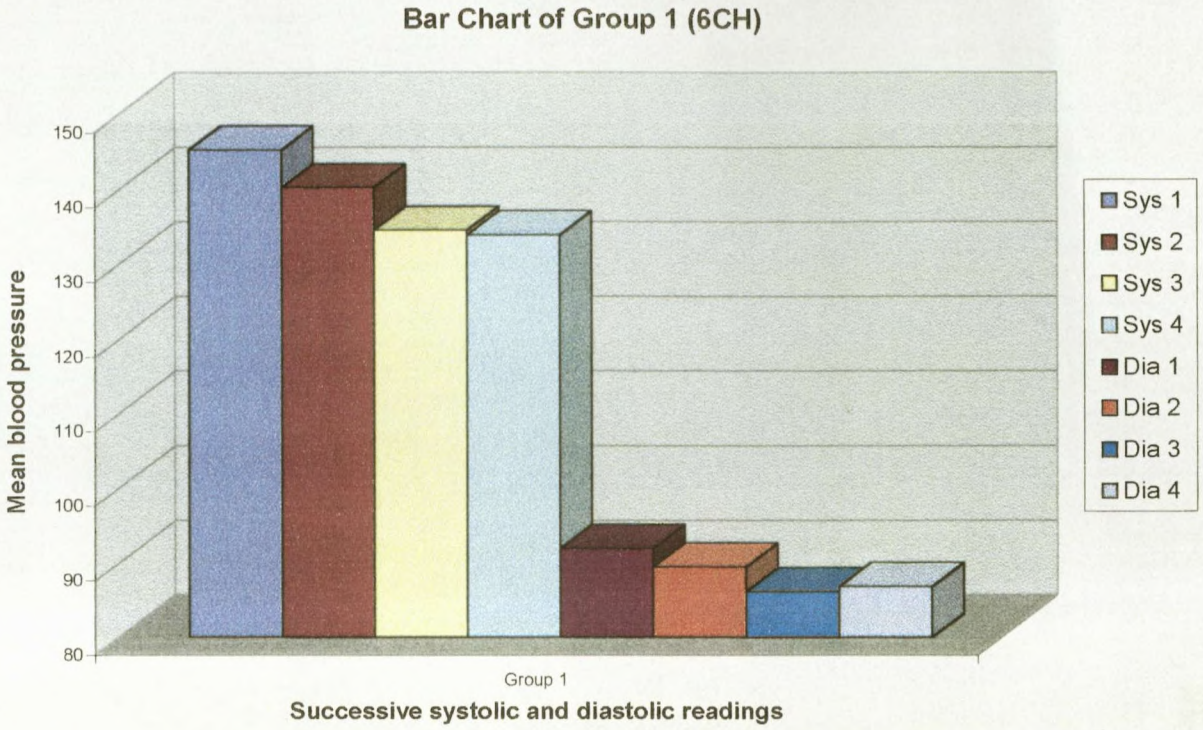


Figure 4.1: Bar chart of successive systolic and diastolic readings for Group 1 (6CH).

