COGNITIVE-BEHAVIOURAL TREATMENT OF ESSENTIAL HYPERTENSION IN AN URBAN XHOSA WOMAN - A CASE-STUDY

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ABSTRACT

Essential hypertension has a complex and multiple biological, psychological and social aetiology and remains one of the most serious physical disorders affecting the Black population of South Africa today. Pharmacological treatment has been the predominant approach to blood pressure reduction, but considering that the greater part of essential hypertension has its origin in bio-behavioural and cognitive functioning a non-pharmacological treatment approach to essential hypertension is receiving extensive interest both in research and therapeutic practice. This study attempted to implement a specific cognitive-behavioural treatment "package' which was tailored to the emergent aetiology in an urban Xhosa woman suffering from Mild hypertension who was on anti-hypertensive medication and to evaluate the treatment using a single case-study methodology. A combination of relaxation training and cognitive-behavioural modification was provided over a fifteen week period and evaluated both quantitatively and qualitatively. It was found that a combination of over-weight, occupational stress and anger could have contributed and/or caused the patient's hypertension. The results also show a significant reduction in blood pressure during the treatment phase as well as a reduction in weight, experienced anxiety and angry emotion, and by the end of the study the patient's blood pressure had been reduced to normal levels. Finally the feasibility of using such a treatment approach is discussed in relation to the South African context and the case-study method is evaluated as a research tool in light of the findings.

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THAT THE BIRDS OF WORRY AND CARE

FLY ABOVE YOUR HEAD

THIS YOU CANNOT CHANGE

BUT THAT THEY BUILD NESTS IN YOUR HAIR

THIS YOU CAN PREVENT

CHINESE PROVERB

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INTRODUCTION

High blood pressure is a world-wide condition of almost epidemic proportions and is considered by most medical professionals as the single most common chronic disorder encountered in practice. It has been estimated that in Western countries somewhere between fifteen and twenty per cent of the adult population have high blood pressure (O'Brein & O'Malley, 1987), while in the United States of America alone, estimates have risen to as high as twenty-five per cent of the population (Herd & Weiss in Matarazzo, Weiss, Herd, Miller & Weiss, 1984, p789). Approximately sixty per cent of those with high blood pressure are unaware that they have it, since for most people with high blood pressure there are no tell-tale symptoms and, in fact, the majority of people with this condition have no symptoms whatsoever. This means that high blood pressure - or **hypertension** as it is termed by the medical profession - is usually diagnosed only by chance, for instance at a routine physical checkup or when a person presents at a clinic or doctor on a quite unrelated matter such as a common cold or backache.

Such an alarmingly high percentage of people suffering from hypertension has captured the attention of the medical profession and other health-care providers who, in recent years, have become increasingly concerned and aware of the problem and its effects on well-being. It has become conclusive that treating people with high blood pressure, whether mild or severe, can prolong life. This has been supported by various studies with the Framingham study showing that a positive, increasing relationship existed between baseline blood pressure and subsequent risk of cardiovascular disease (Gordon, Solie & Kannel, 1971 in Matarazzo et al, 1984). In

addition, the study suggested that increasing evidence made it obligatory to treat all patients with hypertension whenever possible. This recommendation was also supported by several clinical trials, namely from the Hypertension Detection and Follow-up Programme Cooperative Group (1979) and from results of the Australian National Blood Pressure study (1979) which both reported that patients who received aggressive treatment of mild hypertension had lower mortality from cardiovascular complications than patients in control groups (in Matarazzo et al, 1984, p790).

Although hypertension is a serious health problem, it is not a disease in the ordinary sense of the word but a **risk factor** for other more serious and debilitating disorders. In this way it differs from other diseases such as pneumonia, which you either have or you do not have. It is not itself a disease, like one which is contagious, but it increases the probability of developing disease. High blood pressure, high blood cholesterol and smoking have been identified as the major risk factors associated with coronary heart disease (Safran, 1987).

The reason that doctors and other health-care providers are concerned about these high levels of hypertension in the population is that if left undiagnosed and untreated it increases the probability of heart attack and stroke. If treated with medication and other non-pharmacological treatment strategies, the risk of stroke and heart failure is reduced with related extension in life-span (Pickering, in Safran, 1987, p6). As a disease without symptoms the condition is viewed with indifference by many people, even those who have been diagnosed and are receiving treatment. The truly grave problem is that for millions of hypertensive people

their condition remains undetected, undiagnosed, and untreated. Hypertension thus deserves the reputation as "the silent killer", because as many as twenty-five million people may be unknowingly suffering from an ailment that could lead to premature death (Decker & Keiser, 1987, p1).

UNDERSTANDING HYPERTENSION

Hypertension and the Circulatory System

Before one can understand what high blood pressure is, it is necessary to appreciate what blood pressure itself is. This can quickly debunk many of the common myths surrounding hypertension, such as the idea that hypertension is a kind of tension like anxiety or stress. Although mental stress and anxiety may often go hand in hand with high blood pressure, the two disorders are not the same. Hypertension is a physiological response of the circulatory system to a number of physical and psychological factors.

The cardiovascular system of the human body comprises of the heart and an extensive network of blood vessels called arteries, veins and capillaries. In one circuit known as the **pulmonary circulation** the blood which has returned to the heart is pumped through the lungs where it takes up oxygen and releases carbon dioxide and water vapour, a process long understood by man. The blood then returns to the heart and is pumped to the rest of the body through a second circuit called the **systemic circulation.** The proper functioning of these systems depends on three main factors: (1) the heart must be in working condition so that the blood is pumped adequately and with the correct force around the circulation system

[adequate cardiac output]; (2) the blood vessels must be in good condition so that they can carry the blood to every part of the body [low peripheral resistance] and (3) there must be sufficient pressure in the system for it to work efficiently. Although the anatomical and physiological mechanisms involved in these processes are highly complex and intricate, for the purpose of simplicity the above description of the circulatory system is sufficient for now. This force or pressure exerted by the blood pumped from the heart against the walls of the arteries is known as **blood pressure.** Initially, the blood moves along the aorta or large blood vessel leading from the heart and begins to branch-off at various points along the aorta to find its way to the other regions of the body. The blood vessels divide several times into smaller and smaller vessels called capillaries which supply the organs and tissue with oxygen and nutrients and carry away carbon dioxide and other waste-products produced during cell metabolism. On the return journey these capillaries connect with the veins which eventually return the blood to the heart where the cycle starts once more. The circulatory system is a closed system and strategically placed valves on each side of the heart and pressure regulators called baroreceptors within the arteries ensure that the pressure of the blood is maintained at an optimal level for efficient supply of blood to the body mass.

Blood pressure is thus kept within certain limits by the inter-action of many factors, but basically by the pumping action of the heart and the calibre of the blood vessels. If for any reason the pumping action of the heart gets stronger or the resistance offered by the blood vessels increases, blood pressure tends to rise. During the course of an ordinary day blood pressure constantly

fluctuates. Strenuous exercise tends to make the pressure go up as does stress. If you lie down or rest, pressure will go down. This is all quite normal, however, it is when pressure rises markedly and remains high that a person can be said to have hypertension.

Measuring Blood Pressure

Thus, there is no clearly defined blood pressure level above which pressure is too high and below which pressure is normal. In fact, when blood pressure is measured in large groups of people it is found that the readings are distributed over a wide range so that some people have quite low readings and others very high, but most people have levels between these extremes.

With each heartbeat blood pressure undergoes a pattern of change. The highest pressure occurs shortly after the blood is pumped from the heart into the large blood vessels and this peak pressure is known as the **systolic pressure.** There is also a lower pressure referred to as the **diastolic pressure** and this occurs when the heart is relaxed and being filled by the blood returning through the veins, preparing for another heartbeat. Without having to gain access to the blood vessel itself, blood pressure can be measured reasonably accurately by using a pressure gauge called a **sphygmamonometer.** This apparatus consists of a cuff which is placed around the upper arm and is connected to a small rubber pump and to a mercury column. When the cuff is inflated with the pump, the pressure of the cuff increases until it exceeds the pressure in the artery. Then the pressure in the cuff is slowly released by means of a valve, and by listening with a stethoscope over the artery for certain changes in sound in the returning blood flow, the systolic and diastolic pressures can be read off the mercury column. The

initial heartbeat heard [phase 1] when the cuff is deflated marks the systolic pressure and when the heartbeats [Korotkoff sounds] cease, the last beating sound [phase 5] marks the diastolic pressure. At the systolic and diastolic points, the pressure is read off the mercury column attached to the cuff. The calibrated mercury column which is read in millimetres of mercury, thus gives a measurement of arterial blood pressure in millimetres of mercury (mmHg).

Although blood pressure fluctuates, arbitrary cut-off points have been defined by the World Health Organisation as characterising normal and raised blood pressure levels. The following designations have been laid down:

	Diastolic
Classification	Blood Pressure level (mmHg)
Normal	< 95
Mild	95 - 104
Moderate	105 - 119
Severe	> 120

(in Seedat, 1989, p2)

Although hypertension has been classified in terms of diastolic pressure, systolic pressure also provides an accurate reflection of raised pressure levels and according to Kaplan (1982), systolic pressure may even be a better indicator of overall risk. As such, systolic cut-off points have also been identified, with a systolic pressure of 160mmHg or greater designating hypertension ; 140 - 159mmHg labeled borderline hypertension (in Matarazzo et al, 1984, p382). Thus, a sustained pressure above theses levels constitutes high blood pressure, but it is important to note the word

'sustained' in this definition, for isolated rises in pressure above the normal limits are often of no consequence and may occur with normal activities. A rise in blood pressure above the normal is only important when the rise is sustained over a period of time. To diagnose hypertension accurately, multiple measurements of systolic and diastolic pressures need to be acquired over an extended period of time:

> For the diagnosis of hypertension, repeated measurements are recommended, eg. more than three occasions over a period of several weeks with at least three readings per occasion (Appel et al, in Hersen & Bellack, 1985, p383).

General Physiological Mechanisms In Hypertension

Although the preceding description of the circulatory system and blood pressure is simplistic, what becomes clear is that at least two primary mechanisms are involved in elevating blood pressure. If increased cardiac activity and output occur then blood pressure within the vessels increases. If blood vessel resistance increases through blockage or constriction of the vessels then blood pressure increases. It is through these two simple mechanisms that blood pressure is altered and hypertension can result:

> The principle cause of hypertension is increased resistance to blood flow. Resistance to blood flow occurs for a number of reasons, but more often than not it is the result of arteries that have become too narrow as a result of one of several possible factors: excessive activity of the sympathetic nervous system, or hormones that control the dilation (widening) and constriction (shrinking) of the blood vessels, or by cholesterol-rich deposits that

clog the vessels (Decker & Keiser, 1987, p8).

In addition, if the volume of blood increases then more pressure is applied within the vessels and blood pressure increases. It will become evident in later chapters how these mechanisms are influenced by various biobehavioural factors. What has been hypothesised is that prolonged elevation of blood pressure alters the sensitivity of the baroreceptors so that higher pressures do not have this effect. The circulation thus alters its homeostatic point and somehow seems to become accustomed to operating at a higher level.

Although the finer details of this process are not clearly understood, it appears that other factors besides baroreceptor desensitization play a role in converting sustained blood pressure elevation into the chronic disorder of hypertension. The **renin system** is another important mechanism which has been identified in the body for maintaining blood pressure. A change in pressure, say a reduction, causes a release of renin from the kidneys. Renin is an enzyme that is responsible for the production of a substance called **angiotensin** which constricts the blood vessels. When the vessels contract, resistance in the circulation increases and so the pressure rises. Angiotensin also has a second physiological action. It increases the release from the adrenal gland of the hormone **aldosterone** which controls the amount of salt and water excreted by the kidneys. More salt and water is then retained which, in turn, increases the volume of fluid in the circulation and the pressure. **Adrenaline** another hormone involved in the control of blood pressure and its close relative **noradrenaline** are produced by increased stimulation of the sympathetic nervous system

and elevate blood pressure mainly by increasing cardiac activity and output through increased stimulation of the heart muscle. It has been suggested that in many patients with hypertension there is an increased level of these substances in the blood stream, but this has never been proven conclusively. The **kidney** has a central role in blood pressure regulation because it is the site of action of aldosterone and it metabolises the body's salt. One theory about the cause of high blood pressure says that a defective salt metabolism by the kidneys is a major factor. According to this theory, the kidney fails to eliminate as much salt as it should do, so the blood volume and with it blood pressure increases (O'Brein & O'Malley, 1987).

It becomes evident that many physiological mechanisms may be involved in elevating blood pressure and sustaining its elevation to produce hypertension. Although the precise mechanisms and their processes remain unclear, what seems to be occurring is that the **nervous system, heart, blood vessels** and **kidneys** all play central physiological parts in causing hypertension. When only physiological reasons can be found in the aetiology of hypertension, then these are called **'secondary'** causal factor. Otherwise other possible factors become responsible for the raise in blood pressure and express their effects via the physiological mechanisms already described. Then these factors are referred to as **'primary'** causal factors and these are apparently the initial cause of hypertension in the majority of cases.

THE AETIOLOGY OF SECONDARY HYPERTENSION

For the vast majority of hypertensive patients, the precise causes

for their ailment remain unknown. This form of hypertension is usually called 'primary' or 'essential' hypertension - primary because other causes besides impairment or dysfunction of the anatomy are responsible. However, when it said that there are no identifiable causes it is really meant that there are no obvious organic causes which are directly responsible, for many behavioural and environmental factors have been identified which raise blood pressure. It has been estimated that nearly ninety-five per cent of all hypertensive patients suffer from 'essential' hypertension. Of the remaining five per cent, particular anatomical and/or physiological dysfunction such have been described can be held directly responsible for the high blood pressure and to this minority is ascribed the term 'secondary hypertension' (Coleman, 1985).

Detection and correction of a number of anatomical and physiological dysfunctions can reduce and even cure secondary hypertension. Here the cause/s can be identified and alleviated, making secondary hypertension the easier of the two kinds to treat.

Endocrine glands are hormone-secreting glands in the body that affect specific organs or perform regulatory functions by discharging into the blood hormones that are circulated throughout the body. Sometimes endocrine glands fail to work normally and secrete excessive amounts of hormones: this abnormality is called **hypersecretion.** Some hypersecretions raise blood pressure, resulting in hypertension. Specific to hypertension is the secretion of the **adrenals.** Abnormal functioning of these glands can usually be ascribed to a tumor in the gland, either in the central portion, called the medulla, or in the surrounding portion, the

cortex.

If a tumor forms in the medulla of the gland it causes the gland to release excessive amounts of the hormone **adrenaline** and/or nor-**adrenaline.** This tumor is then known as a **pheochromocytoma.** These growths are usually benign, however, when present they increase the size of the adrenal gland, causing more hormones to be produced than normally required. The excessive amounts of these potent vasoconstrictors raise the blood pressure to very excessive levels, overwhelming the mechanisms that usually return the system to proper balance. Though it was previously stated that hypertension is asymptomatic, most patients suffering from a pheochromocytoma will have attacks of sweating, palpitations, severe pounding headaches, sudden increases in blood pressure and heart rate, apprehension, nausea and vomiting, or a tingling in the extremities.

If a tumor develops in the cortex of the adrenal gland another problem arises. The cortex produces a variety of potent steroid hormones, including **aldosterone.** This hormone raises blood pressure by causing salt and fluid retention, which increases the volume of fluid in the circulatory system. In the presence of the tumor, the gland produces excessive amounts of the hormone, causing a form of hypertension called **primary hyperaldosteronism.**

A third high blood pressure problem associated with the dysfunction of the adrenal glands is **Cushing's syndrome.** In this disorder, hypersecretion of **glucocorticoids** occurs from the cortex of the adrenal gland. This may also be caused by a tumor of the adrenal gland, or as a result of the hyperfunction of the anterior part of

the pituitary. Symptoms relating to Cushing's syndrome are fatigue, osteoporosis, impotence, broken capillaries in the skin, skin discolouration and excess hair growth. It causes edema (water retention) which leads to high blood pressure because the excess tissue fluid increases blood flow resistance. Most of these tumors are benign and well localised, thus easily removable by surgery which usually cures the patient of hypertension and other symptoms. The **kidney** is an organ that is both affected by and can be the underlying cause of hypertension. One of the diverse functions of the kidneys is to regulate the concentrations of many important chemical substances in the blood and the urine. In particular, the kidneys help maintain the fluid-sodium balance by excreting extra sodium in the urine or, when the body needs salt, re-absorbing it from the urine. When a long-standing infection has inhabited the kidneys or physical damage has resulted in kidney dysfunction, the normal function will become impaired, causing abnormal amounts of salt and water to be retained by the body. As a result, the fluid volume of the blood increases exerting more pressure against the walls of the blood vessels and causing a rise in blood pressure.

The classic form of kidney disease that causes high blood pressure is **renal artery stenosis** or renovascular hypertension. This occurs when the arteries in the kidney become narrow causing a decrease in the blood supply to the organ. Alongside the arteriole leading to each kidney is a group of **baroreceptors,** that detect this abnormality. These cells release **renin.** A series of complex chemical reactions occur in which renin produces the powerful vasoconstrictor **angiotensin** that raises blood pressure throughout the body. As renal blood flow falls, more renin and angiotensin are produced which causes blood pressure to rise. As blood pressure

rises more blood flows to the kidneys; however, if the obstruction remains, so will the high blood pressure to ensure an adequate supply of blood. By this action the body attempts to overcome the narrowing of the renal artery and to provide the kidney with the blood supply it needs. This form of renal artery stenosis can be corrected either by surgically removing the narrowed portion of the artery or by balloon dilation of the renal artery so that adequate blood flow is re-established and the baroreceptors no longer cause excessive release of renin. The result then - blood pressure is reduced.

OTHER PROBLEM AREAS IN SECONDARY HYPERTENSION

Another problem of reduced blood flow can occur in the **aorta,** which is the main trunk of the entire arterial system. It runs up to the neck and down to legs taking blood from the heart and supplying it to all the key organs and glands. If the aorta is pinched between the heart and the origin of the renal arteries so that blood flow to the kidneys is inhibited, a problem for some people which can result in elevated blood pressure by the same mechanisms as renal artery stenosis. What happens is that the blood pressure in the arteries above the pinched area **(coarctation)** is elevated while the pressure in the lower arteries is not. Several surgical procedures are possible to correct arterial damage that blocks blood supply to the kidneys.

Another type of hypertension is **neurogenic hypertension,** a condition due to emotional stress and can result from any number of life situations. Whatever the circumstance, if one is under significant stress the sympathetic nervous system is activated in

preparation for the 'fight-or-flight' response. With increased and prolonged activation of the nervous system excessive amounts of hormones are secreted into the blood stream, such as adrenaline and noradrenaline, which excite the heart muscle and constrict blood vessels which result in a rise in blood pressure. In most people when the stress is removed the blood pressure returns to normal, however, in some people the hypertensive response to stress continues even when the stress is no longer present. Although Neurogenic hypertension is classified as a secondary cause of high blood pressure, it will become evident that it is this form which may also be operating in the vast majority of essential hypertensives, where environmental stress is present.

A common cause of hypertension in young women is the use of **oral contraceptives.** Oral contraceptives raise blood pressure in women because they cause the kidneys to produce more renin through the action of oestrogen and progesterone in the contraceptive, resulting in increased blood volume. With some women, however, it can be quite extreme and also can cause irreversible damage to the kidneys.

Toxemia or **eclampsia** of pregnancy is another dangerous cause of hypertension and one which is more common than previously believed. Moodley (in Seedat, 1989, p107) reports that toxemia occurs in approximately eighteen per cent of all admissions to the obstetric unit at King Edward VIII hospital in Durban. Some pregnant women develop very high blood pressure that is extremely difficult to control. The cause of this hypertension appears to be an elevation of both fluid volume and vascular resistance, perhaps as a result of hormonal changes during pregnancy. This high blood pressure is

usually associated with swelling of the legs (edema) and protein residue in the urine (protenuria) and may lead to convulsions. Usually the hypertension subsides soon after the infant is born, but in some cases the woman is left with chronic residual hypertension.

Although hypertension can cause heart disease through enlargement of the heart muscle from over-exertion, there are no diseases of the heart that cause hypertension. But there are conditions that cause the heart to pump too rapidly. If the heart rate alone is increased the cardiac output increases but blood pressure remains constant because resistance falls accordingly. Such a condition occurs in hyperthyroidism because the thyroid gland produces excessive amounts of thyroid hormones which results in the heart beating unnecessarily fast. The solution for this problem is to treat the thyroid gland and not the over-active heart. It should be noted that blood pressure increases in hyperthyroidism are usually not common and when they do occur they tend to be minor.