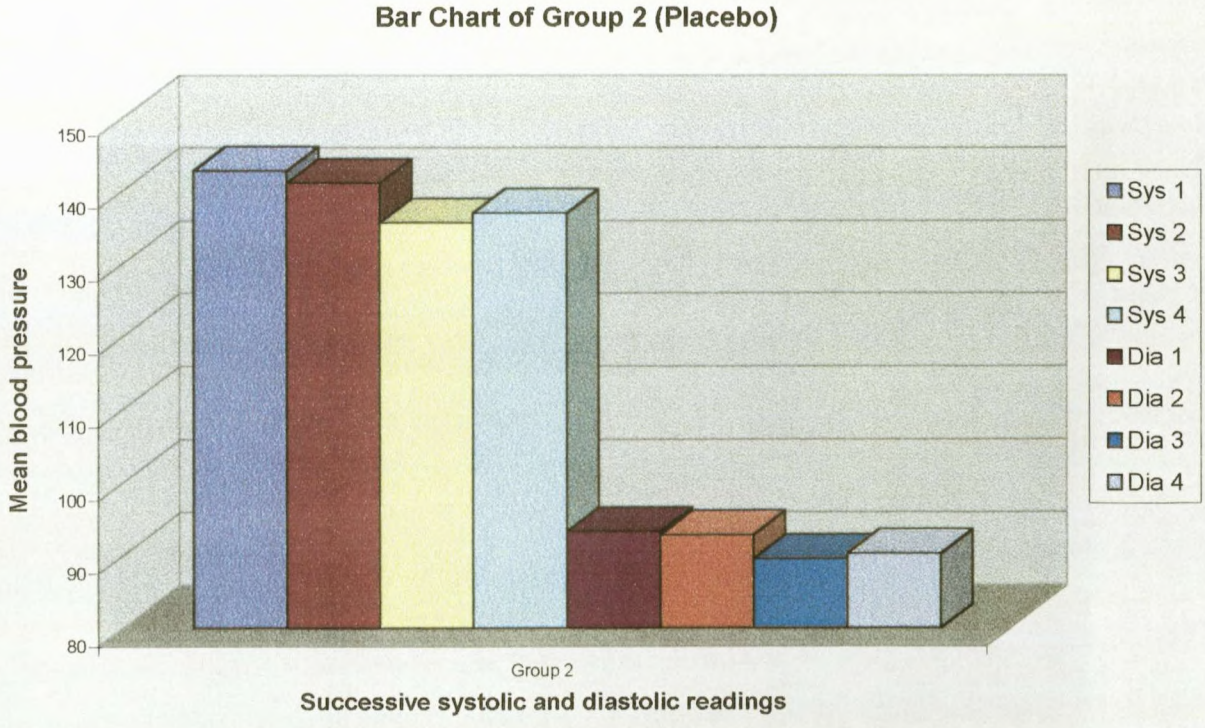


Figure 4.2: Bar chart of successive systolic and diastolic readings for Group 2 (Placebo).

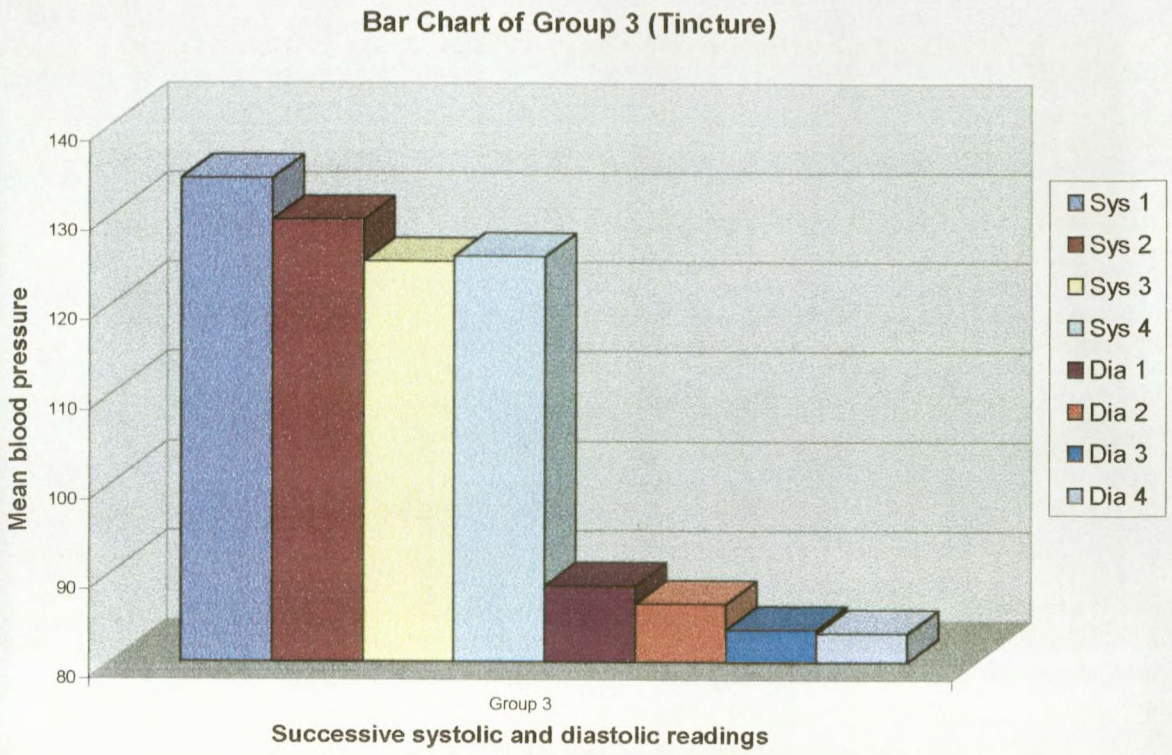


Figure 4.3: Bar chart of successive systolic and diastolic readings for Group 3 (Tincture).

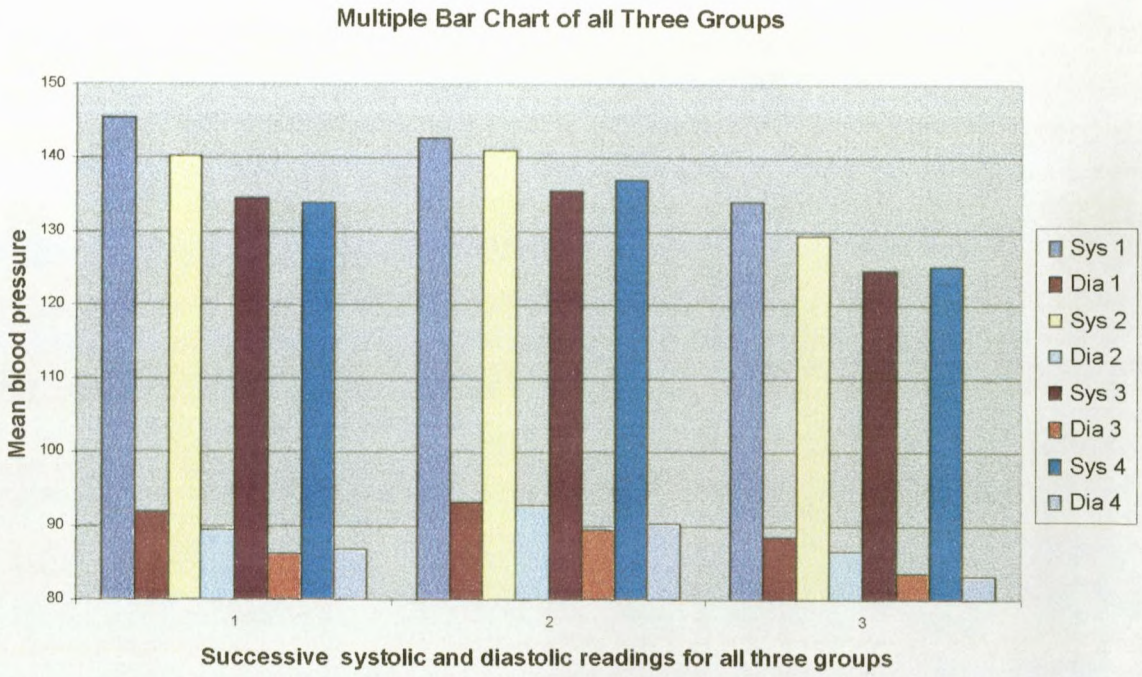


Figure 4.4: Multiple bar chart of successive systolic and diastolic readings for all three groups.

CHAPTER FIVE

DISCUSSION

5.1 Introduction

The results of the research clearly indicate that there was a significant decrease in blood pressure within all three groups during the trial period. The Kruskal-Wallis test showed that there was no statistically significant inter-group difference between the three. The Friedman test showed that there was a statistically significant intra-group change within the research period (the mean blood pressure readings of all three groups decreased). By use of visual analysis of the changes in the three groups both individually and together it can be seen that Group 1 and Group 3 had a greater degree of change than Group 3.

5.2 The Kruskal-Wallis test

This is a non-parametric test equivalent to one way ANOVA and tests whether several independent samples are from the same population. It was shown, by using this test that statistically the three groups showed similar tendencies during the research period and that statistically they were similar ($\alpha = 0.05$). Thus the placebo group (2) showed changes that were statistically similar to the 6CH group (1) and the tincture group (3).

5.3 The Friedman test

This tests the null hypothesis that "k" related variables come from the same population. For each case the variables are ranked from 1 to k and the test statistic is based on these ranks. The null hypothesis states that there will be no change in the three groups during the research period. For the purposes of this research study, the Friedman test rejects this hypothesis. In all cases there was shown to be a statically significant change within the groups ($\alpha = 0.05$) i.e. groups 1, 2 and 3 all showed a reduction in mean blood pressure levels over the period of the research study.

5.4. Visual analysis by means of bar charts.

Bar charts of the three groups gave a visual comparison of the mean values of each reading i.e. the difference in the four systolic readings and the four diastolic readings of each group.

5.4.1 Analysis of Group 1 (6CH)

The greatest improvement occurred between visit one and three showing a 10mmHg systolic and 6mmHg diastolic change. There was a further 1mmHg decrease in the systolic reading and a slight increase of 1mmHg in the diastolic reading between the third and fourth visit. This showed a total decrease in the blood pressure of 11 mmHg systolic and 5mmHg. This is a significant decrease as it means that the patents that were classified as mildly hypertensive would drop to the category of high normal blood pressure (Lane, 1997:116).

5.4.2 Analysis of Group 2 (placebo)

A small decrease in systolic pressure of 2mmHg was seen between visit one and two. There was a larger decease between visit two and three (5mmHg systolic and 3mmHg diastolic) but then an increase between visit three and four (increase of 1 mmHg systolic and no change in diastolic). This is not as much a change as Group 1 or 3 and is not enough to bring both the systolic and diastolic readings to a level that is considered high normal blood pressure (Lane, 1997:116).