With secondary hypertension identification, diagnosis and treatment of the causal factors described above can cure high blood pressure.

THE AETIOLOGY OF ESSENTIAL HYPERTENSION

Often the goal of medicine has been to treat symptoms without ever reaching a full understanding of the root cause of the symptoms. Chronic pain is a good example and so is essential hypertension. By definition, essential hypertension is of unknown cause, nor does the cause become clearer over the course of the treatment (Decker & Keiser, 1987, p23). Of most importance is that it has no cure. But

through proper treatment it can be effectively controlled which usually lowers the pressure to manageable or normal levels. Though there may be no known cause for essential hypertension, there are known factors that place an individual at high risk for hypertension. Some factors are avoidable while others are unavoidable. However, even unavoidable factors can be addressed through many indirect means, which will become evident in the forthcoming chapters. Depending on the individual a factor may have some effect on the high blood pressure, but as a result of this indeterminable character of the disorder most patients must understand that the treatment is like therapy for diabetes which is generally continuous and probably permanent. Patients who obtain adequate therapy that returns blood pressure to normal and keeps it there will avoid the complications of hypertension and have a normal life expectancy.

The search for a cause or causes of essential hypertension has been the concern of researchers for many years, with the pace of work seeming to accelerate from year to year. Essential hypertension refers to an ailment with a multiple aetiology and most investigators in the field now consider that there is probably no single factor responsible for this type of high blood pressure. However, by examining the aspects of our environment and the manner in which hypertensives operate in context a number of factors have been identified which contribute to the elevation of blood pressure.

Of the many factors, a good number of them are related to how we live our daily lives. The human physiology does not operate in a vacuum but depends on the environment and its stimulation for its continued survival. For example, the ingestion of food, water and

air serve to sustain the living organism and similarly ejection of waste-products must occur for the body to operate optimally. In a similar manner, human beings continue to survive mentally and emotionally via contact with stimulation from other people in the social context. Without love and affection a person would surely ail, but at the same time excess stimulation leads to stress which puts people at risk for excessive bodily excitation and if prolonged, to health risk, unhappiness and unhealth.

So man operates at an interface between mind, body and environment with each having an effect on the other. This is a known fact which needs no further elaboration. However with hypertension, in particular, this interface between man and his surroundings is highlighted, which makes it understandable that a multiplicity of complex **physiological**, **behavioural** and **environmental** factors could contribute to the aetiology of essential hypertension. Although some evidence suggests that inheritance or genetic factors may contribute to high blood pressure, in the main, behavioural and environmental variables have been emphasised in the aetiology. Approximately seventy per cent of the aetiology of essential hypertension can be explained in terms of environmental factors (Weiner, 1979 in Goldstein & Shapiro, 1982, p842). There is sufficient evidence that high blood pressure is the result of family history, personality, stress and other factors such as obesity, physical activity and diet.

Heredity and Family history in the aetiology of essential hypertension

In many cases hypertension has been viewed to run in families. According to the Report of the Task Force on Blood Pressure control

in Children (1977, in Goldstein & Shapiro, 1982, p842) the presence of high blood pressure in one's parents is one of the primary risk factors for hypertension. This produces the possibility that hypertension may have a genetic constituent which is inherited from the parent/s. The blood pressures of identical twins and first degree relatives of persons with hypertension are more likely to suffer from elevated pressure levels. Furthermore, children of hypertensive parents tend to have higher blood pressure than the children of normotensive parents. What genes could be involved in this process remain unknown, yet, researchers have provided some evidence that people with high blood pressure have genetic defects of some enzymes involved in the metabolism of salt (Canessa et al, 1980; Woods et al, 1982 in Matarazzo et al, 1984, p811). However, it is also difficult to separate the genetic possibilities from the familial and environmental influences, and the influence of parental hypertension could therefore be expressing itself either through the genes or through rearing practices or through both means. Factors such as eating behaviours, exercise patterns and learned coping ability are frequently modes of existence which are influenced by family settings.

Environmental factors in the aetiology of essential hypertension

In the last decade we have seen a great reawakening of interest in the healthy ways of living. The renewed interest in health is readily seen in the weight-conscious slim youths of today, and in the jogging and aerobics phenomena which have swept the world by storm. Associated with this is an increasing interest in the proven dangers of smoking and the possibly harmful effects of excessive eating and drinking. In North America where the population has taken very definite steps to improve life-style there has been

considerable reduction in deaths from coronary heart disease (O'Brein & O'Malley, 1987, p45). With this increased awareness of life-style has arisen the associated awareness of the role of life-style on determining physical well-being. As such, many environmental and behavioural factors have been identified through the years as contributors to elevated blood pressure.

Of all the various environmental factors which have been implicated in essential hypertension few have received more attention than the role of **salt.** Salt has been known throughout the ages as a hydrophilic (water-loving) substance which attracts water. Within the human body this results in elevated salt (NaCl) levels increasing water retention which increases the volume and pressure of the blood in the vessels. Most Western populations consume too much salt, in fact, between three to ten times more salt than they need. Extensive investigation on the effects of salt in raising blood pressure has been conducted and evidence suggests that a reduction in salt consumption has positive effects on lowering blood pressure. It has also been noted that in countries where salt consumption is low, hypertension is rare and that blood pressure does not rise with age as seen in Western society (O'Brein & O'Malley, 1987, p36). In addition, salt also refers to potassium chloride (KCl) which has shown to lower blood pressure when consumption is increased (refer to references under Non-pharmacological treatment). These are tentative new findings which still need further investigation. However, some investigators have concluded that although a high sodium diet probably creates problems in individuals already suffering from hypertension it does not necessarily determine the onset of hypertension.

Obesity and its role in essential hypertension

Considerable research has identified obesity and being over-weight as contributing factors to essential hypertension. Although the precise relationship between the two variables remains unclear, it is thought that merely an increase in body mass which necessitates increased blood circulation which causes an elevation in blood pressure is the mechanism responsible. There are also suggestions that endocrine-metabolic derangements may be a factor, for example where hyperinsulinemia of the obese person may cause increased retention of sodium by the kidneys and a subsequent rise in blood pressure. Additionally, increased sympathetic tone and turnover of norepinephrine associated with over-eating can also lead to an increased renal tubular reabsorption of sodium (Sims & Berchtold, in Goldstein & Shapiro, 1982).

Whatever the exact reasons, excessive body weight is associated with high blood pressure and that overweight people are three times as likely as others to have hypertension (Decker & Keiser, 1987, p117), while in the Framingham study by Kannel et al (1967) it was evidenced that the risk of developing hypertension in people who are twenty per cent over-weight is eight times greater than among people who are ten per cent underweight (in Goldstein & Shapiro, 1982, p846). In the same study a weight loss of fifteen per cent or more in men was associated with an average ten per cent decrease in systolic blood pressure. As a result of this and other findings the cornerstone to hypertension therapy for the obese patient has become weight reduction.

Other dietary factors

Heavy **caffeine** and **alcohol** use are associated with hypertension.

Caffeine is a stimulant which alerts the sympathetic nervous system and has short-term hypertensive effects, but it is not certain how chronic usage of caffeine relates to the disorder of hypertension. Similarly, alcohol raises blood pressure in those who drink excessively, with blood pressure decreasing as the body becomes detoxified. Mitchell, Morgan, Boadle, Batt, Marstrand, & McNeil (1980) suggest that up to ten per cent of hypertension could be the result of alcohol consumption. Consequently, it is strongly recommended that alcoholic beverages be removed from the hypertensive's diet and caffeine products be reduced or eliminated. Reductions in the consumption of alcohol in particular have been associated with reductions in blood pressure (Criqui et al, 1981; Klatzky et al, 1977, in Matarazzo et al, 1984, p816).

Essential to health is a waxy substance called **cholesterol** which is utilised by the body as a building block to produce cell membranes, hormones such as oestrogen and progesterone, Vitamin D, and protects nerve fibres. Yet, when one consumes more than a certain amount of cholesterol the body can no longer decrease its own cholesterol production to compensate. When the body has more cholesterol than it can use, the excess gets deposited in the arteries thickening the walls and thereby narrowing the interior channels and raising blood pressure. Thus, reduced ingestion of dietary cholesterol through eating less animal fat can lower blood pressure and prevent further coronary heart disease and stroke. Dietary cholesterol is a saturated fat and is but one of many saturated fats resulting from animal products. Other fats include monounsaturated and poly-unsaturated which occur from vegetable sources as well. Although there still appears to be much controversy surrounding fat and hypertension it has been suggested

that the following guidelines should be upheld as regarding the effects of fat on physical well-being:

> What the difference means in terms of heart disease and hypertension can be summarised simply: saturated fat is bad because it increases blood cholesterol; monounsaturated fat seems to be neutral, in that it has little documented effect on blood cholesterol; and polyunsaturated fat is good because it reduces blood cholesterol (Decker & Keiser, 1987, p124).

Through **smoking**, such substances as nicotine and tar are ingested which enter the blood system and have negative effects on blood pressure, but these are believed to be short-lived and are unlikely to produce sustained high blood pressure. However, smoking does deposit sticky tar and nicotine residues on the inner walls of the blood vessels and lungs which reduces oxygen uptake and constricts blood vessels and, thus, is one of the risk factors in coronary heart disease and strokes. Therefore, people with hypertension by smoking are exacerbating their condition and are putting themselves at an increased risk of cardiac and circulatory disease.

Sedentary lifestyle and lack of physical exercise

A sedentary lifestyle has been implicated as one of the many factors which may contribute to heart disease. Although the results are not conclusive, physical training appears to reduce blood pressure in mild and moderate hypertensives but has little effect on the pressure of normotensives (Horton, 1981). Although the differing positive effects of exercise on the human body involve numerous complex physiological changes, various studies have found that a controlled increase in exercise improves the circulation by

reducing adiposity and improves metabolic processes. Nevertheless, physically active persons exhibit lower pressure than people who are sedentary, although this may be merely due to the fact that those who are physically active may also be generally less obese.

Stress and hypertension

Emotional stress both in the home and at the work place can cause a marked raise in blood pressure and many different stress-reduction programmes have been assessed in terms of their effectiveness in reducing blood pressure. Many have been characterised by positive results. However, both unacceptable levels of environmental stress and/or inadequate personal coping mechanisms resulting in intolerable strain on the person mentally and physically can elevate blood pressure and contribute to hypertension. Maladaptive or inadequate coping ability, less than optimal ways of expressing emotion and Type A personality characteristics have been identified as possible contributors to high blood pressure (Holroyd, Appel, & Andrasik 1983; Novaco, 1975). Perhaps the most interesting thing about essential hypertension is the possibility that the disorder may be a somatic reaction to the perceived and experienced world in which the person lives. Numerous environmental and psychological factors have been identified as playing important roles in the aetiology of essential hypertension, and as such it becomes challenging for mental health-care professionals to work in the field. Passing the responsibility of treating essential hypertension on to the medical professional when so many psychological variables could be influencing the disorder has neglected the important role psychology could play in treating hypertension. It is only recently that this challenge has been taken up by mental health-care workers and behavioural medicine and

slowly additional evidence supporting the psycho-logical and environmental determinants and the uses of psycho-logical therapies is surfacing in Health Psychology literature. It is not proposed, however, that the responsibility of treating hypertension should be laid solely at the feet of psycho-logists and related mental-health professionals either, but the argument is for a cooperative interdisciplinary effort from all who can contribute to finding a final solution to the disorder.

In an early review by Cochrane (1971) the benefits of this synergy were already implicated, with the rationale for such a move stemming from a number of important facts. Firstly, the principle causes of essential hypertension remain unknown. Secondly, what is known about the physiological mechanisms involved in essential hypertension is consistent with regarding it as a disorder where there is a somatic response to psychological factors.

Although much work has been conducted to assess the relationship between psychological factors, and especially the effects of stress, and the somatisation of essential hypertension since this review was published, the specific conclusions which Cochrane draws remain relevant and valid:

(1) Physiological explanations of blood pressure elevation are consistent with the possibility that some forms of essential hypertension are somatisations of perceived and experienced psychological variables, such as stress.
(2) Elevation of blood pressure can be conditioned to previously neutral stimuli and this is resistant to extinction, and may in fact become exaggerated after conditioning has ceased.

(3) Long-lasting changes in blood pressure can be produced in man and many different species of animal by noxious stimulation.
(4) There is some evidence that hypertension develops in individuals who are emotionally labile, have neurotic tendencies and repress hostile and aggressive impulses.
(5) Despite the enormous energy expended in attempting to find consistent correlates of high blood pressure very few factors have been identified as being conclusively implicated in hypertension. It seems probable that a complex interaction between felt stress in the environment, personality and personal history contributes to the development of hypertension in some individuals. This, however, is unlikely to completely account for the phenomenon of essential hypertension but may account for many of the essential hypertension cases which are seen.

HEALTH RISKS AND CONSEQUENCES OF UNTREATED HYPERTENSION

If untreated both essential and secondary hypertension can lead to a myriad of potentially fatal disorders. High blood pressure can lead to damage of blood vessels and then to coronary heart failure, stroke, loss of vision and kidney damage. Early detection, though as easy as one-two-three, occurs less often than it should because many people do not regularly have their blood pressures checked. The result is that millions of undetected hypertensive individuals suffer the effects of their disease for years. In particular, blood vessels take the largest brunt, while the heart, kidneys, eyes and brain incur damage. Isaacson (1989) writes that the damages associated with hypertension are enormous if untreated and states that without treatment, about sixty per cent of patients with benign essential hypertension die of heart failure; about thirty

per cent of cerebral haemorrhage or infarcation and only ten per cent from various other causes unrelated to hypertension (p34).

Arterial Destruction

The increased pressure leads to thickening of the blood vessels through increased deposition of fatty substances (for example cholesterol) on the inner walls of the vessels. These processes lead to the acceleration of arterial hardening and narrowing, known as atherosclerosis. High blood pressure may also lead to vessels bursting, resulting in haemorrhaging in the body and especially in the brain, causing brain damage and possible death. In these abnormal vessels there is then also a tendency to thrombosis, or clot formation. In the heart this can lead to blockage of the coronary blood vessels resulting in heart failure, while in the brain to paralysis and speech disturbances known as a stroke.

Cardiac Disorders

High blood pressure contributes to the development of coronary artery disease, the commonest form of heart trouble in the Western world. The increase in blood pressure puts an added load on the muscles of the heart which results in enlargement of the heart muscle and, in particular, to left ventricle hypertrophy. In prolonged and severe untreated high blood pressure, the heart may become incapable of normal function. This is known as heart failure and can result in a lethal heart attack.

Kidney Dysfunction

Long-standing and severe high blood pressure may also lead to changes and malfunctions in the blood vessels of the kidneys preventing normal excretion metabolism and resulting in thickening

of the vessels which leads to a reduction in the flow of blood to the kidneys with subsequent deterioration of its function.

Effects on the eyes

As with the heart, brain and kidneys the eye contains numerous blood vessels which may become thickened or may burst due the presence of hypertension. In severe cases these blood vessels can leak blood and plasma into the surrounding eye and cause retinal haemorrhage and loss of eyesight. However, for the vast majority of people with hypertension eyesight is not affected.

It becomes clear that hypertension can be responsible for numerous serious physical malfunctions if remained untreated. In short, high blood pressure can lower a person's life-expectancy by putting him/her at risk for serious arterial, coronary and cerebral disease. People with untreated hypertension cannot expect to live as long as those with normal blood pressure, but of-course this depends on the severity, whether it is detected early or not, and whether it remains under adequate control during the person's life. Kannel (in O'Brein & O'Malley, 1987, p21) reported that in addition to people with high blood pressure being four times more likely to have heart failure, they have three times as many heart attacks and seven times as many strokes as compared with their normal counter-parts. However, when the condition is treated and controlled the outlook becomes much brighter. Appropriate blood pressure treatment prevents the complications described and in many cases allows for a normal life and normal life expectancy.

PHARMACOLOGICAL TREATMENT OF HYPERTENSION

Secondary hypertension can often be treated and cured through surgical procedures, but in some cases it remains chronic even after treatment. For all cases of essential hypertension surgical treatment is useless and so medication is resorted to as a means of controlling these two latter situations. Various pharmacological substances have been shown to reduce blood pressure by acting on the different target physiological dysfunctions associated with the disorder.

> Systematic and sustained medication that corrects aspects of physiological functioning can control hypertension successfully and reduce the risks of life-threatening disorders associated with chronically elevated blood pressure (Edwards, 1990a, p2).

These medications include (1) diuretics which deplete sodium and fluid levels; (2) sympatholytics such as alpha and beta-blockers which counteract the effects of adrenaline and noradrenaline; (3) vasodilators which counteract blood vessel constriction by reducing peripheral resistance; (4) angiotensin converting enzyme (ACE) inhibitors which reduce the production of the vasoconstricting hormone angiotensin and (5) slow channel calcium entry blocking agents which reduce the flow of calcium into the smooth muscle cells in the blood vessel walls which causes vessel dilation and reduction in blood pressure.

Like most drugs, though, many negative side-effects are associated with antihypertensive medication. They may cause undesirable symptoms which can range from impairment of sexual functioning to a

reduction in mental acuity and even to an intolerance for exercise (Seedat, 1990, p20). These side-effects make it important to assess whether or not the patient is able to have his/her blood pressure controlled by any other means. If medication is needed then it often becomes necessary to treat the secondary negative effects of the medication and to remove the undesirable effects of the treatment by using other pharmacological treatment and so a vicious circle of drug treatment is often established. Of the negative side-effects associated with drug therapy, the following have been identified for concern:

(a) Diuretics: elevated blood sugar, potassium loss, gout and excessive fluid loss.

(b) Potassium-sparing diuretics: drowsiness, dryness of the mouth, increased thirst and lack of energy, nausea, diarrhea, vomiting, cramps, breast tenderness, headache, clumsiness, sweating, increased hair growth, irregular menstruation and impotence.

(c) Alpha-blockers: drowsiness, lack of energy, blurred vision, constipation, diarrhea, headache, loss of appetite, nausea, dry mouth, mental depression, vivid dreams and frequent urination.

(d) Beta-blockers: heart problems, breathing disorders, leg fatigue, skin rash, temporary hair loss, bad dreams, depression, cramping, exercise intolerance, insomnia, constipation, cold hands and feet, bleeding and bruising and hallucinations.

(e) Vasodilators: light headedness, palpitations, diarrhea, loss of appetite, nausea, shortness of breath, anginal pains, headaches, swelling, weakness, numbness or tingling in

arms and legs, bleeding and bruising, nasal congestion, urination problems, nervousness, emotional depression, fever, swelling of lymph glands and hair growth.

(f) ACE inhibitors: protenuria, excessive urination, interference in white blood cell production, anginal pains, abnormal heartbeat, swelling of the face, flushing of the skin, dizziness, rashes and loss of taste.

(g) Slow Channel calcium blocking agents: shakiness, confusion, tingling in the legs and arms, hair loss, blurred vision, spotty menstruation, fainting, impaired sexual functioning, angina, swelling of ankles and feet, rashes, light headedness, shortness of breath, dizziness, heartburn, cramps, stomach gas, blocked nose, sweating, flushing, sensation of warmth, diarrhea and nervousness.

(Decker & Keiser, 1987)

The above list of common side-effects of antihypertensive drugs speaks for itself and clearly these drugs have numerous negative consequences and effects which can inflict much discomfort and irritation in the patient. In addition to side-effects, drug therapy is not always successful even if the best medication 'fit' is found for the patient. Johnston (1987) reports that in a British Medical Research Council study more women died in the medicated group than in the control group and that approximately twenty per cent of patients reported troubling side-effects from the medication. Goldstein and Shapiro (1982) also note that on average fifty per cent of patients treated with medication remain uncontrolled.

Medication costs are also excessively high and place considerable

financial strain on the patient and on the state. As an example of the cost involved to treat hypertension with medication, to prevent one stroke, 850 patients must be medicated (Johnston in Edwards, 1990a, p3). Low compliance and non-adherence are additional problems associated with drug therapy. It seems that a lack of understanding and apathy on the part of many hypertensives, combined with the fact that high blood pressure is asymptomatic reduce the person's compliance to drug regimens. It is reported that in the United States of America alone, forty per cent of diagnosed hypertensives drop out of treatment in the first year and of those who do remain in treatment another forty per cent fail to adhere to the treatment sufficiently to control their blood pressure (Morisky, 1986).