5.4.3 Analysis of Group 3 (tincture)

This showed that the greatest decrease was between visit one and three of 9mmHg systolic and 8mmHg diastolic). Between the third and fourth visit there was only a small change in blood pressure (systolic remained the same and diastolic decreased by 1mmHg). This again is a significant reduction in mean blood pressure and would take a patient that was categorised as high normal blood pressure to normal blood pressure levels (Lane 1997:116).

5.4.4. Analysis of multiple bar chart of all three groups.

This showed that all the groups showed a decrease in mean blood pressure with Group 1 having the greatest systolic drop of 11mmHg systolic and 5mmHg diastolic, with Group 3 having the second largest drop of 9mmHg systolic and 5mmHg diastolic. Group 2 showed the smallest drop with a 6mmHg drop in systolic and a 3mmHg drop in diastolic mean blood pressure.

5.5. Argument

Although there was no statistically significant inter-group difference there was a statistically significant intra-group change in all three of the research groups. It can be seen by visual analysis that the placebo group changed the least of the three groups.

Blood pressure can be affected by numerous factors. These include eating habits, consumption of excess salt, sugar, caffeine alcohol, smoking, obesity and stress (Murray & Pizzorno 1995:9-10). Due to the fact that many test subjects were unaware that they were suffering from hypertension before being screened for this study, we can assume that, even though patients were instructed not to modify any of these dietary and lifestyle factors, they might have done so on there own accord.

As most of the change took place between the first and third visits in all groups, two months can be seen as a suitable time frame to determine the effectiveness of any blood pressure treatment. It may also be that the medication had reached its limits of activity at this point and if further reduction is required it would be wise to add another remedy at this point.

It is also interesting that the 6CH potency of Olea europea subsp africana was slightly more effective than the mother tincture confirming Dr Hahnemann's theory that a small dose of a substance can be more curative than a large dose of the same medicine (Hahnemann, 1996:67-70).

This study also confirms that the 6CH potency is capable of having a definite physiological effect without necessarily having to match the subtler internal hierarchy of processes energies and biological intelligence (Hershoff, 1996:314).

5.6. Summary

Although all the groups showed improvement it can be seen by visual analysis of the results that the placebo group (2) showed the smallest improvement of the three groups. This may be explained by changes in lifestyle factors of previously undiagnosed patients, and a greater awareness of their condition combined with a placebo effect. All the groups showed a statistically significant improvement which was more marked in Groups 1 (6CH) and 3 (tincture).

CHAPTER 6

CONCLUSIONS AND RECOMMENDATIONS

6.1 Conclusions

It has been concluded from this research study that Olea europea subsp africana has a role to play in the management of mild to moderate hypertension and should be expected to bring about a 10mmHg decrease in the systolic and a 5mmHg decrease in the diastolic blood pressure level.

Further we can conclude that the 6CH Olea europea subsp africana is as effective as the tincture in reducing high blood pressure levels.

Olea europea subsp africana recommended dosage can be suggested at 15-20 drops three times a day in both tincture and 6CH.

6.2 Recommendations

Olea europea subsp africana may be used as treatment for mild to moderate hypertension and will possibly be more effective when used as part of a holistic Homoeopathic treatment program, which takes into consideration the patient's similimum.

Further research is needed to clarify the action of Olea europea subsp africana:

1. Increasing or decreasing the dosage in drop amounts.

2. Increasing or decreasing the frequency of the dosage.
3. Study the effect of other potency levels.
4. Determine the active constituents of Olea europea subsp africana and the relative quantities of these as compared with Olea europea.

REFERENCES

Abasolo, G., Aizpuru, B., Mar, J., Ruiz, D., Domingo, R. 1999. White coat and non-dipper hypertension in patients recently diagnosed with mild hypertension. Aten Primaria. **23**(6): 332-338.

Akinkugbe, O.O. 1969. Anti hypertensive treatment in the African context. The Practitioner. **202**(202): 549-552.

Akinkugbe, O.O. 1969. Arterial pressures in rural and urban populations in Nigeria. British Medical Journal. April: 222-224.

American Heart Foundation. (1967). Recommendation for Human Blood Pressure Determination by Sphygmomanometers. Dallas:AHA, 1967.

Balch, J.F. 1997. Prescription for Nutritional Healing. United States of America: Avery Publishing Group. ISBN 0 89529 727 2. 600p.

Berkow, R (Ed). 1996. The Merc Manual of Diagnosis and Therapy / 16th edition. Rahway, N.J. U.S.A.: Merck & Co., Inc. ISBN 0911910 16 6. 2844p.

British Homoeopathic Association. 1985. German Homoeopathic Pharmacopoeia. Stuttgart: Deutscher Potheker Verlag. Translation of the 1st, 1978 edition. 4th Supplement. ISBN 094 6717 052. 1917p.

Bruneton, J. 1995. Pharmacognosy, Phytochemistry, Medicinal Plants. Paris. Lavoisier Publishing Inc. ISBN 2 7430 0028 7. 915p.

Casserley, C. 1998. Blood Pressure. The Journal of the British Homoeopathic Association. **49**(5): 103-106.

Cherif, S., Rahal, N., Haouala, M., Hizaoui, B., Dargouth, F., Gueddiche, M., Kallel, Z., Balansard, G., Boukef, K. 1996. A clinical trail of titurated Olea europea extract in the treatment of essential arterial hypertension. Journal of Pharmacology, **51**(2): 69-71.

Clarke, J, H. 1992. The Prescriber. Great Britain. Hillman Printers (Frome) Ltd. ISBN 0 85032 088 7. 382p.

Denston, T. C. 1951. A Textbook of Pharmacognosy 5th ed. London: Sir Isaac Pitman & Sons, Ltd. 637p.

Duke, J.A. 1997. The Green Pharmacy. United States of America: Rodale Press Inc. ISBN 0 312 96648 2. 617p.

Edwards C. (ed). 1995. Davidson's Principles and Practice of Medicine. United States of America: Pearson Professional Limited. ISBN 0 443 04961 0. 1203p.

Ellis, C. 1995. Hypertension in the Black Patient. Natal: Academy Publications. ISBN 0-620-19053-1. 95p.

Feiter, U. 1999. Parceval Pharmaceutical Company. Personal communication to G. Ronander, 3 February 1999.

Fehri, B., Aiache, J. M., Memmi, A., Korbi, S., Yacoubi, M.T., Mrad, S., Lamaison, J.L. 1994. Hypotension, hypoglycaemia and hypouricemia recorded after repeated administration of aqueous leaf extract of Olea europea. Journal of Pharmacology Belgium, **49**(2):101-108.

Finch, S. 1998. Underground Cures. Baltimore: Agora Health Books. ISBN 1 891434 02 0. 236p.

Fisher, L.D. and van Belle, G. (1993). Biostatistics: A Methodology for the Health Sciences. New York: John Wiley and Sons. ISBN 0-471-58465-7. 991p.

Gaier, H.C. 1991. Thorsons Encyclopaedic Dictionary of Homoeoepathy. London: Thorsons. ISBN 0 7225 1823 4. 601p.

Gallery, E. 1997. Hypertension in pregnancy: practical management recommendations. Modern Medicine of South Africa, February: 36-47.

Gutman, W. 1995. Hypertension: concept and cases. The Indian Journal of Homoeopathic Medicine, **30**(3): 171-173.

Hahnemann, S. 1997. Organon of the Medical Art. Edited and annotated by W.B. O'Reilly. Washington. Birdcage Books. ISBN 1 889613 01 0. 407p.

Lanfranchi, A., Spaziani, D., Servalle, G., Turri, C., Delloro, R., Gassi, G., Mancia, G. 1998. Sympathetic control of circulation in hypertension and congestive heart failure. Blood Pressure. Suppl 3: 40-45.

Lockie, A. 1998. The Family Guide to Homoeopathy. London. Hamish Hamilton Ltd. ISBN 0 241 13572 9. 415p.

Lockie A., Geddes, N. 1992. The Women's Guide to Homoeopathy. London. Hamish Hamilton Ltd. ISBN 0 241 13151 0. 352p.

Maurice, M. 1993. Handbook of African Medicinal Plants. United States: CRC Press, Inc. ISBN 0-8493-4266-X. 435p.

Moll, E. 1992. Trees of Natal. Cape Town: University of Cape Town. ISBN 0-7992-1428-0. 542p.

Munro, J. (Ed). Edwards, C. (Ed). 1995. Macleod's Clinical Examination. London: Churchill Livingstone. ISBN 0443 048568. 379p.

Murray, M.T. Pizzorno, J.E. 1995. Encyclopaedia of Natural Medicine. London: Little, Brown and Company. ISBN 0 1316 877794. 622p.

Naidoo, D.P., Randeree, I.G.H., Seedat, Y.K. 1995. Current trends in hypertension management: controversies over JNC V recommendations. Hypertension: Bridging the Gap. March: 32-39.