Nevertheless, drug treatment continues to be the most used therapy and despite its numerous disadvantages and ineffectiveness it is hailed by the medical profession as the treatment of preference for severe cases of hypertension. However, alone it appears to be an unsatisfactory treatment for the majority of hypertensive cases and needs to be viewed as a treatment option rather than as a panacea for the disorder. Recently, other non-pharmacological treatment strategies have been devised which increase the resources to deal with the disorder.

NON-PHARMACOLOGICAL TREATMENT OF ESSENTIAL HYPERTENSION

The goal of treatment of hypertensive individuals is to reduce cardiovascular risk by lowering pressure to at least 140/90mmHg (Seedat, 1989, p4) with pharmacological or non-pharmacological methods, or both. Behavioural techniques may be used to fulfill

various purposes in hypertension therapy: (a) as an initial step before resorting to drug therapy in newly diagnosed mild or moderate hypertensive patients; (b) to produce further control in patients who respond inadequately to drugs or in whom dosage must be limited due to adverse side-effects; (c) as an adjunctive treatment to medication even in well-controlled patients to reduce drug requirements and dependence, and (d) as preventative measures for at-risk individuals.

Although severe emergency cases of hypertension may need fast and effective control through medication, the prospects for non-pharmacological treatment and the associated role of psychology clinicians have increased markedly. Considering that numerous bio-behavioural factors have been implied in essential hypertension it becomes feasible to exploit the services and abilities of mental health professionals where it is applicable.

The recommendation that hypertensives alter their lifestyle has become routine in the treatment of hypertension and consequently, the patient is encouraged and aided in changing a variety of behaviours in the hope that the changes will reduce blood pressure and with the assumption that, at the least they will not impart negative side-effects.

In line with Rosendorff's recommendation (1989), patients with mild and moderate hypertension should receive non-pharmacological therapy as a first option followed by medication only if the behavioural changes do not exhibit an adequate control on blood pressure. All of the non-pharmacological interventions can be effective in lowering blood pressure to normal and may, therefore,

reduce the need for antihypertensive medication. Indeed the inconvenience, cost and complications of antihypertensive medication may be undesirable in patients with low levels of arterial hypertension and so non-pharmacological therapy becomes the modus operandi of choice.

With lifestyle implicated in a large way in the aetiology of essential hypertension the time has come to evaluate all methods of treatment equally, be they medical, psychological or behavioural. If synergy between behavioural methods, weight control, diet, exercise and drugs can be demonstrated, money can be saved, patients can be spared the negative side effects and toxicity of drugs, and treatment regimens can indeed be tailored to an individual patient's lifestyle and preferences.

The causes of essential hypertension are unknown, but there is substantial evidence to suggest that psychological factors play an important role in the development of the condition, either directly through stress-related effects on the cardiovascular system, or via behaviours necessarily associated with physical risk factors such as obesity, lack of exercise, heavy drinking or inappropriate expression of emotion as a response to stress-inducing situations. Non-pharmacological treatment is thus intended to follow two specific routes in reducing high blood pressure: either by **indirectly** modifying contributing factors such as diet, exercise and responses to stress, or by **directly** modifying physiological self-control through procedures such as biofeedback and relaxation training.

Following on the kinds of psychological and behavioural factors

which have been identified as playing a role in the aetiology of essential hypertension, the major areas for behavioural intervention are aimed at weight reduction, reduction of salt consumption, alcohol and caffeine; instituting regular vigorous exercise and stress management and reduction strategies. In addition, personal responses to stress is often inadequate or regulation of emotion is maladaptive, and in such cases it may also be appropriate to introduce new coping abilities and/or restructure emotional behaviours. Anger, for example, has been reported to play a role in hypertensive functioning, with an inappropriate suppression of anger leading to rises in blood pressure. Holroyd et al (1983) and Novaco (1975, 1976, 1977) have recommended the inclusion of anger management in the repertoire of possible treatment strategies.

However, not all psychological and behavioural factors necessarily operate in any one individual and as such it becomes important to identify those which may be causing blood pressure elevations in the person. Each identified factor can then be "worked-on" systematically using non-pharmacological approaches until satisfactory blood pressure reductions are attained. The literature on non-pharmacological approaches abounds with evidence supporting its use in reducing blood pressure, however, only a synopsis of this is possible in this paper. For a more detailed documentation the reader is referred to two excellent texts on the subject by Matarazzo et al (1984) and Masters et al (1990).

Behavioural Relaxation Therapies

There is a large number of studies documenting the beneficial effects of relaxation, meditation and biofeedback in reducing

hypertension. **Behavioural relaxation therapies** are among the most popular and effective behavioural interventions used to promote health, prevent illness and treat health-related problems. Masters et al (1990) summarise some of the evidence which supports the use of relaxation therapy in reducing hypertension. The overall trend appears to be that for mild and moderate elevations in blood pressure due to essential hypertension, relaxation training, meditation, biofeedback and systematic muscle relaxation are useful strategies in lowering blood pressure and even in severe cases, is a useful adjunctive treatment which may sometimes result in the reduction of medication as well. Studies by Agras et al (1983), Brauer et al (1979), Hafner (1982), Hatch et al (1985), Hoelscher et al (1986), Surwit et al (1978), and Wadden (1983) all document significant reductions in blood pressure from initial levels after treatment compared with control groups (in Masters et al, 1990, p513). Irvine, Johnston, Jenner & Marie (1986) found that relaxation was superior to the control procedure in reducing blood pressure as assessed by nurses blind to the patients' treatment at post-treatment for diastolic and systolic blood pressure.

Patel (1973, 1977; Patel & Marmot, 1981) suggest the particular use of **biofeedback** as a treatment strategy, but in a recent paper by Patel and Marmot (1988) it is summarised that combination therapies using systematic muscle relaxation, biofeedback, relaxation imagery and meditation are more effective than simple component therapies and that the therapeutic benefit cannot be expected unless the patient believes in the treatment and complies to the regimen. However:

> We need to view stress reduction therapy in its proper perspective. It is by no means an easy alternative to

> drug therapy. It requires both time and commitment on the part of the carer as well as those who are cared for. However, it does seem to be free from untoward side effects and may actually enhance the quality of life. In the long-term it can also be cost-effective. It compliments the effects of antihypertensive medications and there is a strong suggestion that it may reduce coronary heart disease (1988, p288).

Extensive research by Benson spanning the last twenty years has provided convincing evidence that **meditation** alone can produce significant reductions in diastolic and systolic pressures (1973, 1974a, 1974b, 1974c, 1975), which has also been supported by Blackwell & Bloomfield (1976) and Barr & Benson (1984). **Yoga** as a relaxation therapy has also received some attention and Brownstein & Dembert (1989) report that yoga-therapy should be considered as a non-pharmacological adjunct or alternative for medical disorders especially where side effects from medications are of great concern and could prevent persons from carrying out their duties safely. In an early study by Patel (1973) where yoga was used as an adjunct to biofeedback, associated reductions in blood pressure were observed. It was suggested that yogic relaxation, meditation and relaxation in general, reduce sympathetic discharge in response to environmental stimuli, making the neurohormonal factors concerned with the production of hypertension less effective:

> Mental concentration reduces the external interference, making the subject less aware of the external environment. This increases his own perception of his own internal environment. Additionally helped by biofeedback, the subject becomes more aware of the smallest changes in

> anatomic function (in this case blood pressure), which allows him to make necessary changes in the control of that function (p1055).

However, not all studies have reported effectiveness of behavioural relaxation techniques in blood pressure reduction, and in a study by Surwit and Shapiro (1978) no significant reductions were measured from baseline measurements and at a six week follow-up the average reduction had only been 1mmHg systolic and 2mmHg diastolic. On the basis of data available from about half the sample at a one year follow-up, pressures were back to pre-treatment levels. Luborsky & Crits-Christoph (1982) also found that compared with medication treatment, patients treated with behavioural methods did not achieve significant benefits in blood pressure reduction.

Although some studies have not added much support for the use of behavioural treatment and especially relaxation therapy, there appears to be an overall trend pointing towards the potential benefits of relaxation training as a treatment strategy for essential hypertension. Black (1979) adds that whether effective or not in reducing blood pressure, relaxation training has far reaching positive effects on the general well-being of a person and that:

> The most dangerous side effect of behavioural therapy is that some patients, feeling so well, assume they no longer need medication or regular follow-up visits (p839).

Exercise management as a treatment strategy in essential hypertension

In the area of **exercise management**, two lines of evidence have been suggested on how regular exercise and physical conditioning might be effective in controlling levels of arterial blood pressure. The first line of evidence is based on the physiological effects of physical conditioning which result in lower heart rates, lower concentrations of plasma catecholamines and lower rates of uptake and production of norepinephrine, which reduce blood pressure. The likelihood that physical conditioning reduces activity of the sympathetic nervous system, at least during exercise, suggests that physical conditioning might reduce the neurogenic component of hypertension (Matarazzo et al, 1984, p836). Additional benefits from exercise have been demonstrated in normalising metabolic disturbances associated with obesity where plasma insulin and triglyceride levels are reduced as well as blood glucose. The second line of evidence is based on the observation that populations or individuals with high blood pressure displaying physical activity have a lower prevalence of coronary heart disease and lowered death rates from cardiovascular diseases (Matarazzo et al, 1984, p836).

Although arterial hypertension is only one risk factor which contributes to coronary heart disease, reduced incidence of arterial hypertension probably contributes to the reduced incidence of morbidity and mortality. Experimental studies on non-human primates have demonstrated that regular exercise reduces the severity of coronary heart disease, suggesting that cardiovascular response to exercise and physical conditioning apparently has an effect on levels of arterial blood pressure. This effect of training also appears to be independent of changes in body weight

or body fat content, but does appear to be related to other metabolic abnormalities associated with obesity such as hyperinsulinemia and hyper-triglyceridemia (Horton, 1981). Thus modifications of, or introduction to exercise can be used as a treatment strategy to help reducing a person's blood pressure. Fitzgerald (1981) provides evidence that regular aerobic isotonic exercise like jogging depresses labile pressure values and forces them down to near basal levels and prevents rises in previous blood pressure levels for several hours at a time. Wilcox, Bennet, Brown, & Macdonald (1982) found that moderate exercise was associated with a significant and sustained fall in absolute systolic pressures as compared with pre-exercise values with the average reduction being about twenty-five per cent. Horton (1981) suggests that physical training may be similar to low-calorie or low-carbohydrate diets which may result in altered renal tubular handling of sodium secondary to decreased plasma insulin concentrations. An additional blood pressure lowering effect of training may be the result of decreased sym-pathetic nervous system activity, lower plasma concentrations of epinephrine and norepinephrine and decreased vascular resistance (p165). The mechanisms by which physical activity lower blood pressure are not fully understood, yet as a strategy to be included in a multiple non-pharmacological treatment package for the hypertensive patient, exercise has encouraging potential.

Dietary modification and weight reduction in essential hypertension

Dietary modification and **weight reduction** have also been proposed as options to treatment of essential hypertension. Since excessive consumption of sodium chloride has been shown to elevate arterial

blood pressure levels, reduction in dietary salt - especially in patients who are identified as salt-sensitive - becomes a logical and feasible step in reducing hypertension. Beard, Gray, Cooke, & Barge (1982) found that patients who reduced intake of dietary sodium experienced a significant drop in pressure levels and reduction of sodium intake permitted drug treatment to be substantially reduced without side effects or loss of blood pressure control. On the other hand, such impressive results were not obtained by Grobbee & Hofman (1986) who found on analysis of thirteen randomised trials on the effect of sodium restriction on blood pressure that sodium restriction seems to be of limited use in those who are most eligible for non-pharmacological treatment - namely, young patients with mild hypertension. Swales (1980) argues that while extreme salt restriction will lower blood pressure it is not a practical possibility:

> Evidence in favour of a more modest limitation of salt intake is conflicting and the possible harmful effects of salt restriction have not been assessed. Until more conclusive evidence is obtained it would be premature to advocate such massive public-health measures as reducing sodium content of food (p1177).

Increasing **potassium chloride** intake has also been proposed as an alternative to pharmacological treatment as a strategy like sodium restriction which can be implemented with relative ease. Skrabal, Aubock, & Hortnagl (1981) found that moderate salt restriction combined with a high potassium intake helps to prevent hypertension, that salt sensitive subjects exist, and that these individuals would profit most. In a controlled study by Matlou, Isles, Higgs, Milne, Murray, Schultz, et al (1986) and an analysis

of seven other studies, they conclude that potassium supplimentation does lower blood pressure, but that the changes are small.

Reduction in **animal fat** consumption has also been associated with reductions in blood pressure levels in patients who consumed a vegetarian diet. Mean systolic and diastolic blood pressures did not change in the control group during the vegetarian diet and rose significantly in the experimental group which reverted to the omnivorous diet. Although the exact nutrient(s) causing these blood pressure changes were not conclusively established, a reduction of saturated animal fats was proposed to be the effector, while the effects were not mediated by changes in sodium or potassium uptake (Rouse, Armstrong, Beilin & Vandongen, 1983). Results from a study by Brown, cited in a recent edition of **Time magazine** (October, 1990, p49) report that reduction of animal fat consumption by thirty per cent produced significant reductions in total cholesterol levels and in approximately thirty-five per cent of cases, decreases in arterial plaque were observed.

General weight reduction with reduction in adiposity and body mass has been associated with significant reductions in blood pressure. Alexander (1984) summarises the role of obesity and weight reduction in hypertension in the following way:

> Whether an individual is obese or not, development of sustained hypertension with advancing age is partially preventable by the maintenance of stable body weight. If hypertension has already supervened in an obese person, weight reduction, even of modest degree, and maintenance of stable body weight thereafter result in a sustained decrement in blood pressure in about one-half of these

> individuals. If the pre-existing degree of obesity is severe, the potential for subsequent development of congestive heart failure is thus minimised. Postural hypotension, largely secondary to suppression of sympathetic nervous activity, is an important complication of fasting or markedly restricted caloric regimens (in Matarazzo et al, 1984, p883).

Basler, Brinkmeier, Buser, Haehn, & Molders-Kober (1985) report that after six months of group therapy, including education aimed at weight reduction, improvement of stress coping and reduction of salt-intake, mean weight reduction was 5,2kg and blood pressure had decreased by an average of 14,4mmHg (systolic)/ 7,4mmHg (diastolic). After a follow-up period of four months, the beneficial effects of the group treatment were confirmed: patients needed less antihypertensive medication and consistent reduction in body weight prevailed. In a well designed large study from Israel an attempt was made to separate the effects of weight loss from dietary sodium reduction. All patients were encouraged not to restrict their salt intake and it was found that the average decrease in blood pressure of the patients treated with diet alone was 25mmHg systolic and 20mmHg diastolic with a normal blood pressure being attained in seventy-five per cent. Diet treated patients lost an average of 9,5kg of body weight and the regimen was successful in patients with all degrees of obesity (Morgan et al, 1978 in Black, 1979, p839). Unfortunately patients do gain much of the weight lost during dieting and the long-term usefulness of these programmes has not been established. Weight reduction usually accompanies other dietary modification and should be considered as a treatment approach in all patients who are noticeably overweight. Many additional studies have been conducted which lend support to

the use of weight reduction in lowering blood pressure (refer to Matarazzo et al, 1984, p877 for a detailed synopsis).

Cognitive-behavioural treatment of essential hypertension

The goal of cognitive-behavioural interventions is to modify the feelings and behaviours of a person by influencing his or her patterns of thought. The use of cognitive therapies is based on the assumption that thoughts or cognitions are important mediators of both the emotional and behavioural responses associated with any given event. Cognitive techniques are used in medical settings with a similar goal to modify thoughts that may negatively influence the client's health. A person's health may be influenced negatively by his or her cognitions in at least three ways: (1) Many people hold irrational beliefs about themselves which influence whether or not they will perform certain health-related behaviours; (2) second, due to maladaptive cognitive appraisals of potential stressors individuals may experience excessive amounts of stress, putting them at great risk for the development of chronic illness; and (3) a patient's cognitions about his or her illness may cause the person to experience anxiety and depression which, in turn, may exacerbate existing symptoms or increase illness-related distress.

Cognitive-behavioural interventions have been used to decrease the Type A or coronary-prone behaviour pattern. As mentioned earlier Type A behaviour, characteristic of hostility and anger, has been linked to coronary heart disease (Dembroski et al, 1985 & MacDougall et al, 1985 in Masters et al, 1990, p542). The rationale for using cognitive-behavioural techniques in modifying Type A behaviour stems from the assumption that these individuals respond to daily stress in an "all or nothing fashion" in which they

usually accomplish their goals, but only with an unecessarily high cost in energy expenditure and disturbed interpersonal relations (Roskies et al, 1986 in Masters et al, 1990, p542). The goal of cognitive-behaviour therapy has, therefore, been to teach the **Type A** individual to respond to potential stressors in a less stereotyped, more differentiated manner using such coping strategies as relaxation training, communication skills, cognitive relabeling, stress inoculation and problem-solving. Anger as one component of the Type A disposition has received large attention over the last decade as a possible cause of essential hypertension. Following on the stress-inoculation programme devised by Meichenbaum (1975), Novaco (1975) has applied such a strategy to deal with anger-control in hypertension. The maladaptive and in-appropriate suppression of anger has been identified as a possible reason for essential hypertension and what stress-inoculation tries to achieve is a modification in the expression of that anger. Instead of repressing angry thoughts and increasing negative "self-talk" regarding the event or person which is leading to the anger, stress-inoculation teaches alternative ways of expressing anger outward in an appropriate manner and reducing inwardly directed anger. As Safran (1987) remarks on the relationship of anger to one's health:

> Anger has two ways to go, out or in. If you express it outwardly, at the person or thing that is making you angry, that's what's meant by releasing your anger. Your letting it go, though obviously some ways of doing that are better than others. If you express it inwardly, you are directing it at yourself. You're holding on to it, brooding about it, letting it simmer. It's true what they say about anger. It does 'burn you up'. It does 'eat you

up inside' (p121).

Similarly, any other negative emotion or cognition can have the same effect and can potentially also lead to increases in blood pressure. Cognitive-behavioural therapy has been shown to reduce high blood pressure by altering these self-destructive cognitions and emotions, with anger-reduction having received the most attention. Novaco (1975, 1976, 1977) and (1979 in Kendall and Hollon, p241) has identified anger as possibly the most important factor leading to depression and essential hypertension and has observed significant reductions in blood pressure levels after cognitive-behavioural treatment using a stress-inoculation programme. Props (1990) has also identified that excessive control of anger leads to higher systolic blood pressure levels, while Scholten, Maes and van Elderen (1990) have proposed that patients who score high on "anger-in" and low on "control anger-in", as measured by the Anger-expression scale developed by Spielberger (1985), and patients scoring low on "anger-out" and low on "control anger-in", measuring that they fail to control or bring down quickly the level of their suppressed anger, have a significantly higher variability in systolic blood pressure before physical exercise. Vinck (1990) found that especially control of the expression of anger is related to blood pressure as a single factor: higher levels of control are related to higher levels of blood pressure.

A worksite based study by Drazen, Nevid, Pace, & O'Brein (1982) used a cognitive-behavioural approach based on **Rational Emotive Therapy** (RET), which included training in the identification and appropriate expression of emotions like anger, to lower blood

pressure. A further experimental group received training in anxiety management so as to identify stress symptoms and to use them to cue relaxation. Compared with a control group which received education about hyper-tension and were encouraged to reduce salt consumption and weight-loss, the experimental groups achieved lower diastolic pressures, while the RET group showed a reduction in systolic pressure (Edwards, 1990a, p10). It thus appears that self-destructive means of regulating negative emotion and coping with stress may lead to an increase in blood pressure and that modification of personal reactive and coping abilities may lead to reductions in blood pressure levels.

Any one individual may experience any one aetiological factor or a combination of various factors at the exclusion of others in his particular case of essential hypertension. The use of complimentary cognitive and behavioural techniques in the treatment of essential hypertension may be the best approach. In this way, a broad spectrum behaviour therapy or multimodal therapy approach including such strategies as cognitive-behaviour modification, relaxation training, weight reduction, dietary modification and increased exercise may be the most beneficial approach. There are two major advantages to the multimodal approach: first, many health-related problems are multifaceted, involving a complex interaction of many factors in both the development and maintenance of the problem. Second, multimodal therapy emphasises the individual needs of each patient. Multimodal therapy not only permits the inclusion of more than one behavioural procedure, but also posits that these techniques should be carefully selected to influence the target behaviour in the most effective way. In addition, it becomes of utmost importance that the particular programme implemented,

whether standardised or not, meets the particular needs of the patient. Also, a multimodal treatment package may include pharmacological treatment as a component:

> The basis for combining treatments of different modalities is the hope of an additive or synergistic effect that would be greater than the effect of either intervention alone (Masters et al, 1990, p546).