Olendorf, D. (Ed), Jeryan, C. (Ed). 1999. The Gale Encyclopaedia of Medicine. U.S.A.: Gale Research. ISBN 0 7876 1871 3. 2018p.

Palmer E., Pitman, N. 1961. Trees of South Africa. Cape Town: A.A. Balkema Ltd. 351p.

Palgrave, K. C. 1977. Trees of Southern Africa. Cape Town: C Struik (Pty) Ltd. ISBN 0 86977081 0. 959p.

Pooley, E. 1993. The Complete Field Guide to Trees of Natal Zululand and Transkei. Durban: Natal Flora Publications Trust. ISBN 0 620 176970. 365p.

Randeree, I.G.H., Seedat, Y.K., Naidoo, D.P. 1995. Metabolic effects of anti-hypertensive agents. Hypertension: Bridging the Gap, March: 14.

Reynolds, J (Ed). 1989. Martindale, The Extra Pharmacopoeia 29th Ed. Great Britain: The Pharmaceutical Press. ISBN 0 85369 210 6. 1896p.

Roy, M. 1994. The Principles of Homoeopathic Philosophy. Edinburgh: Churchill Livingstone. ISBN 0 443 04821 5. 151p.

Sareli, P. and Strugo, V. 1995. Can we still justify the use of low-dose thiazide diuretics as first-line treatment for Black South African Hypertensive? Hypertension Bridging the Gap, March: 28-29.

Schroyens, F. 1995. Synthesis Repertorium Homeopathicum Syntheticum. London: Homoeopathic Book Publishers. ISBN 0 9522744 18. 1720p.

Seedat, Y. K., 1990. Detection, Evaluation, and Treatment of high blood pressure. Hypertension: New Perspectives, March: 24.

Seedat, Y. K., 1995. Is the pathogenesis of hypertension different in black patients? Hypertension Bridging the Gap, March: 42-44.

Smith, J.L., Nel, P.D., Joelson, L. 1987. Hypertension management in a Cape Peninsula coloured community. South African Medical Journal, **72** October: 530-532.

Steyn, K. 1990. Hypertension in the Coloured Population. Hypertension: New Perspectives, March: 27-28.

Taylor, E.J (Ed). 1988. Dorland's Illustrated Medical Dictionary 27th ed. Philadelphia: W.B. Saunders Co. ISBN 0 7216 3154 1. 1887p

Van den Hoogen, P.C., Feskens, E.J., Nagelkerke, N.J., Menotti, A., Nissinen, A., Kromhout, D. 2000. The relation between blood pressure and mortality due to coronary heart disease among men in different parts of the world. The New England Journal of Medicine. **342**(1): 1-7.

Van Wyk, B. and Van Wyk, P. 1997. Field Guide to Trees of Southern Africa. Cape Town: Struik Publishers (Pty) Ltd. ISBN 1 86825 9226. 536p.

Van Wyk, E., Van Outshoorn, B., Gericke, N. 1997. Medicinal Plants of South Africa. Pretoria: Briza Publications. ISBN 1 875093 09 5. 304p.

Van Wyk, P. 1993. Southern African Trees a Photographic Guide. Cape Town: Struik Publishers (Pty) Ltd. ISBN 1 86825 307 4. 378p.

Villiers, L.S., Serfontein, W.J. 1989. Your Heart, The Unrefined Facts. Cape Town: HAUM Educational Publishers. ISBN 0 7986 2804 9. 252p.

Werbach, M.R. 1995. Healing through Nutrition. London: Harper Collins Publishers. ISBN 0 7225 2941 4. 354p.

Wiesner, D. 1992. Your Health, Our World. Great Britain:Prism Press. ISBN 1 85327 071 7. 337p.

Wing, J.R., Joffe, B.I., Milne, F.J. 1995. Metabolic aspects of essential hypertension in Black South Africans- a focus on insulin resistance. Hypertension: Bridging the Gap. March: 11-14.

Ziyyat, A., Legssyer, A., Makhfi, H., Dassouli, A., Serhrouchni, M., Benjelloun, W. 1997. Phytotherapy of hypertension and diabetes in oriental Morocco. Journal of Ethnopharmacology, **58** September: 45-54.