A study by Taylor, Agras & Sevelius (1977) where pharmacologically treated hypertensive patients were assigned to one of three conditions: relaxation training plus medication; non-specific psychotherapy plus medication and medication alone, found that at the end of the treatment the patients who had received a combination package of relaxation and medication had significantly lower blood pressures than did the patients in either of the other two conditions. A case-study reported by Hersen and Bellack (1985, p393) using a multidisciplinary approach including relaxation training, dietary modification and exercise achieved blood pressure reductions from an initial 148mmHg (systolic) and 98mmHg (diastolic) to a final 138mmHg (systolic) and 84mmHg (diastolic), blood pressure in the normotensive range. In addition, cardiovascular fitness had improved, triglyceride and high-density lipoproteins were at optimal levels and personal self-confidence had increased. The literature abounds with replication and comparative studies assessing the relative effectiveness of the various treatment strategies and many have been met with success, attesting to the postulate that essential hypertension has a bio-behavioural aetiology and is potentially amenable to non-pharmacological treatment.

EPIDEMIOLOGY AND SOCIO-CULTURAL FACTORS OF ESSENTIAL HYPERTENSION IN A SOUTH AFRICAN CONTEXT

Today there are about 4,5 million people suffering from high blood pressure in South Africa, a number which is expected to more than double to approximately 10 million by the year 2000 (Seedat, 1989). Considering that the total population of the country measures nearly 35 million at present, the number of hypertensives presents 1 in 8 persons. Clearly, the situation forces the health care professionals of the country to adopt an aggressive stance in combating such a disease. However, essential hypertension - which accounts for the majority of cases - by definition is unrelated to gross organic impairment and is believed to have a multiple bio-behavioural aetiology which has made the task of detection and treatment that much more difficult. Many patients, often with idiosyncratic behaviours, need to be treated by few health-care workers. This has provided an enormous challenge to the health-care providers of the day, and considering how such an illness debilitates the population both physically, emotionally, and financially it seems that an urgent reappraisal of the situation is required and more effective strategies provided on various levels of intervention to alleviate the problem. As Beard et al (1982) report:

> No single advance in community health would improve the quality of life more than the control of hypertension. Drug treatment has been recommended even for mild hypertension, but implementing this recommendation would mean that up to one-fifth of the entire population of a country like Australia would require medication for life with substantial problems of cost and compliance and with

no effect on primary prevention (p455).

Such a problem is pertinent to the South African context and if one considers the continued expenditure on medication for the average man in the street, a need exists for alternative, less expensive yet equally effective strategies to control the disorder. Accepting that multiple environmental and psychological factors have been identified as playing a role in the aetiology of essential hypertension, it would seem reasonable that such factors could be addressed by psycho-behavioural therapeutic regimens, thus reducing dependence on medication which often has negative side effects and actively increasing the individual's personal resources to control the disorder himself, leading to individual enpowerment and other positive repercussions into his general well-being.

Taking an even closer look at the statistics around hypertension it has been estimated that of the 4,5 million sufferers in South Africa nearly 2,5 million are urban Blacks (25%); 750 000 are Whites (23%); 500 000 rural Blacks (10%); 300 000 Mixed group (18%) and 98 000 Indian (19%). These figures provide startling information about the differing proportions of the hypertensive population and raise important questions concerning the particular effects of socio-economic factors on the aetiology of the disorder. Urban Blacks constitute the highest proportion of hypertensives, a fact which has long been known in South Africa.

As early as 1960 researchers had identified the stark difference between the occurrence of hypertension among urban and rural Blacks in South Africa. Studies by Scotch (1960) and Gampel, Slome, Scotch and Abramson (1961) reported a higher incidence of hypertension

among urban Black populations. It was suggested that the individual living in the town is exposed to considerably more emotional stress than the person living in the country, and that this difference may largely account for the urban-rural differences observed (Scotch, 1960). Gampel et al (1961) found evidence that modified diet, increased alcohol consumption, obesity and increased emotional stress correlated with the increase in hypertensive cases in the urban environments. These findings also support the research of Scotch, which identified environmental stress as contributing to the aetiology. Sever, Gordon, Peart, & Beighton (1980) found that more hypertension was found in urban areas among Xhosa inhabitants than in their counter-parts living in rural areas and that diet and obesity were among the most important causes contributing to the higher incidence.

Gampel et al (1961) also identified the process of urbanisation as an important contributing factor to essential hypertension among the Black population:

> The finding that, in our sample, there was evidence of more hypertension among persons who had spent a short period in town, suggests that stress associated with urbanisation, i.e. the process of adaptation to the town environment, may itself be an important contributory factor (p70).

Scotch (1963) reported the following from his study which corroborated Gampel's findings that differences between hypertension in urban and rural settings could be due to stress factors located in the setting itself:

> This [difference in hypertension between urban and rural populations] was attributed to differences in severity

> and variety of stress in the urban setting as against the rural. In the rural community certain presumably stressful features of the social situation were evident, particularly poverty and the problems associated with wide-spread migratory labour; however, culture change is relatively slow in the reserve [rural setting] in contrast to rapid acculturation in the city...In addition to the familiar and predictable strains of urbanisation and detribalisation, the process in the Union of South Africa is further complicated by the political policy of apartheid, which effectively blocks the Zulu from assuming any but the most elementary forms of Western culture. Thus, a new often dysfunctional and initially nonintegrative culture "accumulates" in the urban slums. Apartheid is an academic issue in the reserve since the presence of whites, particularly white officials is rare; in the city, however, apartheid is very real and police harassment and arrest, a daily fact of life, must be seen as stressful in the extreme (p1205).

Although it is now almost thirty years since that report was published and since the laws of apartheid became entrenched in the political policy of the country the very real stress of living in a Black township under the remaining laws of apartheid continues to exist, despite that many changes which have occurred. Presence of the military police and emotional/physical harassment continue to plague the minds and bodies of many people, while instances of faction-fighting between certain Black social groups have risen to new proportions, endangering the lives of each individual. Thus, it seems that the urban stress of the 1960's has escalated steadily

over the last three decades. This has also been exacerbated by the fact that urbanisation has brought with it all the additional first-world worries and stressors, such as those associated with modern employment and unemployment. However, of noticeable importance was the finding that urban Black individuals lacked the coping ability to adapt successfully to those demands of urban living (Scotch, 1963). Seftel, Johnson and Muller (1980) also found that blood pressure was inversely related to educational status and was higher in young adults who lacked broader social support networks, and with individuals who grew up in rural areas and then migrated to the city compared with those who had lived in the city all their lives.

A picture emerges over the decades of research in South Africa which points to the importance of psycho-social variables in the possible aetiology of essential hypertension among the Black population. It seems that a lack of personal resources such as coping ability, adaptation, self-responsibility, paired with actual and perceived environmental stressors characterise the role of the hypertensive. Such a situation appears to follow global trends where a higher prevalence of hypertension is found among urbanised, high technological communities and increases with acculturation to a technological/urban lifestyle (Goldstein & Shapiro, 1982). More recent research, especially by Seedat (1981, 1982 & 1990) draws much the same conclusions. Hypertension has presented as largely an urban phenomenon with the highest prevalence found among the Black urban sector of the population. Seedat, Seedat, & Hackland's (1982) findings also support those of Scotch and Gampel's previous work:

> The main difference between the urban hypertensive and the normotensive is that the former is not able to meet

> or adapt to the demands of urban life...[and] there was a relationship between variables observed to be stressful and hypertension (p1002).

In relation to the prevalence of hypertension on a world-wide scale Nissinen, Bothig, Granroth and Lopez (1988) rated the level of high blood pressure among urban Blacks in Africa in the years 1983-1984 to be only second highest after city workers of Sao Paulo, Brazil. With such high numbers of financially impoverished hypertensives living in countries with relatively meager social welfare aid, a situation has arisen where drug treatment on a large basis is no longer financially viable:

> Treatment of hypertension in the developed world relies to a great extent on drug treatment. The cost estimates for drug treatment for 50% of hypertensive patients indicate that developing countries cannot afford equal treatment for their hypertensive populations. Non-pharmacological therapy, avoiding obesity, excess salt and excess alcohol is, in the main, the solution for controlling hypertension in developing countries and, in the long term, in the developed countries as well (Nissinen et al, 1988, p152).

Despite the many studies assessing the relative effectiveness of weight reduction, reduced salt consumption and relaxation training on reducing blood pressure in essential hypertension, the need exists for more evidence that these and other non-pharmacological treatment strategies can be put into practice beyond the well-controlled laboratory study. In the South African Black community little systematic appraisal of these techniques has been conducted,

with the subsequent case that health-care workers have not been encouraged to adopt alternative practices to treat essential hypertension. Medication continues to be the dominating treatment in most community-based programmes and even in private-practice little attempt is made to introduce non-pharmacological treatment into the patient's hypertensive regimen.

As such, it was the intention of this study to assess the effectiveness of using a non-pharmacological approach in the treatment of essential hypertension. This study will document an individual's particular case of hypertension; identify the possible aetiological factors which may be contributing to the sustained high blood pressure; devise and implement a specific "treatment-package" tailored to the needs of the patient, and systematically evaluate its efficacy. In this way, it is hoped that some additional light will be shed on the possible psychological, behavioural and environmental factors which contribute to essential hypertension, and from the evaluation of the treatment regimen, whether or not such a non-pharmacological approach is a viable and feasible alternative or adjunct to traditional pharmacological care.

METHODOLOGY

According to Bromley (1986) the individual case-study, or situation analysis is the bedrock of scientific investigation which has played a central role in the advancement of the knowledge in various fields of investigation. In law, for example, or jurisprudence the establishment of "case-law" lays the fundamental guidelines for its application, while in medicine individual pathology has enriched the understanding of human-health. In psychotherapy, the understanding of the mind and behaviour began with documentation of individual cases which later were generalised into broader conceptual frameworks and applied to the general understanding of man as a collective. However, over the past century the benefits of individual analysis have been overlooked in the pursuit of enhanced scientific credibility through large sample size and statistical research methodology. Edwards (1990b) argues that these large-scale research efforts have done little to extend our understanding or enhance the repertoire of practical therapeutic strategies:

> The fundamental method by which knowledge is advanced in the development and testing of interventions [especially] in cognitive therapy has been the systematic analysis of individual cases. The development of case conceptualisation within the cognitive model as well as the development of specific treatment strategies and tactics owes little or nothing to elaborate research programmes using large sample sizes, statistical analysis and complex experimental designs. Within the current ideology within which research methodology is explicated and taught, this has been lost sight of (p1).

Such a viewpoint has been expressed and supported by numerous methodologists and practitioners in the field of psychology including Kratochwill, Mott and Dodson (1984) who write that:

> Increasingly, researchers...are recognising the importance of case study and single case investigations for the development of a knowledge base...that is unobtainable through traditional large-N-between-group-designs in therapy research (p55).

Polkinghorne (1986) concurs that daily work by practitioners is influenced little by the disciplinary research and that practitioners do not find the research findings helpful, for the findings most often are about group performance rather than particular individuals.

Although it is beyond the scope of this section to include a detailed account of case-study methodology or meta-theoretical analysis, it is essential that a clear contextualisation of the present study in case-study methodology be provided. For it is directly out of the case-study method that the present study extracts its methodological procedures.

Bromley (1986) describes the case-study as an account of a person in a situation, where there is usually something interesting or problematic about the person or the situation or the relationship between them, and the usual purpose of a case-study is to find a **solution** to the person's problem. In this way, the value of such an exercise is that it reveals facts and relationships which make the particular case explicable and at the same time adds to the "case-law" or understanding in that general area of interest. A psychological case-study is essentially a reconstruction and inter-

pretation of an event or occurrence in a person's life, where the facts have to be established through what is in part an exercise in historical research and having to arrive at a verdict (a conclusion, a solution, decision or recommendation) on the basis of a rational argument about the relevant evidence. Of importance, though, is that a case-study is not an exhaustive description and analysis of the person or the situation, but is selective in the sense that it addresses itself to some issues which hold direct relevance to the presenting problem and ignores others. Thus, some facts about the person and the situation become relevant and so contribute evidence, whereas others do not.

> Case-studies are often carried out so that a desirable course of action can be implemented, whether in the interests of the subject, or the investigator(s) or the wider community...In order to arrive at a decision about the best course of action to take, one needs to estimate the likelihood of achieving a desired outcome, and the costs and benefits of implementing the associated course of action (Bromley, 1986, p5).

In this way, supporting or refuting evidence can be accumulated regarding a phenomenon or a particular course of action which has been implemented in finding a solution. Briefly, the study of individual cases is the **idiographic** aspect of personality study which can be contrasted with the study of individual differences - the **nomothetic** aspect of personality study. Often it is supposed that the idiographic approach is concerned only with particular unique individuals. Although this is often one of the aims of case-studies a generalisation of this sort is incorrect. Individual case-studies

can only be described and interpreted in terms of an existing, general conceptual framework with which other cases can also be described and interpreted. As such, there exists a reciprocal benefit in doing a case-study: the study uses pre-existing theory to be made sense of, and provides conceptual support, refutation or alteration to the existing conceptual framework. However, both the idiographic and nomothetic approaches have a common aim - the advancement of scientific knowledge about human nature. The difference between the two approaches is one of strategy or method. The idiographic approach is the intensive study of individual cases with the expectation that detailed description and analysis will gradually lead to a deeper understanding and to practical application in more and more areas of related interest. The nomothetic approach endeavors to study samples of subjects in an extensive psychometric way with the expectation that individual variations can be averaged out to reveal basic factors common to all.

A special feature of case-study methodology is its flexibility to incorporate any evidence which is relevant or important. As such, both qualitative and quantitative data can be utilised as either a heuristic or definitive source. The tendency in the social sciences has been to use the case-study method in association with other strategies of data collection, such as participant observation, life-stories, documentary reports, questionnaires and especially methods which provide quantitative data. If evidence from several independent sources or several independent methods point to the same conclusion, then this adds to the confidence one feels about its correctness (Bromley, 1986). In general sampling-methodology this would be referred to as **triangulation**, while in case-study

methodology this approach provides what is known as an **"extended case-study".**

Results from effective case-methods of inquiry are, by definition valid and reliable and capable of being replicated or otherwise confirmed. The idea that a case-study, even an extended case-study, can only be exploratory, whereas a social experiment or survey can provide definitive results is an incorrect assumption. Case-studies like other methods can generate data relevant to the investigator's theory about, or model of the phenomenon under investigation and can provide data on the generic properties of the class of cases under consideration, even if these generic properties are not fully understood. The case-study method in clinical psychology, as in psychoanalysis, has been based largely on the concepts and procedures of clinical medicine. The present study follows this approach where the emphasis is on the signs, symptoms and history of the factors, which, when properly diagnosed may respond to a specific treatment.

On a pragmatic methodological level, Bromley (1986) outlines a number of steps which need to be followed when a psychological case-study is being prepared:

(1) The first and most important rule is that the investigator must report truthfully on the person, his life and circumstances and must be accurate in matters of detail.

(2) The aims and objectives of the case-study should be stated explicitly and unambiguously.

(3) The case-study should contain an assessment of the extent to which the stated aims and objectives have been achieved. The factors which prevent the attainment of the objectives must be

described.

(4) If, as is so often the case, the inquiry deals with episodes of deep emotional significance to the person, then it can be carried out properly only by someone trained and equipped to establish and manage a close, fairly long, and possibly difficult personal relationship.

(5) The person must be seen in an "ecological context"; that is to say, a full account must be given of the objects, persons, and events in his or her physical, social and symbolic environment. The proper focus of the case-study is not so much a "person" as a "person in a situation".

(6) The case-report should be written in good plain English in a direct, objective way without, however, losing its human interest as a story.

It is all too easy to carry out a psychological case-study and overemphasise the psychological aspects of the case whilst neglecting the biomedical and environmental aspects. As such, this study adopted a holistic inquiry approach where the psychological, behavioural, environmental, and medical factors were taken into account in the diagnosis, with the intention of leading to a multimodal treatment of the disorder:

> The main part, however, is that human behaviour is a function of the interaction between personal characteristics (disposition factors as well as momentary states of mind) and situational factors (constraints as well as opportunities). As we have said, a case-report is an account of a person in a situation (Bromley, 1986, p33).

The case-study method also provides an opportunity to pursue a

quasi-experimental approach to single subject research, where the subject acts as his/her own control group to produce legitimate research findings. When designing an experimental study with only one subject, it is necessary to use some sort of time-series design, where repeated measures on the dependent variable are taken before and after the treatment condition is introduced. The repeated measures of the dependent variable make it possible to detect whether any effect was produced by the treatment condition. If no change in any independent variables other than the treatment can be identified, then one can be sure that the treatment led to the observed differences in the dependent variable, if such differences were observed. In line with Christensen (1980) the present study followed a simple A-B design, where B the treatment intervention was introduced to reduce blood pressure levels following a pre-treatment phase A. Multiple blood pressure measurements (the dependent variable) were obtained during the pre-treatment phase to establish a stable baseline measurement within the person and blood pressure was monitored from the start of the treatment phase to the end of the treatment phase to evaluate the effectiveness of the treatment.

The lengths of the pre-treatment and treatment stages were determined in accordance with following suggestions made by Christensen (1980):

> One of the issues that must be given consideration when designing a single-subject study is the length of each phase of the study...Most experimenters would advocate that each phase should be continued until some semblance of stability has been achieved...The second issue related to length of phases is cyclic variations. Hersen and

> Barlow (1976) consider this a most neglected issue in the applied single-subject literature. This issue is of paramount concern when using subjects that are influenced by cyclic factors such as the estrus cycle in females. Where the data may be influenced by such cyclical factors, it is advisable to extend the measurement period during each phase to incorporate the cyclic variation in both the baseline and treatment phases of the study (p260).

As such, the length of the pre-treatment phase was determined for the following three important reasons:

(1) Enough time to acquire sufficient information on the case.
(2) For some sense of stability in blood pressure levels to be obtained, and
(3) The measurement period had to include cyclic variations such as the menstrual cycle which could possibly influence blood pressure levels, so at least 28 days was the minimum period of time.

The length of the treatment phase depended on the following two factors:

(1) Sufficient time to implement the treatment intervention and
(2) A similar period of time as the pre-treatment stage to include the menstrual cycle.

Although the existing literature does not point to any specific effects of the menstrual cycle on raising or lowering blood pressure in females, this may be a fact or it may be a variable which has been over-looked in previous research. To control for any possible effects of the oestrus cycle on blood pressure levels, its possibility was included <u>a priori</u> into the research design.

Since essential hypertension has a multiple biobehavioural aetiology which differs in character from individual to individual, it is thought that specific treatment regimens tailored to the individual's needs are best suited as a treatment strategy. For this reason, any systematic analysis of such a treatment approach would necessarily acquire an intensive observation of that particular individual who is obtaining that antihypertensive intervention. As such, the case-study method was chosen as the most suitable for this type of inquiry.

It was the aim of the study to document and assess the effectiveness of using a non-pharmacological approach in the treatment of essential hypertension in an urban Xhosa woman who had been diagnosed as suffering from essential hypertension. The study documents the individual's particular case of hypertension, identifies the possible biobehavioural aetiology which may have contributed to the sustained blood pressure, devises and implements a specific treatment regimen tailored to the emergent needs of the patient, and systematically evaluates the effectiveness of the treatment in reducing blood pressure.

The subject of the study was a fifty-one year old qualified nursing-sister who lives in the Eastern Cape district and used to work at a day-care clinic in Grahamstown. The subject - who will be known as Sandi - volunteered to assist in the department's research efforts and subsequently became involved in the present study. At the initial three meetings Sandi and myself talked generally about the aims of the study, the part she would play in the research, and organised a schedule for meeting in the following weeks. In this way good rapport was established which helped facilitate interest

and motivation in the subject. Sandi remained highly motivated towards myself and the study throughout the duration of the study which eventually spanned close to three months.

Following on the recommendation of Bromley (1986) the study needed to acquire as much quantitative and qualitative data in order to diagnose, devise a treatment, and implement the treatment in her particular case of hypertension. Thus, over an initial six-week period following the first introductory meetings, Sandi was met on a regular weekly basis for approximately one hour at a time at the hospital where she worked. During this time, extensive qualitative information pertaining to her life-history and prevailing circumstance was collected via the use of **unstructured interviews, observations** of the subject in her working environment, and through data acquired from **medical records.** In accordance with Edwards' suggestions (1990a, p14), this provided a broad-base of data from which to construct (1) a **Life-history** and (2) a **Lifestyle analysis.**

Each interview was taped and later transcribed and from this a tentative hypothesis was made regarding the possible psychological, behavioural and medical factors contributing to her hypertension [complete transcriptions of the interviews have not been included in this thesis, but are available on request]. Each of these factors was further investigated and evaluated until particular dominating issues were identified as the possible aetiology. From the initial meeting, the subject also recorded her own blood pressure twice a day, in the **morning** and at **night,** on a regular daily basis, using a portable aneroid sphygmamonometer which she carried with her throughout the study. The blood pressure readings were documented in a personal diary which she kept. This diary was

also used to note any special events which occurred on a daily basis and which the subject felt were important factors raising or lowering her blood pressure on that day. In this way it was possiible to acquire extensive, rich data on the psychological characteristics of the patient's functioning and to monitor her blood pressure over an extended period of time.

Since dietary behaviour has been implicated in raising blood pressure, a typical **eating schedule** for a period of two days was obtained and analysed for elevated salt consumption, cholesterol consumption and caffeine intake. The subject's **body mass** was also measured and recordings of her weight were recorded on a regular basis and checked against a weight-chart for indications of over-weight and obesity. A **fasting-cholesterol count** was also obtained by the subject's general practitioner to assess the likelihood of elevated cholesterol levels in the blood and possible blocking of the arteries.

In addition, various self-report inventories were used to collect information on the psychological functioning of the subject and these helped guide the researcher to identifying the possible psycho-behavioural component(s) responsible for raising her blood pressure. The following self-report inventories provided valuable data on subjective functioning:

(I) **Beck Anxiety Inventory** (II) **Zung Anxiety Inventory**
(III) **Novaco Anger Inventory** (Novaco, 1975)
(IV) **Weiman Occupational stress scale** (Weiman, 1977)
(V) **Rotter Locus of Control scale** (Rotter, 1966)
(VI) **The Taylor Manifest Anxiety Scale** (Taylor, 1953)

(VII) **Dwight Dean's Social support Schedule** (Dean, 1961)

(VIII) **Rathus Assertive Behaviour Assessment scale** (Rathus, 1973)

The Beck and Zung anxiety inventories were also used throughout the study as indications of general anxiety and over-arousal, and as a subjective measurement of psychological and physical well-being as a result of the treatment. The daily blood pressure record over this six-week period provided a pre-treatment baseline blood pressure measurement. This pre-treatment data collection phase was concerned only with obtaining information and at no time was any therapeutic suggestion or intervention implemented.

From constructing the life-history and lifestyle analysis a (3) **Case-conceptualisation** was the third step (Edwards, 1990a, p14), where target psycho-behavioural factors were identified as playing a possible role in the aetiology of the hypertension and combining this with an analysis of the literature on non-pharmacological treatment strategies available, a specific treatment regimen was devised. In step (4) the **Therapeutic intervention** was implemented in line with Edwards (1990a, p14) over a further eight weeks and systematically evaluated for its effectiveness by comparing the pre-treatment blood pressure levels with those from the treatment phase, assessing the general level of psychological and physical well-being using the anxiety inventories over the duration of the study, and by obtaining further qualitative data from the therapeutic sessions by interview. The latter also provided an additional subjective appraisal of the treatment regimen and a heuristic data source for evaluating the effectiveness of the treatment programme.

LIFE HISTORY

Name of Patient: **Sandi**

Date of Birth: **1 June 1939**

Age of Patient: **51 Years**

Occupation: **Nursing Sister**

Marital Status: **Divorced**

Number of Children: **Three**

Onset of Hypertension: **1986**

History of Familial Hypertension: **Mother, Father and Sister**

Anti-hypertensive Medication: **Valium, Capoten. Tritace, Natrilix**

Average Blood Pressure level at start of study: **154/100mmHg**

Known physiological dysfunction /disorder: **None**

World Health Organisation Classification: **Mild Essential Hypertension**

Sandi is a 51-year-old, Xhosa nursing sister who was suffering from mild essential hypertension. She was born in Alexandria, a small farming town in the Eastern Cape in 1939 to a relatively poor family, but was educated in a multi-racial convent in Uitenhage where she obtained her secondary education. From here she moved to Cape Town where she obtained a Teaching Diploma at the local Teachers Training College and thereafter taught near Salem [another small community town near Alexandria] where she met her husband to be who was also a teacher. Sandi was married at the age of twenty-two years and had the first of her three children in 1961; the second child in 1963 and the third child in 1966. Since the Teaching profession did little to encourage married women to work and provided relatively little occupational security, Sandi decided

to pursue Nursing as a career and in 1968 left her family in the care of her husband and relatives and studied nursing at Livingstone Hospital in Port Elizabeth. Soon afterwards her husband and children moved from Salem to Umtata in the Transkei where he was offered a teaching post. Sandi found it very difficult being away from her children and so transferred her training to Umtata and moved back to her family. Once she had completed her basic nurse's training, the family again moved, this time to Alice in the Eastern Cape, where Sandi completed her Midwifery training and practiced at the local hospital for a further ten years.

At this time (1985), she divorced her husband and relocated to her present location in the Eastern Cape [which will remain un-named to ensure anonymity], while her three children pursued careers at university and college after they left home. It does not appear that Sandi's hypertension was related to or began at the time of her divorce since no changes in blood pressure were observed at this time. However, she did experience a certain amount of anxiety around the divorce proceedings and considerable anger towards her husband for being irresponsible towards her and the children. It was because her husband had become irresponsible that Sandi had decided to divorce him, yet this decision was not sudden but a response to a gradual decline in their marriage over a number of years. Although she lives alone at present, her mother used to live nearby before she died and two sisters, whom Sandi visits often, have remained in the town. Her children also visit during vacations and, in general, she claims to be quite content with her situation.

According to the patient, her case of hypertension began some four years ago when she moved to her present location and began working

at the local hospital. Sandi ascribes the onset of her hypertension to the working conditions and to the adjustment which she has had to make to the stressful occupational demands:

> I had been working at Victoria Hospital in Alice for ten years. I was doing orthopedic work there and was only concerned with orthopedic patients, although I was allocated to the "out patients" department...It didn't entail much work...But here it is a terrible situation. Here I was supposed to be assistant to all the out-patients regardless of it being orthopedic or not. In fact I was not orthopedic at all. I left off that part and started with ordinary work of casualty at "out patients"...I had to adjust myself to this, now you know, and I started developing hypertension.

When Sandi first became aware of her hypertension she experienced dizziness and extreme headaches at her place of work. Often she would have to rest for periods of time in the adjacent ward and no tranquilizers helped calm her down and reduce her blood pressure:

> Here I was feeling this hypertension and started having headaches, headaches, constant headaches and one day I took my blood pressure which was about 170/110mmHg and unfortunately nothing would take it down; valium, all the sedatives, nothing would take it down. I was made to rest in the side-ward. I tried this and that and at last I tried a new drug which the doctor knew would be helpful...The drug started to take it down nicely, but it was very expensive. I asked the doctor if she had a substitute for this drug, which is acting the same way but is less expensive. This is why doctor is trying me

on this new drug.

Although no additional records exist which support the patient's claimed onset and duration of hypertension, it can be accepted with near certainty that the patient's report is accurate and reliable, taking into account that the patient is a trained nursing sister who is intelligent, aware and astute at her job. Further circumstantial evidence was provided in additional interviewing which corroborates her previous report:

> I have known since 1986 that I have had hypertension. I moved from Alice to _ . I moved in June 1986 and by the end of the year I had to have medication...Whenever I was sick during my stay in Alice my blood pressure was taken and I never had high blood pressure...I don't foresee that it can wear off while I'm still in this situation.

After nearly two years of working at the hospital, Sandi requested a transfer to a quiet clinic in the town, where normal working hours were maintained and the level of experienced stress was reduced considerably. Although her blood pressure never returned to normal, the medication succeeded in controling it at near normal levels and only occasionally did it "flare-up" when she experienced extra stress either at work or in her personal life. Sandi remained at the clinic until approximately three weeks before the present study began and then was transferred back to the hospital due to a chronic shortage of staff. When systematic blood pressure monitoring commenced three weeks after her leaving the clinic, her pressure level had risen to an average 154/100mmHg, with no concomitant changes in medication.

Of further importance to this case is the presence of a family history of hypertension. Both Sandi's mother and father apparently suffered from high blood pressure:

> In fact my mother was hypertensive and my father died of arteriosclerosis in 1962. I think that was due also to that hypertension because his blood vessels were narrow. So he was hypertensive and my mother died of high blood pressure here in hospital last year. So I think its familial and my age, I think I should have it now.

During the course of the study, Sandi's older sister was hospitalised for a sudden acute attack of high blood pressure with no known physiological cause. According to Sandi her sister had not suffered from hypertension previously. The sister remained in hospital for over a week and was still on medication by the end of this study.

The first anti-hypertensive medication which Sandi was prescribed was **Capoten,** at 50mg twice a day, which is a strong Angiotensin-Converting Enzyme Inhibitor. But due to its high financial cost this was replaced by **Tritace**, at 2,5mg per day, which is a similar ACE Inhibitor that is far less expensive. Although the relative strength of the two drugs is comparable, the dosage of Tritace was insufficient to control her blood pressure and an additional medication **Natrilix**, at 2,5mg per day, was added to her medication regimen. Natrilix is a thiazide diuretic with a maximum suggested dosage of 2,5mg per day. Sandi had already been on this combination of medication for close to three weeks before this study began and remained on the said regimen for the duration of the study.

According to the pharmacokinetic action of both drugs, maximum absorption and peak concentrations in the blood are reached after

two hours [for information on the medication, refer to scheduling pamphlets in Appendix 1]. At no time was any additional non-pharmacological treatment, such as diet modification or weight reduction, suggested or implemented by the doctors, as far as could be ascertained from the patient.

It is evident that her case of hypertension was resistant and stubborn, when after three weeks her blood pressure had only been reduced to and controlled at 154\100mmHg on medication.

LIFE-STYLE ANALYSIS AND CASE-CONCEPTUALISATION

It became increasingly apparent that Sandi placed much importance on the stress of her occupation as a factor which raised her blood pressure. In the interview sessions to come, she spoke at length about her job and how she perceived it to be stressful. Since much of her daily life was spent at the workplace and her occupation filled the largest part of her life, it was important to assess in as much detail as possible the extent to which occupational stress played a role in raising her blood pressure. At the beginning of the study Sandi was administered the Beck and Zung anxiety inventories, which displayed recurring incidents of "nervousness", feelings of "loosing control", and feeling "panicky":

> When you are somebody who likes things to go right all the time, you know, always, all the time and you see that things are not going right you become panicky. You explain how things should go and then the next thing you find they are not done the way they are supposed to be done. Then I start flaring up and become angry. I shout at them [the other nurses]...

> It's the pressure of work really. And when things are not done, you know properly. We are on friendly terms, but when it comes to work not being done, then I forget about that...This [smacking the arm-chair], I'm angry. I would like to smack somebody, I would like to smack it...I don't know why you have to repeat things over and over. I don't know if I'm taking things too much, because perhaps if I could take things lightly I wouldn't be uhm...It's because I'm a person who likes to have things done rightly, that's the problem. I like things to go right.

Sandi expressed great frustration about her working situation and how the staff were often unproductive, lazy and irresponsible. This went against her perceived personality traits of being punctual, hard-working and responsible. Clearly, this conflict produced frustration and anger, which often manifested in emotional outbursts in the ward and the nursing staff being scolded. She also exhibited a lack of patience with the nurses, with her pent-up frustration having no outlet other than an angry outburst. However, Sandi was well aware that she became angry often, how it felt and that being angry on the job was not always in her best interests:

> I don't think it's right [getting angry], but I can't help myself, I don't know, I can't help myself. It's because of the working situation makes you do like that. If it was less, peaceful situation, enough working staff, everybody peaceful it would go alright ...When I'm angry, I feel it in my head. It feels hot and big. A big hot head and flushing face. Tense. And I shack it. It grows big, man. But it is of short

duration, it doesn't stay long. I'm very grateful for that, that it doesn't stay long. I just get upset there and then and it wears off soon....I work with good people. They understand me, you know. They don't take me otherwise. Sometimes you get people who would resent being spoken to in a harsh way. They would prefer that you call them outside and say things privately and so on and so on....I would like to do it like that, but I can't stop. Like you know, in a soft way, like: please could you...in a normal way.

It appeared that the work situation and its associated stress was a possible contributing factor to her hypertension, yet it was unclear why such stress had not been experienced previously at any of the other hospitals where she worked. During a subsequent interview Sandi raised this point and said the following:

I found myself to be like this [angry] all of a sudden, it's not in me, I'm not that kind of person, I'm so calm and like to make jokes. I've got so many friends, people like me; I don't know why they should (laughs). Perhaps it is because where I was before [Victoria Hospital in Alice] we had student nurses and when you have students training, the students really depend on you for their training...When I came here I said: Oh well I'm going to relax. Everyone knows everything. Student nurses are student nurses, you take their reasons, they are students, you know yourself when you were a student. I know you can accommodate their mistakes. But when it's people who are already trained, you don't know. I mean I don't know what to do. I'm puzzled.

> Perhaps it's one of the things that has made me be this way [angry]...I cannot take it, I am going to go away from this hospital...I must have a place where I can relax. Come and feel content and happy. I can't be subjected to pressure all the time. Since I have come to this place I have not been [happy]. I am forcing myself to work, because it is work and I must earn a living. I want to be recognised for the work I do. They don't appreciate what people do, they don't encourage you. I don't like working for this hospital. It is better when it is a training hospital, a training college. Things are right, they go according to rules. You know what and what and what, everything is clear. I must not make myself suffer...I'm not taking working alone anymore. As long as it is going on they will say she is coping, she's alright, she doesn't complain, she's alright, she's a good one. I'm not a good one as far as that is concerned. So I don't want to talk much about these things because it seems as if I have got so many complaints to you. Complaints, complaints; a person who doesn't take things as they come. But I can't take it like that. They must be aware.

It is possible that much of the current occupational frustration and stress may be traced back to unfulfilled expectations about what the job was to entail and how Sandi experienced the staff compared with what she expected. The experienced stress in the workplace may be a result of this conflict between her expectations and lived-reality and resulting frustration. It further appeared that most of Sandi's stress was limited to the working environment:

> All these things occur at work. I don't get any flaring up and shakiness when I am off duty. All these things occur at work, it is the work situation to sum it all up, really. It is not in my nature to get like that, but when I come to work then it starts things off. I can't think of a time at home when I flared up. That's why I said to you, when I'm at home these situations do not occur. I don't talk to anybody and there is nothing that upsets me really. I make my own things, make my own food. I do my own things. When it's going to be tomorrow I regret about getting up in the morning. Although I do my work properly. I don't neglect my work. I lose interest, I lose interest which I used to have. I have to work again, frustrating work ....No flare ups when I visit friends and nothing when I go shopping to town.

Further data were collected to assess what factors may have been contributing to Sandi's anxiety and occupational stress. On the **Taylor Manifest Anxiety Scale** she scored in the normal range with a score of 19 (score <= to 19: less anxious than the average person), indicating a relatively low level of generalised anxiety. It was hypothesised that most of Sandi's experienced anxiety was related to her work, which was further supported by testing on the **Weiman Occupational Stress Scale** [refer to Appendix 1] where she scored particularly high with a score of 3,2. According to statistics provided by Weiman (1977), approximately 97,7% of people in his sample scored less than 3,2, while only 2,3% of subjects reported scores greater than 3,2.

There was a possibility that sources other than the workplace may

have exacerbated her experienced stress. According to Greenberg (1983) occupational stress is often the result of or is exacerbated by a lack of broader familial and social support networks and by a self-perceived lack of social support. Since Sandi was living on her own it was speculated this may have contributed to her high levels of occupational stress. However, her subjective rating of social support on **Dwight's Social Support Schedule** indicated no lack of social support. She scored a total of 15 with the scale ranging from 0 to 36 (the higher the score, the more socially isolated the person believes herself to be) [refer to Appendix 1].

Often occupational stress is characterised by a lack of assertive behaviour on the part of the stressed worker with the result that personal opinions, beliefs and feelings are not expressed and lead to a build-up of internal frustration and anxiety. Perhaps Sandi's occupational stress was partially a result of an inability to express herself assertively in the workplace, however, no such indication was evident from the **Rathus Assertive Behaviour Inventory**, where she scored a total of 39 with the scale ranging from -90 (lack of assertive behaviour) to +90 (complete assertive behaviour) [refer to Appendix 1].

Lastly, Greenberg (1983) suggests that another factor which can lead to occupational stress is an inadequate internal locus of control. If a person does not believe that he has control over what happens and that he is unable to determine his own life but believes that everything is manipulated and determined by external forces, then the person experiences such things as low self-esteem and powerlessness. In the workplace this often leads to stress, when the person feels inadequate and incapable of altering his own

circumstances. On the **Rotter Locus of Control Inventory**, Sandi showed no inadequacy on internal locus of control and scored relatively high on internality with a score of 15 (scores greater than 10 reflect internality with the highest score being 20, while scores less than 11 reflect externality with the lowest score being 0) [refer to Appendix 1].

It appeared that most of Sandi's stress and anxiety were associated with her occupation. Similarly, it was evident that her stress on the job was not a result of a poor internal locus of control, nor due to a lack of assertiveness or to inadequate social support, but rather due to unfulfilled expectations about her work position and resulting frustration and anger from this conflict. It seemed that this frustration and lack of occupational-satisfaction led her to angry outbursts and "flare-ups" which were contributing to her raised blood pressure levels and that this inadequate and often inappropriate mode of expression was an important aetiological factor in her hypertension. It was confirmed by **Novaco's Anger Inventory** that Sandi was in fact experiencing high levels of anger. Her score on the inventory before any treatment was implemented was 398, with the scale ranging from 90 to 450.

[It must be remembered that all the subjective rating scales used, including the Beck and Zung anxiety scales were originally constructed and provided normative data on predominantly middle/upper class, first-world American or British university students. This may reduce the validity of the scales when used cross-culturally such as in this study, however, since the present subject is well educated, articulate in the English language, and certainly first-world in her outlook and lifestyle any reduction in validity would

probably be minimal, and since the scales were merely used as adjunctive devices to analyse subjective functioning and not to make a decisive diagnosis, cross-cultural validity of the measures poses no real problem.]

Although stress has been identified as important in the aetiology of essential hypertension, other factors such as physiological disorders, diet, high levels of cholesterol and being over-weight can play an important role in raising blood pressure levels. Sandi had no apparent physiological malfunctions or known hormonal disorders, so secondary hypertension was ruled out and a diagnosis of essential hypertension could be made with certainty.

A typical diet-schedule obtained over a two day period reflected no unusual dietary habits which could have caused raised blood pressures [refer to diet schedule in Appendix 1]. Sandi's consumption of dietary salt was minimal since she hardly ever cooked with salt and never used salt at the table. Also, her consumption of caffeine was negligible and limited only to drinking tea. Sandi also did not smoke. She did eat red-meat and prepared her dishes with a saturated fat called "Holsum", which is known to be high in cholesterol. However, a "fasting" cholesterol blood count taken by her General Practitioner showed no elevation of cholesterol in the blood (cholesterol count = 6,3mmol/l) and was within the normal range of cholesterol for her weight and age (3,1 -6,5mmol/l) [refer to Appendix 1].

At the start of the study, Sandi's weight was assessed and measured 76kg which was approximately 9kg over-weight for her medium frame and height of 170cm, according to the weight-table provided by

O'Brein and O'Malley (1987, p57). Thus, it appeared that besides stress and inappropriate emotional expression Sandi's excess body weight could have been a further aetiological factor in her case of hypertension.

To conceptualise this particular case requires a synthesis of the information gathered from the life-history and the life-style analysis with the current thinking and treatment strategies provided by the theory and literature. It became apparent from the information collected in interviews, through self-report inventories and from other sources that at least three important factors could have been operating to maintain the high level of blood pressure seen in Sandi's case. She was experiencing much frustration and anxiety stemming from unfulfilled expectations and excessive demands placed on her in the workplace which was being expressed in recurring incidents of angry behaviour on the job, which was hypothesised to be the main contributing factor in her hypertension.