APPENDIX

DETAILED RESULTS

Blood Pressure Research Patients Group I (6CH)

No.	First Reading			Mean 1				Mean 2	Second Reading			Mean 3				Mean 4	Third Reading			Mean 5				Mean 6	Fourth Reading			Mean 7				Mean 8
	sys	sys	sys	SYS 1	dia	dia	dia	DIA 1	sys	sys	sys	SYS 2	dia	dia	dia	DIA 2	sys	sys	sys	SYS 3	dia	dia	dia	DIA 3	sys	sys	sys	SYS 4	dia	dia	dia	DIA 4
1	132	126	134	131	84	88	90	87	132	130	130	131	90	90	90	90	122	130	120	124	82	82	90	85	130	128	128	129	98	92	94	95
2	172	168	168	169	102	98	96	99	160	158	170	163	90	88	100	93	154	132	130	139	88	84	80	84	144	146	142	144	90	88	92	90
3	144	139	148	144	80	90	90	87	138	126	128	131	98	98	96	97	126	128	128	127	86	90	88	88	134	132	132	133	90	92	90	91
4	170	166	168	168	110	108	110	109	150	142	148	147	82	80	88	83	174	170	166	170	94	90	94	93	150	150	150	150	90	92	90	91
5	168	158	154	160	110	110	94	105	168	168	158	165	100	100	102	101	150	144	142	145	100	94	94	96	136	136	126	133	100	96	92	96
6	150	144	148	147	90	88	86	88	138	138	138	138	80	80	80	80	138	134	138	137	82	80	78	80	136	136	138	137	80	78	76	78
7	140	130	134	135	90	90	90	90	130	120	120	123	90	80	90	87	120	120	118	119	80	80	80	80	120	116	118	118	80	80	78	79
8	138	136	138	137	94	92	92	93	146	130	132	136	90	86	90	89	120	120	122	121	86	90	88	88	126	120	126	124	88	86	90	88
9	150	150	152	151	100	100	104	101	150	150	150	150	100	100	90	97	150	150	150	150	90	92	90	91	150	150	150	150	92	94	90	92
10	140	140	144	141	90	100	90	93	138	136	148	141	90	90	94	91	140	130	134	135	90	90	90	90	140	142	138	140	90	88	88	89
11	142	140	142	141	90	88	90	89	140	140	142	141	90	88	90	89	130	134	136	133	90	94	90	91	136	132	130	133	90	90	88	89
12	170	164	168	167	100	102	100	101	160	150	150	153	100	96	90	95	154	140	150	148	90	90	92	91	150	144	144	146	90	92	90	91
13	132	130	130	131	80	84	80	81	128	120	120	123	88	86	84	86	110	110	108	109	78	78	80	79	114	110	110	111	78	78	76	77
14	130	130	130	130	90	90	88	89	130	130	128	129	94	92	88	91	130	128	128	129	90	88	90	89	130	126	126	127	88	90	88	89
15	130	134	130	131	90	86	86	87	130	120	124	125	90	80	80	83	130	120	124	125	80	80	80	80	128	124	126	126	80	80	80	80
16	140	144	142	142	70	72	70	71	150	150	150	150	84	76	76	79	140	144	140	141	74	72	76	74	144	144	140	143	78	72	74	75

Blood Pressure Research Patients Group 2 (Placebo)

No.	First Reading			Mean 1				Mean 2	Second Reading			Mean 3				Mean 4	Third Reading			Mean 5				Mean 6	Fourth Reading			Mean 7				Mean 8
	sys	sys	sys	SYS 1	dia	dia	dia	DIA 1	sys	sys	sys	SYS 2	dia	dia	dia	DIA 2	sys	sys	sys	SYS 3	dia	dia	dia	DIA 3	sys	sys	sys	SYS 4	dia	dia	dia	DIA 4
1	136	140	142	139	94	94	98	95	140	140	136	139	92	90	94	92	130	130	132	131	94	92	90	92	142	146	140	143	98	98	98	98
2	144	130	130	135	88	88	88	88	140	134	136	137	88	86	90	88	130	130	134	131	86	86	88	87	132	130	130	131	88	86	86	87
3	152	152	150	151	90	90	88	89	142	140	140	141	90	90	88	89	144	140	140	141	90	90	90	90	140	144	140	141	90	88	90	89
4	146	144	148	146	90	90	88	89	150	148	148	149	88	82	86	85	140	142	142	141	88	88	88	88	140	140	142	141	88	86	86	87
5	140	140	142	141	90	90	88	89	140	138	140	139	90	88	90	89	140	140	138	139	88	88	90	89	140	138	138	139	88	88	86	87
6	132	134	134	133	100	98	100	99	134	132	130	132	92	100	96	96	120	120	122	121	100	90	96	95	134	126	130	130	96	96	94	95
7	140	134	136	137	90	98	98	95	134	140	136	137	100	104	104	103	120	132	128	127	100	100	102	101	140	138	138	139	104	100	100	101
8	140	140	144	141	80	80	84	81	150	148	148	149	90	90	88	89	128	126	130	128	76	76	78	77	130	128	132	130	78	80	78	79
9	152	150	154	152	102	104	102	103	156	150	150	152	100	100	104	101	150	150	152	151	100	102	100	101	150	148	148	149	100	104	102	102
10	162	158	160	160	98	102	100	100	152	158	158	156	100	108	104	104	144	142	140	142	88	90	88	89	140	144	140	141	88	90	90	89
11	144	148	146	146	94	96	96	95	140	140	138	139	92	90	90	91	130	130	132	131	80	82	82	81	140	138	134	137	90	90	90	90
12	148	140	142	143	100	98	98	99	136	136	140	137	90	84	86	87	138	138	136	137	88	84	84	85	138	136	136	137	86	86	82	85
13	144	138	138	140	90	90	100	93	140	140	138	139	90	98	92	93	140	136	140	139	90	88	88	89	130	140	142	137	88	88	88	88
14	140	140	132	137	80	82	80	81	140	140	140	140	100	100	102	101	138	138	144	140	102	98	98	99	132	134	140	135	104	100	100	101
15	140	140	138	139	100	100	98	99	130	130	130	130	80	76	80	79	132	132	130	131	82	80	78	80	120	122	120	121	76	76	78	77
16	140	140	146	142	92	96	98	95	140	140	144	141	98	98	96	97	140	138	138	139	90	92	90	91	140	142	140	141	90	88	88	89