The role of stress in the aetiology of essential hypertension has already been discussed in the introduction to this study, however its importance needs to be recapitulated. The effects of stress on the central nervous system are an increased secretion of hormones and the consequent elevation of blood pressure. After short periods of exposure to stress the body usually returns its hormonal levels and blood pressure levels to normal, but when stress continues unabated for extended periods of time, the theory suggests that a increase in the homeostatic level of blood pressure occurs. It is possible to reduce blood pressure levels through pharmacological and non-pharmacological means albeit not permanently as a cure, but

as a means of control.

If stress is a factor in the aetiology of hypertension, then an attempt must be made to alleviate the source of stress or at least to reduce the experience of it. It was not possible in Sandi's circumstance to suggest that she leave her job nor was it feasible to change the working situation. However, it was potentially possible to alter the way stress was experienced and how stress was coped with, in the hope that this would reduce her stress level and ultimately reduce her blood pressure level. Extensive work by Benson during the 1970's and 1980's, found that meditative relaxation evoked the "relaxation response" which counteracted over-stimulation of the nervous system. This was observed to aid in the reduction of stress and decrease blood pressure levels. To treat Sandi's general stress, a programme of simple relaxation was designed according to Benson's technique (1975, p158) which could be practiced on a daily basis, was easy to learn and required only twenty minutes per day. Although many different relaxation techniques are available, it was decided to use Benson's Relaxation Response for two reasons: (1) the researcher was well acquainted with the method and practiced meditative relaxation personally and so patient instruction would be simple and (2) of all the relaxation techniques, the Relaxation Response appears to have withstood the test of time and is considered one of the most effective relaxation methods to reduce heart rate, blood pressure, oxygen consumption, respiratory rate and to increase pleasant and relaxing alpha waves in the brain (Benson, 1975 & Barr et al, 1984). It was also hoped that a schedule of daily relaxation would help counteract some of the patient's excited and angry emotional reactivity to her working conditions, reduce her anxiety level, and

promote general psychological well-being.

However, relaxation training cannot be regarded as a panacea in itself, but rather as one type of cognitive-behavioural adjunctive treatment strategy with broad-based positive effects. In Sandi's case relaxation training alone would not have been adequate, since "anger" presented as the single most important factor in her case and required therapeutic intervention beyond simple relaxation therapy. A treatment intervention was also designed and implemented to reduce the level of anger, with the hope that anger-reduction would help lower blood pressure.

Recently, the possibility that chronic anger can raise blood pressure and contribute to hypertension has come to the attention of psychologists. Novaco (1975) conducted extensive research into the emotion of anger and found a relationship between anger and the occurrence of hypertension. Using a student population of individuals suffering from a high incidence of anger, in addition to formulating the first comprehensive 'anger-therapy' based on principles previously devised by Meichenbaum (1972), Novaco found that **stress** can be understood as an imbalance between environmental demands and the response capabilities of the person or system to cope with these demands. It is asserted that people who are prone to provocation and are without the psychological resources for coping are susceptible to health impairments (eg, cardiovascular disorders), to diminished work efficiency and to disruption of personal relationships (Novaco, 1975, p137).

Anger can be understood as one type of emotional and behavioural response to external stressors and environmental demands which

cannot be coped with successfully. In other words, anger is an affective stress reaction to aversive events that are called **provocations** (Novaco, 1975, p139). Consistent with psychological views of stress, anger is determined by the person's cognitive structuring or understanding of the situation and an attempt to extort a validation of one's personal constructs in an effort to control aversive situations. Thus, anger can be understood as an attempt to control and cope with a stressful situation, usually by an initial arousal and then by directing an emotional outburst towards another person, group of people, to oneself or to the situation. It is this increased state of arousal that accompanies anger which over-stimulates the nervous-system and is believed to result in raised blood pressure. Often, anger becomes an exclusive mode of coping and of emotional expression, with the relative exclusion of other coping abilities.

It appeared in Sandi's case that anger was being used as a primary mode of coping and emotional reactivity to the stress in her workplace and so a therapeutic intervention, based on Novaco's Stress Inoculation Model, was designed which could provide her with **insight into her angry behaviour, increase her repertoire of coping skills, and reduce her arousal.** In terms of Novaco's conceptual-isation of anger, the angry experience can be divided into four separate provocation stages where at each stage cognitive-behavioural modification of the **appraisal** and **response** to the stressful provocation is made. These stages follow a temporal sequence from (1) **preparing for a provocation** to (2) **confronting the provocation** to (3) **coping with arousal and agitation** to (4) a stage of **self-reward.** Of prime importance is the "self-talk" which accompanies each stage and is associated with both the appraisal of

and response to the provocation. The patient is directed to alter the negative and deprecatory "self-talk" at each stage and to increase positive self-statements and thereby increase her perceived ability to cope with and handle the provocation and reduce her anger. It is the persistence of negative self-statements and an inability to make positive self-statements and thereby prevent a positive personal belief in coping which throws the person into an angry reaction.

Scholten, Maes, & Van Elderen (1990) have also found a relationship between anger and hypertension and further divided anger in their theoretical understanding into four components: (1) **Anger-In,** (2) **Anger-Out,** (3) **Control Anger-In** and (4) **Control Anger-Out,** where Anger-In refers to the internalisation of anger whereas Anger-Out refers to the externalisation of anger. Control Anger-In refers to control over internalised anger and Control Anger-Out to control over externalisation. Stated in simple terms, "anger-in" describes a situation where the person is highly critical of him/herself and directs blame and angry emotion towards him/herself, sometimes in an excessive manner. "Anger-out" denotes the situation where the person externalises criticism and blame, and expresses angry emotion towards others, sometimes in an uncontrollable manner. "Control anger-in" represents an ability to control, moderate and limit the amount of anger directed towards the self using some type of active strategy to reduce arousal, while "control anger-out" represents an ability to moderate the amount of anger expressed towards others by responding in less antagonistic ways and by redirecting the "arousal-energy" into other more constructive actions.

It was hypothesised that people developing high blood pressure must be frequently angry (High Anger trait), must hold their anger in (High Anger In) and must fail to control (bring down quickly) the level of their suppressed anger (low Control Anger-In). However, findings from their study showed that patients scoring **low on Control Anger-In** and patients scoring **high on Control Anger-Out** had a significantly higher variability in systolic blood pressure before exercise, while patients scoring **low on Anger-In** and patients scoring **high on Anger-Out** had a significantly higher variability in systolic blood pressure after physical exercise. It is also possible to conceptualise the present case into this new theoretical scheme, with Sandi representing **low on Anger-In** and **high on Anger-Out.** This suggests that anger does not necessarily have to be directed inwards towards the self but can occur solely with a high expression outwards, which was evident in the present case and that extensive use of anger as a mode of expression is associated with a rise in blood pressure and with hypertension. The therapeutic intervention was also targeted at increasing Sandi's "control anger-in" and increasing and modifying her "control anger-out" response via the principles of cognitive-behavioural modification.

Simply stated, anger is not caused by the external situation itself but is a response to the situation as a result of an appraisal of that situation. Thus, in addition to alterations in self-statements, the therapeutic intervention was designed to modify the way in which an event was appraised, if it was appropriate to respond to that situation in an angry manner, and if not, then how to cope with stressful events in alternate ways, such as remaining relaxed, being assertive, or using problem-solving strategies to find the

best coping solution to a stressful situation. Novaco (1975) also suggests the inclusion of relaxation training in anger-therapy as an adjunct to cognitive-behavioural modification, which was to be achieved through the use of regular meditative relaxation sessions with the patient and on-going meditative relaxation by herself in the home and in the workplace (For a detailed description of Novaco's Stress Inoculation Model and treatment strategy refer to Novaco, 1975 and Novaco in Foreyt and Rathjen, 1978).

The possibility that being over-weight was a factor in the aetiology of Sandi's hypertension also had to be included in the case-conceptualisation. The contribution of obesity and being over-weight to hypertension has already been discussed at length in the introduction and was identified as a target for treatment in Sandi's case. In the Framingham Study, it was shown that three times more people with normal blood pressure who were over-weight developed higher blood pressure than expected and that weight loss of 15 per cent or more in men was associated with an average 10 per cent decrease in systolic blood pressure. Other studies have provided similar results and have shown that people with high blood pressure who reduce weight need fewer drugs to control their blood pressure (O'Brein & O'Malley, 1987, p56).

However, during the pre-treatment phase of the study Sandi had her teeth extracted which lessened her consumption of most solid foods over the course of the study and reduced her overall food consumption markedly. This had the effect of reducing her weight significantly during the study so any additional weight-reduction programme - as part of a treatment intervention - was deemed unnecessary.

THERAPEUTIC INTERVENTION

The entire duration of the study spanned approximately fifteen weeks with the first seven weeks comprising a pre-treatment period where a baseline blood pressure level was obtained and information was collected to determine the possible aetiology of the hypertension. At no time during this period was treatment or advice of any sort provided, while the patient's medication regimen remained unchanged. The treatment phase of the study began in the seventh week, where two one hour sessions were held to teach the patient meditative relaxation. A synopsis of the relaxation training was provided to the patient, in written form, for reference purposes and contained a step by step account of the relaxation process [refer to Appendix 2]. A meditation music-tape was also provided for home use to facilitate concentration during the relaxation. In the remaining weeks of the treatment phase, a one hour relaxation session was held each week with the researcher-therapist to facilitate optimal learning of the technique, provide guidance and to supply encouragement and reinforcement.

In addition to one "anger-therapy" session in the first week of treatment, every second week beginning in the eighth week, a one hour "anger-therapy" session was provided giving in total fourteen therapeutic sessions including relaxation training.

The first "anger-therapy" session was unplanned and was only due to be held in the second week of the treatment phase. However, Sandi presented as extremely distressed, in the first week at the second relaxation session, after being informed that day that she would have to do a three-month period of night-duty commencing two weeks

hence. This angered her greatly, which was apparent from her harsh antagonism towards the hospital system and towards the matron who had informed her of the new routine. She was unwilling to accept her predicament, often flushed in the face and periodically slapped her hands on the arms of the chair and looked around the room with an angry, glazed look. After some time of complaining about this "injustice", Sandi became sad and full of self-pity and asked me over and over why she had to do night-duty when the hospital knew she suffered from hypertension and that night-duty made things worse. She continued to explain how bad the last experience of night-duty was (approximately two years previously) and how she could not cope with the number of patients and that her blood pressure rose to very high levels. She informed me that following our session she was to speak with the matron, tell her how she felt about doing night-duty and how it raised her blood pressure and attempt to change her new schedule. However, she was extremely anxious about her meeting and wanted to know from me how she should conduct herself taking into consideration that she often flared-up and became angry. She also expressed an inability to cope with the situation on her own and needed my assistance.

I asked her what she intended saying to the matron, to which she replied that the matron must do something to change the decision or else she would tender her resignation. I said that this may not be the best way to approach the matron, but that she did have the power and the ability to cope with the meeting is she remained calm and spoke to the matron in a reasonable manner without losing her temper or becoming antagonistic. I asked her whether resigning was really what she wanted, to which she answered that nursing is what she wanted to do and was quite prepared to work normal day-time

hours, but not at night in a busy ward with a lack of assistance, because the stress of the job raised her blood pressure. I suggested that perhaps saying that she would resign would anger the matron and that it would be better for Sandi to focus on telling the matron the truth about how she felt towards doing night-duty and that it raised her blood pressure. I again told her that she was strong enough to do it alone and had the ability to cope with the meeting and made her repeat these words aloud. She repeated what I said and agreed that she 'might' be strong enough to do it alone, then changed the 'might' to 'I am strong enough to do it alone without becoming angry'. I told Sandi that there was still a possibility that in the end she would have to do night-duty, but that it was important to tell the matron how she felt and give her reasonable justification for not doing night-duty, without becoming aggressive and antagonistic and then leave the decision in the hands of the matron. I suggested that she remain calm and relaxed, take some breaths of relaxation and not become antagonistic for this might only enrage the matron, make her feel unsympathetic and unconcerned. To this, Sandi agreed.

Compared with the start of the session, Sandi had calmed down markedly and wanted to continue with the planned relaxation training, which we did. She felt sure that following the relaxation she would be in the right 'frame of mind' (relaxed) to face the matron. I then asked her how she rated her level of anxiety on a subjective scale of 1-10 compared with at the beginning of the session. She replied that at the start she 'was a **10**' and now she felt 'like a **3**'. Using such a subjective rating-scale or **Subjective Units Of Disturbance Scale** (as it is more technically referred to) is a recognised strategy in cognitive-behavioural clinical practice

and can provide useful information on the extent to which a particular emotion is being experienced.

Of significance was the fact that on that Wednesday her blood pressure had risen to 150/100mmHg compared with the previous day's measurement of 130/90mmHg. Of therapeutic importance was to empower the patient by substituting negative self-statements for positive and reinforcing self-statements, to highlight the fact that often anger is not always the most appropriate form of behaviour in that it often enrages other people and sometimes worsens an already tense situation, and that remaining calm and relaxed facilitates rationality and reasonableness and helps keep a 'cool head' and enhances coping.

Initially during this session, Sandi was highly aroused and needed a time to cathect or release her energy before it became feasible to proceed with therapy. For this reason, the therapist merely provided empathy and occasionally mirrored questions and answers to the patient for clarification, in a Rogerian manner. Later, when the patient had calmed, it was necessary to proceed with some "problem-solving" therapy as described by Spiegler (1983) which centred upon leading the patient through a series of possible solutions to her problem of "night-duty" accompanied by expression and discussion of related feelings and emotions. In addition, an initial attempt was made to modify the patient's understanding of anger and expression of anger to others, in line with the 'Anger therapy" principles advocated by Novaco (1975). Finally, a period of relaxation-training further calmed Sandi and reduced her general arousal and level of anger.

At our following meeting, Sandi expressed that all had gone well, that she hadn't flared-up and had remained calm during the meeting with the matron. Although she would still have to do night-duty, the matron had been most supportive and understanding and had said that Sandi should try the night-duty and that if need be, she could stop if she was unable to cope with the stress. At a session two weeks later the following conversation occurred:

Therapist: You felt very angry at the time when you heard that you had to do night-duty?

Sandi: Jaaaa [Yes]. But matron explained everything, and why I had to go on night-duty.

Therapist: Was this a good enough reason for you?

Sandi: I think it was good. There is a shortage and a need for more staff. A lot of nurses have left and have not been replaced. But they should have told me, warned me before, at least a month before going on night-duty.

Therapist: It is now two weeks after you started night-shifts. How do you feel about having to do night-duty?

Sandi: It's not bad, so far...It's not as heavy as it used to be. I feel different somehow, that I'm taking things in a calmer way. I think therapy is changing me. It's good.

The subsequent "anger-therapy" sessions followed the basic therapeutic process as outlined by Novaco (1975):

(1) where the patient was informed about what anger was and that anger is a person's response to a situation and not something caused by other people (session 2)

(2) A synopsis of Anger Management Principles was given to the patient and discussed in detail [refer to Appendix 2 for a copy of

the synopsis] (session 2)

(3) The different stages of the angry-experience, as described by Novaco, were also discussed at length. Particular examples of anger experienced by the patient were used to illustrate the different stages of an angry experience and a variety of appropriate positive self-statements were provided for each stage [refer to Appendix 2 for Novaco's self-statements]. In addition, the patient was encouraged to create her own positive self-statements which were rehearsed until the patient had attached emotion to the statement and had "owned" the statement. If the words seemed to mean nothing to the patient, then the statement was altered or substituted by another (sessions 3-5).

It is impossible to include transcriptions of, or discuss each session in any particular detail as this would merely prove to be laborious and redundant. However, for heuristic purposes and as an example of anger-therapy, the transcription of session 4 has been included in its entirety. [Transcriptions of therapy sessions often provide useful protocols from which to extract an understanding and appreciation of the therapeutic principles used, especially when they may be unknown to a clinician of another theoretical persuasion. This type of data also provide an insight into the person's behaviour and thought patterns which either subjective scales or superficial questionnaires are unable to provide]. All other sessions followed a similar format and included the therapeutic principles outlined in (1-3) above:

Sandi: I'm just thinking that you are asking me about an angry event. Somebody says you're stupid and everybody looks at me and laughs.

Therapist: You're getting hot and angry. What would your "self-talk" be?

Sandi: You're starting to be smart, you're always trying to be smart. I'm not being stupid.

Therapist: How are you feeling towards the person?

Sandi: Angry. I don't want to talk to him. I march out.

Therapist: What would you like to do?

Sandi: I would like him to apologise. Is that the right answer? That is what I would do.

Therapist: What would you like to do to him?

Sandi: Call him names too. Call him stupid too. But I know that is not the right thing to do. I will tell him, since its a provocation, yes I can see it coming. I must try and avoid a confrontation. I will ask him to stop it.

Therapist: And what would you do to help yourself in this situation?

Sandi: I will tell myself, Hmmm don't mind him, ignore him. Ignore him as if he said nothing. People will all be looking at him and I'll pretend he's not there.

Therapist: How does it make you feel by doing this?

Sandi: I'm trying to control my anger, I'm trying not to be angry. I feel that if I don't proceed with the provocation it will stop where it is. I know I can do it.

Therapist: What other positive things could you be saying to yourself during this provocation stage to help?

Sandi: I know myself, I will not be taken in to this. I will not let him go on with this as much as I can, because if I answer back, I become aggressive [silence].

Therapist: How about taking the time for a few deep relaxation breaths. Would that help?

Sandi: Yes, yes it would help. After a few deep breaths I feel better, I feel more relaxed. The flushing of my face has gone.

Therapist: Let's pretend he becomes wiser and doesn't stop, but goes on provoking and saying things to you. You can feel yourself getting hot again and a bit angry.

Sandi: How should I act?

Therapist: How would you act, not how should you?

Sandi: I would say to myself I know that he wants me to come out with something and I'm going to disappoint him. I'm not going to do that. He wants me to fight and I'm going to channel my anger into something else.

Therapist: How would you do that?

Sandi: I would try and change the subject [laughs]. Hmmm.

Therapist: How about saying some positive things to yourself, like 'stay calm' or 'just continue to relax'.

Sandi: I would continue to remain calm and take more deep breaths and just keep quiet for a time and not answer. And if he sees that I'm not proceeding with his provocation, then I've won.

Therapist: Can you say 'I don't need to prove myself to him'?

Sandi: I don't need to prove myself that I can overcome my anger. I know myself, I know, I know. And I tell myself that there is no point in getting mad, nobody ever makes me angry except myself. And it's doing me no good, I'm getting nothing from it. Yes, I'm going to tell myself that he is not going to get to me. And he looks foolish to the crowd, he's the one who is now making a fool of himself. It wouldn't take me anywhere to be angry with him, it's just a waste of my time.

Therapist: Can you think of a situation at work where these

statements can be used to help confront a provocation?

Sandi: Yes, it's with a nurse that was late, she's always late and doesn't excuse herself. I always think that she thinks I didn't see her. [silence]. Those provocations make me angry, because time is important; it should be important. I tell her that it's my duty and her duty to report on duty on time. So such situations sometimes provoke anger and I don't know if whether I should just keep quiet. I say I must keep calm and relaxed.

Therapist: Do you feel it is important to tell her that she is late and should do something about it?

Sandi: Ja [Yes], I do. I feel I think I must tell her. I must tell her not aggressively; not aggressively but in a friendly manner just try to explain. And let her see my point-of-view.

Therapist: Say this time it works and she doesn't come late anymore. How would you feel?

Sandi: I would feel that, well I have managed to solve the problem without really getting angry. Because anger would have made it worse, perhaps. Perhaps she would not have listened to me if I became angry. She would march-off. But since I've been calm and relaxed, it shocked her because I'm not usually like that [laughs]. I makes me feel good that I've won.

Therapist: Now let's say she sits there and your response hasn't worked. Sometimes it may work and the anger may stop there and things won't go further.

Sandi: But sometimes it can continue. I'll ignore her and just leave her, to cool-off.

Therapist: But you aren't cool. You are hot.

Sandi: You go off and become hot somewhere else. Don't say a thing.

When you feel you are hot, just take a few deep breaths and go out of the situation where she is and try and think of a way.

Therapist: Is it always a feasible idea to leave the situation?

Sandi: No, you are not going to leave it, you are just thinking of another way. Call a nurse or a colleague to evaluate the situation.

Therapist: You have coped with preparing for the provocation, You have confronted the provocation, but now you also have to cope with it. It still doesn't matter what you say , the agitation continues and you are still feeling hot and your muscles are tight.

Sandi: You tremble.

Therapist: You tremble. What should you now do to help matters? Yes, yes...what would you be doing now?

Sandi: What, what, which way now. I said I would ask someone for help.

Therapist: Would you be doing that or is that what you would like to do?

Sandi: I would be doing that!

Therapist: Would you not be getting really angry with her by now and starting to shout at her?

Sandi: Well, see, I wouldn't like to be angry with her because it isn't going to solve anything. Now, but you know that is what I usually do [laughs]. I would start shouting by saying: don't be so stupid! You don't get anything into your head, I've been telling you the reasons, but you don't seem to care and so on and so on [laughs].

Therapist: Maybe that would work?

Sandi: It won't work. With some of them it might work, with others

it won't. With this person it might work, it might not work. Perhaps if she sees that I am serious about it, really, it's not for my own good, it's not for myself, its for the good of the patients we're looking after. She's not working for me. Perhaps she might see the cause. But being angry doesn't help in all the cases.

Therapist: A little bit of a shout, but not getting angry.

Sandi: Can you?

Therapist: Yes, you can be firm can't you?

Sandi: Ah yes, that's what I was trying to say. But I don't want to be angry, I want to be firm.

Therapist: You can still be firm, raise your voice a bit and be more assertive and firm. This does not mean you have to be angry. Cool inside and calm, still taking some deep breaths.

Sandi: Maybe she has got a problem which she has not told me about. Maybe she can't drive to work, or at home the maid has not arrived, or maybe she does not stay with her family and has to leave the kids alone. But if she doesn't tell me, I won't know. Or perhaps find out if there is a reason why.

Therapist: It would be a good idea instead of getting angry to find out if there is a reason for her being late. Perhaps she doesn't have the courage to tell you.

Sandi: That is right, don't get angry...I like to work with the other nurses, really. They miss me during the day. I miss them too. I don't get angry for long, you know it's not my way of doing it. I get angry and then get off it. The nurses never look at me as if I have got angry with them, they forget easily too. They are apologetic sort-of. What-else would you like to know?

It can be seen from the dialogue above that the therapeutic intervention was conducted, primarily, through the use of recall and modification of cognition surrounding previous incidents of anger and aimed at altering the patient's associated "self-talk" by using a number of alternate positive self-statements which facilitated a sense of self-efficacy and empowerment. It also aimed at teaching the patient to remain calm and relaxed during a provocation which facilitates the maintenance of rational and "clear-headed" behaviour and provides an enhanced sense of coping. The dialogue also illustrates a progression through some of the different stages of provocation, as described by Novaco (1975), where the patient is taught how to divide an angry provocation and reaction into a series of stages and at each stage to provide herself with appropriate self-statements. Being able to sub-divide a stressful event, such as a provocation, also increases the person's perceived sense of coping, by portioning the event into more manageable segments.

In addition, the intervention aimed at increasing the patients coping skills by increasing her repertoire of cognitive responses to a provocation. Although Novaco provides some general anger-events to be used as working-examples in therapy, it was essential that cognitive and behavioural modification were based on events that were close to the patient's own experience. For this reason, a general anger-provocation like 'being laughed at' was used at the start of the therapy-session to prevent feelings of intimidation, but was then followed by recall of a situation which was more personal to the patient. Novaco's suggested examples of anger-provocations are suitable as points of departure, but more significant therapeutic progress can be made if the situations are

taken from the person's own experience. All other therapy-sessions followed the same general structure as the session transcribed above, with a progression through all the different anger-provocation stages and the provision of appropriate positive self-statements for each stage. Various examples of angry experiences were elicited from the patient and used to implement the different principles of cognitive and behavioural modification, as described above.

QUANTITATIVE ANALYSIS OF THE TREATMENT INTERVENTION

Using a combination of quantitative and qualitative analyses, and visual inspection of blood pressure and anxiety levels from graphs an assessment can be made of the trends in blood pressure activity and treatment effectiveness over the period of the study. Sandi's **pre-treatment** blood pressure level varied from an initial **154/100mmHg** (Week 1) to **135/88mmHg** (Week 6) with no accompanying alteration in medication and no treatment intervention. In Week 5 and 6 of the pre-treatment phase it became apparent that her blood pressure had stabilised and in Week 7, the treatment intervention was implemented. Over the duration of the treatment, Sandi's blood pressure dropped to **126/80mmHg** by the end of the study. Also a marked decrease in blood pressure was noted during the pre-treatment phase which will be discussed under the qualitative analysis of the results [refer to Figure I below].

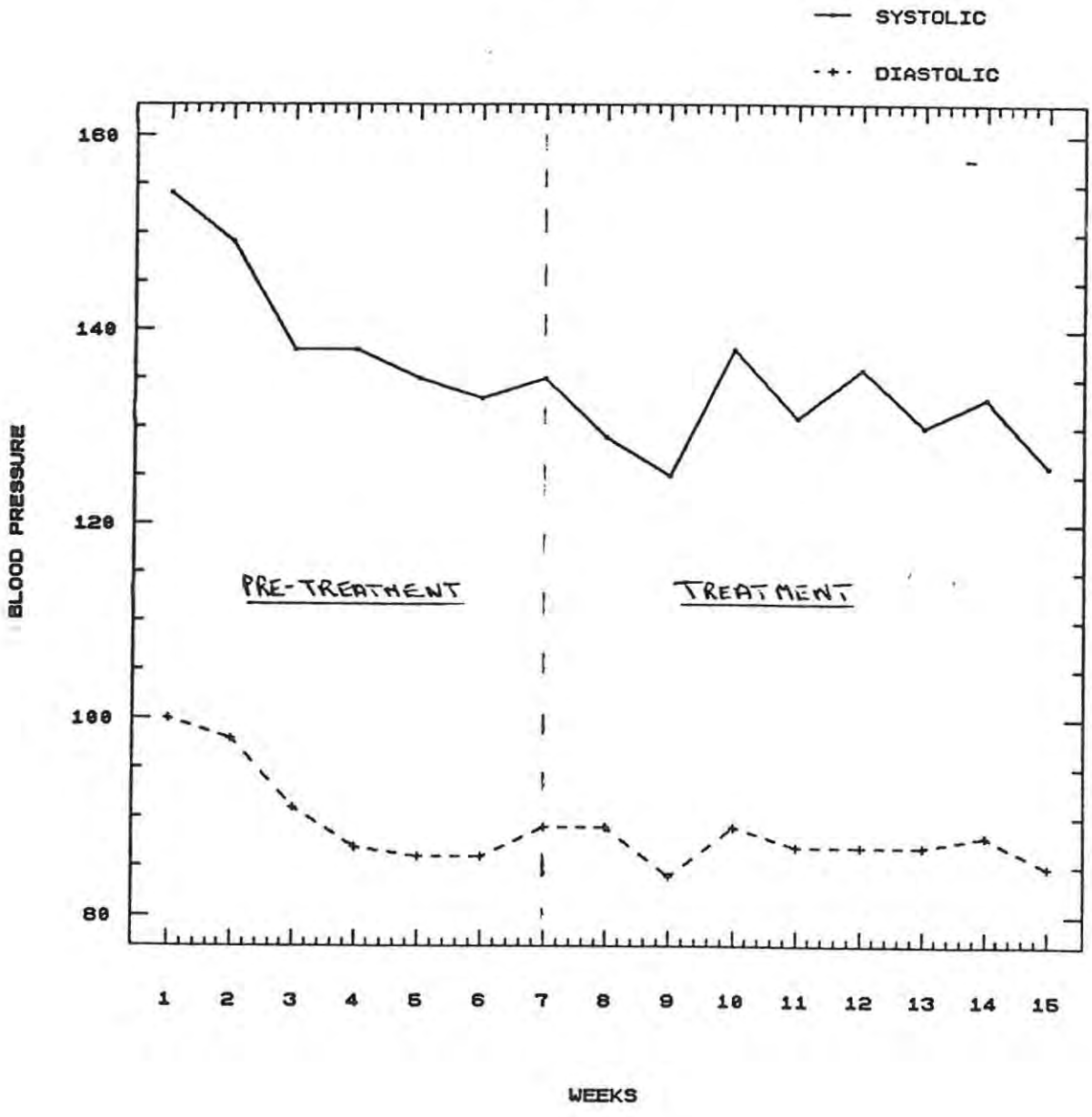
Compared with pre-treatment levels of blood pressure, where the average systolic pressure was **139,91mmHg** and diastolic pressure measured **91,02mmHg;** systolic pressure during treatment was on average **130,49mmHg** and average diastolic pressure measured

86,79mmHg. This represented an average reduction in systolic blood pressure of **9,42mmHg** and an average drop in diastolic pressure of **4,23mmHg.** An Independent t-test showed a highly significant reduction in systolic and diastolic blood pressure levels between the pre-treatment and treatment phases of the study:

Systolic blood pressure: $t = 5,510$; $p = 0,001$; $df = 101$

Diastolic blood pressure: $t = 3,865$; $p = 0,001$; $df = 101$

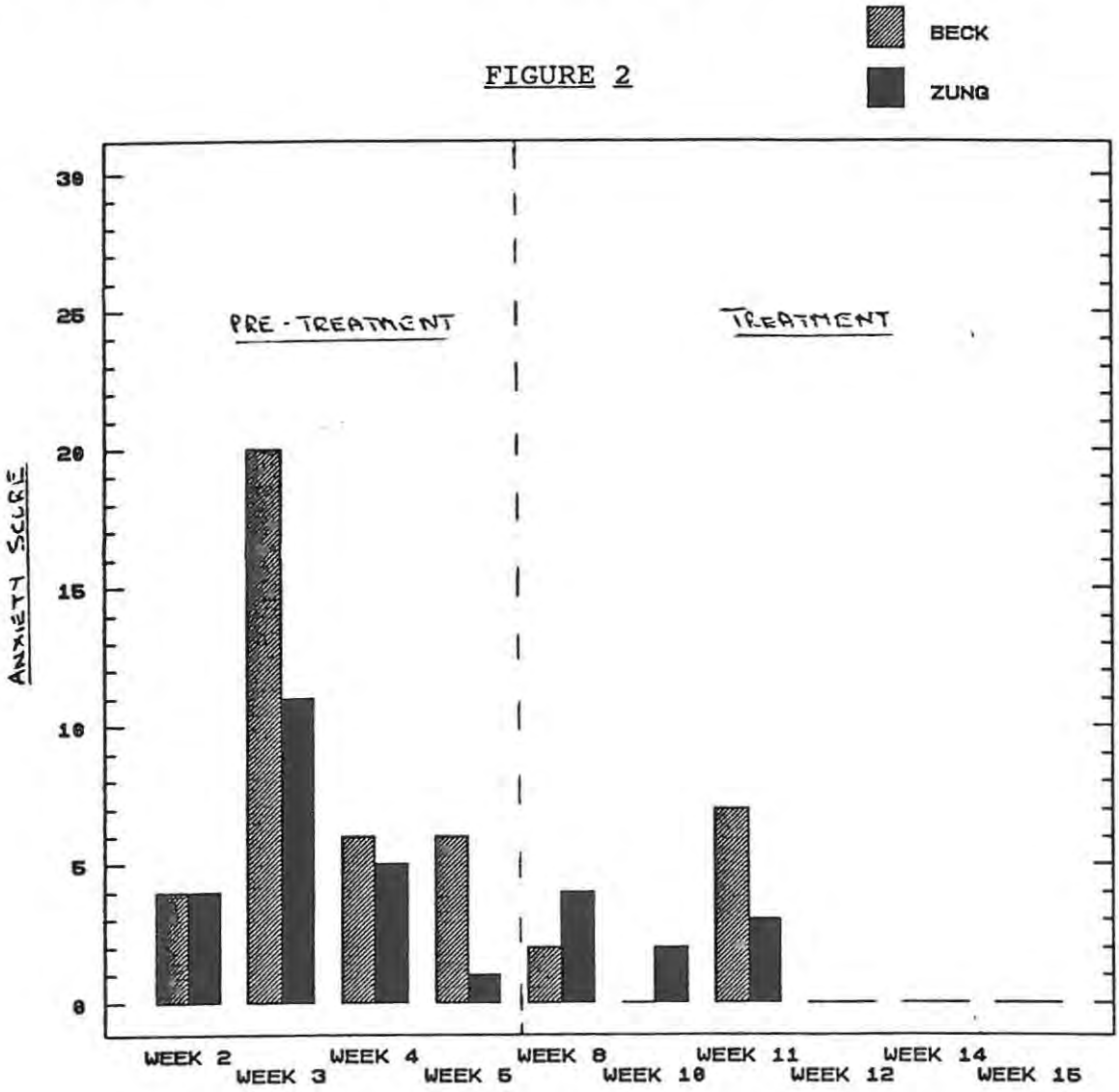
FIGURE 1



In addition to there being a highly significant reduction in blood pressure, a reduction from an initial average pressure of **154/100mmHg** during Week 1 at the start of the study to an average blood pressure of **126/80mmHg** obtained by the end of the treatment phase also represents a meaningful and substantial decline which placed the patient within the **normotensive** blood pressure range.

Similarly, the patient's general anxiety - as measured by the Beck and Zung Anxiety inventories - declined during the treatment phase which is represented by an overall decline in the height of the bars in the graph. Here a substantial and significant reduction in anxiety was observed [refer to Figure 2 below].

FIGURE 2



During the pre-treatment phase the patient scored a maximum of **20 points** on the Beck inventory and a maximum of **11 points** on the Zung inventory, while she scored a maximum of **7 points** on the Beck inventory and **4 points** on the Zung inventory on single occasions during the course of treatment. In addition, over the last three weeks of treatment, the patient obtained zero scores on both inventories.

The average anxiety, as measured by the **Beck Inventory,** during the pre-treatment phase equalled **9 points,** while during the treatment phase equalled **1,5 points.** Thus the average reduction in anxiety measured **7,5 points.** On the **Zung inventory** the average pre-treatemnt anxiety level measured **5,25** points while the treatment anxiety level was **1,5 points.** This represented an average reduction in anxiety of **3,75 points** over the study.

In addition, a reduction in anger was observed on Novaco's Anger Inventory compared with scores during the pre-treatment phase. The patient initially scored a total of **398 points** on the scale during pre-treatment which declined to **360 points** by the end of treatment. This represented a **10 per cent** reduction in subjectively perceived anger over the duration of the study.

A moderate reduction of body mass was also noted over the course of the study. In Week 2 of the study the patient weighed **76kg** which had declined to **70,5kg** in Week 15 of the study. This represented a loss of **5,5kg** in body weight over the duration of the study, or a weight loss of **7,24** per cent.

QUALITATIVE ANALYSIS OF THE TREATMENT INTERVENTION

On inspection of Figure 1 and Figure 2 it is apparent that blood pressure and anxiety varied considerably over the course of the study. Although there was a reduction in both blood pressure and anxiety, it can be seen from the two graphs that this was not uniform. It is of particular importance to determine the possible causes of these fluctuations from week to week. By correlating the quantitative blood pressure and anxiety trends with qualitative data, additional light could be shed on the dynamics involvedin the aetiology of Sandi's hypertension: what behavioural factors and stressful events raise anxiety levels and blood pressure.

From Week 7 to Week 9 of the treatment phase an observable drop in both systolic and diastolic blood pressure levels occurred. Similarly, there was a reduction in anxiety compared with the previous weeks. There was no change in medication nor had any extraordinary event or change in life-style occurred during this time. However, these two weeks followed the beginning of relaxation training and anger-therapy, which suggests that reductions in blood pressure and anxiety could be a result of the initiation of therapy and a result of the beneficial effects of the treatment intervention.

In Week 10 of the study it is apparent that both systolic and diastolic pressure rose significantly. During this week, the patient experienced a stressful time on the wards with one night being extraordinarily strenuous with extensive activity due to a multiple motor-car accident having occurred with numerous casualties and a death in the ward. In addition, Sandi had to go

home to Alexandria suddenly at the end of that week to look after her ill sister's children.

From Weeks 10 to 15 there is a noticeable cyclic variation in systolic pressure from one week to the next. After the rise in blood pressure in Week 10 there is a sudden drop in systolic pressure in Week 11, followed by a rise in systolic pressure in Week 12, followed again by a drop in systolic pressure in Week 13, followed again by a rise in systolic pressure in Week 14 followed by a final drop in systolic pressure in the final week of the study. This pattern of blood pressure variation corresponded to the weeks when Sandi was on night-duty and when she was off, with an increase in blood pressure occurring when she was on night-shifts. What is also evident is an overall reduction in systolic blood pressure after each rise and fall in blood pressure. This could possibly be ascribed to the positive effects of treatment which were exhibiting their effects in reducing blood pressure even in the presence of continued stress in the workplace.

This provides irrefutable quantitative and visible support for the hypothesis that occupational stress was a contributing factor to the aetiology of Sandi's hypertension and to the claims in the literature that occupational stress causes rises in blood pressure despite there still being a controversy about this relationship.

After a drop in diastolic pressure in Week 11 there was little variation during Weeks 11 to 14, with a final drop in diastolic pressure in the last week of the study. Since no other life-style changes were noted other than the implementation of therapy during these weeks, it can be postulated that reductions in diastolic

pressure were associated with the therapeutic intervention. It also appears that the diastolic pressure was more stable over the final weeks of the treatment than the systolic pressure, which may simply be due to the physiological nature of blood pressure where diastolic pressure is more resistant to reductions as it nears its lower-limits; otherwise the literature provides no clue as to what causes this diastolic stability when the sustolic pressure continues to change.

It is also evident from Figure 2 that anxiety levels fluctuated over the course of the study. From data collected during interviews it was obvious that these changes were associated with factors related to the work-situation and other specific events. Higher anxiety was recorded in Weeks 8, 10, and 11 of the treatment phase. Sandi attributed a rise in anxiety during Week 8 to apprehension about starting night-shifts the following week and in Week 10 to the stressful and strenuous workload that week. However, it is not certain what raised her anxiety level during Week 11. This could have been a cause of her usual occupational stress, or carry-over effects of stress from the previous week or as a result of anticipation for the upcoming week of night-shifts. No qualitative data could confirm this nor provide any additional explanation.

During the pre-treatment phase of the study it was evident that Sandi's feelings of anxiety were associated with being ill in Week 2, where the items on the anxiety inventories were reflecting symptoms of illness and physical discomfort due to influenza rather than anxiety: such as 'being unable to relax', 'pains in the neck and back' and feeling 'weak and tired'. In Week 3 Sandi scored the highest level of anxiety which she ascribed to her anticipation

about having her teeth extracted and which accompanied her dental extractions that week. Again in Week 4 and 5 there was no explanation for her anxiety other than from existing occupational stress and perhaps due to carry-over effects from the teeth-extraction the previous week and from associated physical discomfort.

Despite the reductions in anxiety and blood pressure during the treatment phase, a noticeable reduction in blood pressure occurred during the pre-treatment phase when no therapeutic intervention was implemented, no changes in life-style occurred and the medication regimen had remained the same since three weeks before the start of the study. Blood pressure dropped from an initial level of 154/100mmHg in Week 1 to 135/88mmHg by the end of Week 6. Initially there appeared to be no explanation for this drop in pressure, however it seemed unlikely that this significant reduction was artefactual to the study. It was possible that the patient was reacting to the presence of the researcher, and/or to self-monitoring of her blood pressure during this phase, and/or was altering her behaviour in some way as a result of becoming aware of her hypertension and occupational stress during the course of questioning and interviewing. All three possibilities could have been occurring to reduce her blood pressure.

Such a possibility is supported by theoretical principles, where merely becoming aware of one's behaviour and monitoring that behaviour has been observed to have an effect on altering that behaviour (Mahoney and Thoresen, 1974, p36). It is possible that a reduction in blood pressure during the pre-treatment phase was a result of increased awareness and associated subtle changes in behaviour that reduced anxiety, anger and arousal. Johnston (1986)

also comments on such non-specific effects which can be elicited during a treatment intervention. However, Johnston argues that such "non-specific" effects are not the only results of a treatment, but are combined with the more important specific effects of the treatment and that these non-specific effects are not the same nor as effective in the long-term as the intended effects of the treatment. That this reactivity to self-monitoring could be a cause of the overall reduction in blood pressure can not be ruled out as a possibility, however, Mahoney and Thoresen (1974) report that self-monitoring and its effects are usually short-lived and while they may have an initial effect on the behaviours involved, the effect will eventually fade and the behaviour will return to its pre-monitoring level. Although this possible reactivity to self-monitoring is not a direct result of the treatment intervention, it should not be viewed as any less important than the effects of the treatment per Se, but as a beneficial effect which can aid therapeutic progress and which would accompany any therapeutic intervention to some extent in the initial exploratory stages if self-monitoring is practiced. In fact, Hersen and Bellack (1985, p397) encourage the use of self-monitoring in reducing blood pressure and as a means to enhancing compliance with the treatment intervention.