Blood Pressure Research Patients Group 3 (Tincture)

No.	First Reading			Mean 1				Mean 2	Second Reading			Mean 3				Mean 4	Third Reading			Mean 5				Mean 6	Fourth Reading			Mean 7				Mean 8
	sys	sys	sys	SYS 1	dia	dia	dia	DIA 1	sys	sys	sys	SYS 2	dia	dia	dia	DIA 2	sys	sys	sys	SYS 3	dia	dia	dia	DIA 3	sys	sys	sys	SYS 4	dia	dia	dia	DIA 4
1	140	142	138	140	100	94	98	97	138	134	134	135	82	90	90	87	134	134	134	134	90	92	90	91	134	132	134	133	90	92	88	90
2	140	140	138	139	110	112	110	111	140	136	134	137	114	114	116	115	138	134	140	137	112	110	112	111	138	134	132	135	110	110	108	109
3	140	130	130	133	100	90	90	93	130	122	122	125	82	82	80	81	112	122	114	116	78	82	78	79	116	114	116	115	78	80	76	78
4	134	130	132	132	80	80	90	83	140	138	150	143	90	90	92	91	130	120	130	127	90	80	88	86	120	122	120	121	84	80	78	81
5	154	148	152	151	100	90	94	95	150	142	144	145	100	98	96	98	134	140	142	139	94	90	96	93	134	136	134	135	90	94	90	91
6	142	140	144	142	78	80	78	79	140	142	142	141	78	78	82	79	152	136	138	142	80	78	78	79	140	142	142	141	78	78	78	78
7	130	130	132	131	80	70	72	74	120	114	114	116	68	68	70	69	114	114	114	114	68	68	68	68	140	138	138	139	80	80	80	80
8	132	134	130	132	90	90	88	89	130	130	132	131	86	80	84	83	130	130	132	131	90	80	86	85	130	130	128	129	80	86	80	82
9	140	130	138	136	100	90	94	95	150	150	150	150	100	100	100	100	150	140	146	145	100	90	94	95	150	152	148	150	100	100	100	100
10	130	130	132	131	90	90	90	90	122	120	118	120	80	80	80	80	116	118	116	117	80	78	80	79	120	120	120	120	80	80	80	80
11	150	156	154	153	90	90	92	91	160	160	162	161	90	90	90	90	142	140	138	140	90	90	88	89	142	138	138	139	88	86	86	87
12	140	144	138	141	90	90	90	90	120	120	120	120	80	80	82	81	120	120	122	121	90	80	80	83	120	122	120	121	80	70	80	77
13	170	178	168	172	100	110	100	103	162	162	166	163	106	108	108	107	176	164	162	167	108	100	98	102	170	160	168	166	100	100	100	100
14	146	148	140	145	110	110	108	109	134	130	130	131	110	94	108	104	120	132	132	128	90	96	100	95	140	138	130	136	100	108	106	105
15	160	168	166	165	120	120	116	119	168	156	158	161	120	110	110	113	134	138	138	137	96	100	100	99	130	132	130	131	98	96	96	97
16	140	140	130	137	90	90	88	89	120	124	130	125	80	80	84	81	140	140	140	140	90	80	90	87	130	132	130	131	82	80	78	80
17	140	138	140	139	90	96	96	94	138	130	128	132	100	96	96	97	120	118	120	119	90	90	92	91	120	124	120	121	90	92	90	91