Important in a qualitative analysis of an intervention is the subjective appraisal of the intervention by the patient. At the end of the study the patient was asked to evaluate the treatment in her own personal way and write a short account, which provided an additional valuable source of information to the effectiveness of the intervention package. Sandi found the treatment most beneficial which also increases the possibility that she will continue to

implement the learned strategies in the future. She did, however, find the relaxation difficult to implement in the workplace and at home during her night-shifts, when her normal day-routine was disrupted. However, after a little persistence on her part and mutual discussion, she decided on the best times during the day and night to practice relaxation and the problem was solved. The following passage was her response and evaluation of the treatment:

"When I offered myself to be involved in a research project about the effects of stress as a contributing factor to hypertension I least expected anything like what I have experienced throughout the course. At the time I was under great stress. Working conditions left much to be desired - work overload, long tedious working hours and most inconvenient shift hours. Stress situations in my case, as far as I could ascertain, were mostly work-related than otherwise and, unknowingly in an attempt to combat these situations I was using wrong methods altogether, thus aggravating matters. This resulted in a build-up of anger and frustration. At the same time my General Practitioner was stabilising me on new treatment. Such was the situation when I set off on this project. I started off with great enthusiasm as I was promised this was going to be an interesting exercise especially towards the end. The most interesting part was finding out about anger (which in my case was a way of letting-out) and how to cope with it. As the sessions continued from weekly to biweekly, I gradually became a changed person. Then there were relaxation exercises conducted under the soothing and relaxing accompaniment of musical cassettes. At first it was difficult to apply these exercises on my own especially to apply them in the work situation, but then I soon overcame this problem with the patient coaxing of my coach. My blood pressure readings,

which were taken bi-daily, dropped to normal and became stable, except on occasions when I was ill. We even found time to visit my G.P. and conducted some blood tests under his supervision. Even on my night-duty my blood pressure readings have remained constant. Rory went out of his way to give me good advice on occasions when I wanted to act rashly thereby wasting a lot of unnecessary energy. Actually I can say that I have enjoyed every minute of these sessions and will continue to use the knowledge gained for myself and for the benefit of others who may find themselves in the same situation".

DISCUSSION

It is apparent from this study that psychological and behavioural patterns are highly complex and that many subtle changes could be occurring to lower blood pressure. Similarly, it is evident that the aetiology of essential hypertension is complex and no single factor could be detected which was solely responsible for elevations in blood pressure. However, what does become clear is that a multitude of factors can contribute to sustained elevations in blood pressure and that treatment must focus on altering as many of the causes as possible.

Sandi exhibited excessive occupational stress and frustration in the workplace which was associated with continued emotional and behavioural arousal. In addition, this was exacerbated by an inability to cope with the occupational stress and inappropriate methods of reacting to stressful occurrences in the workplace. Her continued angry reactions limited her repertoire of responses to stress and did little to increase her coping ability across

different stressful situations. Although it is useful to become angry in appropriate situations, such a predominant response to all stressful situations is inappropriate and often leads to a personal frustration rather than a sense of coping. Such a state will undoubtedly lead to sustained emotional and physiological arousal and increased sympathetic nervous system excitation, which were possibly the prime contributors to her elevated levels of blood pressure.

A further possibility existed that a familial predisposition to hypertension could have been operating in the patient and producing a susceptibility to high blood pressure. It is unlikely that genetic factors could determine the actual level of blood pressure or else the intervention would not have had a reducing effect, but, perhaps a genetic predisposition to high blood pressure could have determined a high minimum and a high maximum blood pressure level. Or inappropriate learned styles of coping acquired from the parents could have placed the patient at risk for hypertension. What the possible relationships between these variables are remain unknown and need to be assessed and determined in further research.

Although such causal links between experienced stress, inappropriate emotional expression, inadequate coping, familial hypertension and high blood pressure remained largely intuitive and based on inferences from the hypertension theory in dealing with this case, enough evidence of such relationships exists in the literature to support the logic of this claim and to have necessiitated an intervention aimed at reducing arousal through relaxation training and at modifying existing inappropriate stress-coping strategies and increasing coping skills. This was achieved by

implementing Benson's Relaxation Response and Novaco's Stress Inoculation Model which, as a coping-skills therapy, is concerned with developing the patient's competence to adapt to stressful events in such a way that stress is reduced and personal goals are achieved. A variety of cognitive and behavioural skills are imparted to the patient to facilitate anger reduction and regulation and the management of provocation events.

The present case-study highlights the complexity of essential hypertension and the need to implement a treatment strategy which is tailored to the specific emergent aetiological factors of the case. Not only could occupational stress, lack of coping and anger expression have contributed to this case of hypertension, but increased weight may also have been an aetiological factor. Reductions in blood pressure could, thus, have been the result of a combination of both cognitive and behavioural therapy and the effects of weight loss. However, it is not possible in such a study to assess the independent effects of the treatment strategies such as large statistical research is capable of doing, but the case-study does contextualise the problem of hypertension within the broad experience of human living and thus imparts a meaningful appreciation of the disorder in the attempts to treat it with a mutimodal cognitive-behavioural intervention.

The study also provides significant support for the use of such a cognitive-behavioural intervention as a type of non-pharmacological adjunctive treatment strategy to medication. On medication alone, the patient was unable to reduce her blood pressure to normal levels, eventhough she was taking strong medication and her compliance to the medication regimen was completely satisfactory.

With the addition of a cognitive-behavioural treatment programme blood pressure was reduced to normal levels after only fifteen weeks of the study, even with ongoing stress for the patient in the workplace. In addition, there was a concurrent reduction in anxiety and in anger, as measured by the anxiety and anger inventories respectively, with a presumable increase in general psychological well-being.

Hersen and Bellack (1985) argue that the major limitation of non-pharmacological treatments of hypertension is the modest blood pressure reductions that are achieved. According to Kaplan (in Hersen & Bellack, 1985, p395) medication typically produces a 10% to 30% reduction in blood pressure, whereas non-pharmacological interventions typically yield less than 10% reduction. In this study a more significant reduction in blood pressure was observed with a reduction of **18%** in systolic pressure and **20%** reduction in diastolic pressure from the start of the study. Compared with reductions in blood pressure after nearly two years of medication, the reductions in pressure as a result of these non-pharmacological factors in this study were highly significant. This increased effectiveness may be due to the fact that a comprehensive intervention was designed and implemented to treat as many aetiological factors as possible, which differs from the majority of other studies where limited treatment has been provided which usually only consisted of relaxation training, weight reduction or exercise with the exclusion of more extensive cognitive/behavioural modification. It may also be due to the fact that therapy was conducted on an individual basis where extensive personal involvement was provided by the therapist/researcher and was elicited from the patient, whereas in the majority of other studies

group-interventions have been provided. This could have increased the patient's motivation to comply with the therapist which would have enhanced the effects of the treatment.

Unfortunately, due to time restraints, it was not possible to assess the long-term benefits of the treatment intervention and associated reductions in blood pressure levels. This was an inadequacy of the study which needs to be accounted for in future studies of this type.

Nonetheless, it seems that much potential exists for the use of such a comprehensive cognitive-behavioural approach to the treatment of essential hypertension. While such an intervention is certainly feasible in a clinician's private practice, there may be serious practical and financial limitations to implementing such a treatment on a large scale to thousands of people. It was noted in the introduction to this study that an extremely high incidence of hypertension occurs in South Africa and especially within the Black urban community. With a relative insufficiency of health-care workers in South Africa, the feasibility of adopting such a treatment programme must be questioned. Although significant reductions in blood pressure were obtained within fifteen weeks of therapeutic involvement in Sandi's case, this did take extensive effort on the part of the therapist/researcher. It is unlikely that such intensive attention could be provided to thousands of patients, many of whom live in far-out rural places. However, many of the techniques and principles in the intervention could be adapted or modified to suite therapy with large groups of individuals and would depend only on the ingenuity and creativity of the health-care worker. This could provide further avenues for

community-based research into applicability and feasibility of implementing such a non-pharmacological treatment for essential hypertension.

Another possible drawback to using a cognitively based intervention is the heavy reliance on language as a form of communication and vehicle for therapeutic modification. Although this research subject was highly educated and articulate she represents the exception rather than the rule. In other community-based research, conducted in the same region, it was evident that the provision of therapy to the ordinary Black layman by therapists that are not fluent in the local vernacular was impossible without the use of a translator. Also, communication had to be kept simple to ensure understanding by highly uneducated individuals (Losinsky 1990). This would possibly limit the usage of in-depth therapeutic dialogue and reduce the effects of a cognitive-based treatment. Again, this would need to be evaluated in further research and an implementation of a cognitive-behavioural treatment would only rely on the limits of creativity from the health-care worker.

What is needed is more research into the use of non-pharmacological treatments for essential hypertension in individual-therapy by presenting quantitative and qualitative case-studies of this sort for academic and clinical evaluation. In addition, researchers then need to focus on assessing the feasibility of implementing and modifying such treatments for large scale community application by presenting large sample size research findings for consideration. Either way, it is suggested that meaningful research-findings must begin on the individual level before being applied for use in the community.

Besides the intention of assessing a specific cognitive-behavioural intervention in reducing blood pressure, this study also highlights the usefulness of using a case-study methodology in this kind of research. The case-study is not a mathematico-deductive model of positivist metatheory, but provides a framework for understanding rather than one of explanation. This does not mean that explanation and understanding need to be regarded as exclusive categories as the case-study can also be envisaged to provide explanation that is based on personal understanding for those who work with it in an applied practical context. In this respect, the case-study is a research approach which can provide a holistic and meaningful understanding of a problem to a greater extent than most other research methodologies. Especially with research such as the kind that was conducted in this study, the comprehensive nature of a flexible dual quantitative and qualitative data collection across the disciplines of medicine, sociology and psychology provides a final understanding of the person as a "whole" within the broader context of his environment. The case-study can also illuminate the salient and subtle realities of human behaviour which large multivariate research strategies often oversee. This was made evident in the present study where the subtle changes and events on a day to day basis could be seen to affect blood pressure and contribute to essential hypertension, while the transcription of therapy sessions gives a fine insight into the processes of behaviour and thinking and into the processes of therapeutic intervention.

Associated with a greater insight into the subtleties of human behaviour as a result of the fine attention to detail of the case-study method, the "humanness" of the research problem and of the

participant/s can be secured and enhanced. This facilitates a harmony and symbiosis between research and therapy and between theory and therapeutic practice. The case-study method, then, can bring theory and practice to life and show how the therapeutic technique follows from the logic of the psychological, biological, behavioural and social processes described in it.

However, the case-study method is not without its difficulties. In particular it poses a great effort on the part of the researcher to take into account and consider a problem in extensive detail and requires a sound knowledge of a variety of data collection and analysis techniques, not to mention extensive attention to the literature in the field and considerable effort in the maintenance of a close relationship with the research subject/s over a prolonged period of time.

Despite these difficulties in carrying out such research, the final result of a well planned and operationalised case-study can be most beneficial in the evaluation and enhancement of theory and therapeutic technique and is personally highly satisfying. In the words of Edwards (1990c): "Cognitive therapy has a tradition of common sense pragmatism. The case study method is very much in accord with this approach, and if its place in the research process can be honoured, the future development of cognitive therapy can only be enhanced" (p23).

REFERENCES

Australian National Blood Pressure study. (1979). Management committee of the Australian Hypertension trial. Initial results of the Australian trial in mild hypertension. Clinical Science, 57(5), 449-452.

Barr, B.P., & Benson, H. (1984). The relaxation response and cardiovascular disorders. Behavioral medicine update, 6(4), 28-30.

Basler, H.D., Brinkmeier, U., Buser, K., Haehn, K.D., & Molders-Kober, R. (1985). Psychological group treatment of obese essential hypertensives by lay therapists in rural general practice settings. Journal of psychosomatic research, 29(4), 383-391.

Beard, T.C., Gray, W.R., Cooke, H.M., & Barge, R. (1982). Randomised controlled trial of a no-added sodium diet for mild hypertension. The Lancet, August, 455-459.

Benson, H., Marzetta, B.R., & Rosner, B.A. (1973). Decreased systolic blood pressure in hypertensive subjects who practiced meditation. Journal of clinical investigation, 52, 8a.

Benson, H. (1974a). Transcendental meditation - science or cult? Journal of the American medical association, 227, 807.

Benson, H., Beary, J.F., & Carol, M.P. (1974b). The relaxation response. Psychiatry, 37, 37-46.

Benson, H., Rosner, B.A., Marzetta, B.R., & Lemchuk, H.M. (1974c). Decreased blood pressure in pharnacologically treated hypertensive patients who regularly elicited the relaxation response. Lancet i, 289-291.

Benson, H., & Klipper, M.Z. (1975). The relaxation response. New York: Avon Books.

Black, H.R. (1979). Nonpharmacological therapy for hypertension. The American journal of medicine, 66, 837-842.

Blackwell, B., & Bloomfield, S. (1976). Transcendental meditation in hypertension. Lancet i, 223.

Bromley, D.B. (1986). The case-study method in psychology and related disciplines. Chichester: Wiley.

Brownstein,A.H., & Dembert, M.L. (1989). Treatment of essential hypertension with yoga therapy in a USAF aviator: a case report. Aviation, space and environmental medicine, July, 684-687.

Coleman, V. (1985). High Blood Pressure. London: Severn House.

Christensen, L.B. (1980). Experimental Methodology. Boston: Allyn & Bacon.

Cochrane, R. (1971). High blood pressure as a psychosomatic disorder: A selective review. British Journal of social and clinical psychology, 10, 61-72.

Dean, D. (1961). Alienation: Its Meaning and Measurement. American Sociological Review, 26, 753-758.

Decker, J.L., & Keiser, H.R. (1987). Understanding and managing hypertension. New York: Avon Books.

Drazen, M., Nevid, J.S., Pace, N., & O'Brein, R.M. (1982). Worksite-based behavioral treatment of mild hypertension. Journal of occupational medicine, 24, 511-515.

Edwards, D.J.A. (1990a). Application of psychoeducational and bio-behavioural approaches to hypertension in a deprived Black community: A conceptual analysis and some preliminary case-studies. Grahamstown: Rhodes university.

Edwards, D.J.A. (1990b). Case Study research method: a theoretical introduction and practical manual. Grahamstown: Rhodes University.

Edwards, D.J.A. (1990c). Case study research method: The hidden cornerstone of theory and practice in cognitive therapy. Grahamstown: Rhodes University.

Fitzgerald, W. (1981). Labile hypertension and jogging: new diagnostic tool or spurious discovery? British medical journal, 282, 542-544.

Foreyt, J.P., & Rathjen, D.P. (1978). Cognitive Behavior Therapy: research and application. New York: Plenum.

Gampel, B., Slome, C., Scotch, N., & Abramson, J.H. (1961). Urbanisation and hypertension among Zulu adults. Journal of chronic disorders, 15, 67-70.

Goldstein, I.B., & Shapiro, D. (1982). Biobehavioral perspectives on hypertension. Journal of Consulting and Clinical Psychology, 50(6), 841-858.

Greenberg, J.S. (1983). Comprehensive stress management. Dubuque: Brown Company Publishers.

Grobbee, D.E., & Hofman, A. (1986). Does sodium restriction lower blood pressure? British Medical Journal, 293, 27-29.

Hersen, M., & Barlow, D.H. (1976). Single case experimental designs: Strategies for studying behavioral change. New York: Pergamon.

Hersen, M., & Bellack, A.S. (1985). Handbook of Clinical Behavior Therapy with adults. New York: Plenum Press.

Holroyd, K.A., Appel, M.A., & Andrasik, F. (1983). A cognitive-behavioral approach to psychophysiological disorders. In: D. Meichenbaum, & M.E. Jaremko (Eds.). Stress reduction and prevention. (pp.219-259). New York: Plenum.

Horton, E.S. (1981). The role of exercise in the treatment of hypertension in obesity. International Journal of Obesity, 5(1), 165-171.

Hypertension Detection and Follow-up Program Cooperative Group. (1979). Five year findings of the hypertension detection and follow-up program: I. Reduction in mortality of persons with high blood pressure, including mild hypertension. Journal of the American Medical Association, 242, 2562- 2571.

Irvine, M.J., Johnston, D.W., Jenner, D.A., & Marie, G.V. (1986). Relaxation and stress management in the treatment of essential hypertension. Journal of psychosomatic research, 30(4), 437-450.

Isaacson, C. (1989). How to look at target organ damage - heart, brain, kidney, eye. In: Seedat, M.A (Ed). The challenge of hypertension in the 1990's: "How to" Mini symposium, p34. Durban: 1989.

Johnston, D.W. (1986). How does relaxation training reduce blood pressure in primary hypertension? In: T.H. Schmit, T.M. Dembroski, & G. Blumchen (Eds.). Biological and psychological factors in cardiovascular disease. (pp.550-566) Berlin: Springer-Verlag.

Johnston, D.W. (1987). The behavioral control of high blood pressure. Current Psychological Research & Reviews, 6, 99-114.

Kendall, P.C., & Hollon, S.D. (1979). Cognitive-behavioral interventions: Theory, research and procedures. London: Academic press.

Kratochwill, T.R., Mott, S.E., & Dodson, C.L. (1984). Case study and single-case research in clinical and applied psychology. In

A.S. Bellack & M. Hersen (Eds.), Research methods in clinical psychology. New York: Pergamon.

Losinsky, R. (1990). The establishment of a hypertension education programme for Black women in an urban setting - a preliminary investigation. Unpublished report. Grahamstown: Rhodes University.

Luborsky, L., Crits-Christoph, M.S., Brady, J.P., Kron, R.E., Weiss, T., Cohen, M., & Levy, L. (1982). Behavioral versus pharmacological treatments for essential hypertension - a needed comparison. Psychosomatic medicine, 44(2), 203-213.

Mahoney, M.J., & Thoresen, C.E. (1974). Self-control: power to the person. Monterey: Brooks/Cole publishing compaany.

Masters, J.C., Burish, T.G., Hollon, S.D., & Rimm, D.C. (1990). Behavior therapy: Techniques and empirical findings, (3rd ed.). New York: Harcourt.

Matarazzo, J.D., Weiss, S.M., Herd, J.A., Miller, N.E., & Weiss, S.M. (1984). Behavioral Health: A Handbook of Health Enhancement and Disease prevention. New York: John Wiley.

Matlou, S., Isles, C.G., Higgs, A., Milne, f.J., Murray, G.d., Schulz, E., & Starke, I.f. (1986). Potassium supplimentation in blacks with mild to moderate essential hypertension. Journal of hypertension, 4, 61-64.

Meichenbaum, D. (1972). Clinical implications of modifying what

clients say to themselves. Research Reports in psychology, 42. University of Waterloo, Waterloo, Ontario.

Meichenbaum, D. (1975). A self-instructional approach to stress-management: A proposal for stress inoculation training. In C. Spielberger & I. Sarason (Eds.), Stress and Anxiety (Vol. 2.). New York: Wiley.

Mitchell, P.I., Morgan, M.J., Boadle, D.J., Batt, J.E., Marstrand, J.L., McNeil, H.P., Middleton, C., Rayner, K., & Lickiss, J.N. (1980). Role of alcohol in the aetiology of hypertension. Medical Journal of Australia, 2, 198-200.

Morisky, D.E. (1986). Nonadherence to medical recommendations for hypertensive patients: Problems and potential solutions. The Journal of compliance in health care, 1(1), 5-20.

Nissinen, A., Bothig, S., Granroth, H., & Lopez, A.D. (1988). World Health Statistics quarterly, 41, 141-154.

Novaco, R.W. (1975). Anger Control. Lexington: Lexington Books.

Novaco, R.W. (1976). Treatment of chronic anger through cognitive and relaxation controls. Journal of consulting and clinical psychology, 44(4), 681.

Novaco, R.W. (1977). Stress inoculation: A cognitive therapy for anger and its application to a case of depression. Journal of consulting and clinical psychology, 45(4), 600-608.

O'Brein, E., & O'Malley, K. (1987). High Blood Pressure: What it means for you and how to control it. London: Macdonald Optima.

Patel, C.H. (1973). Yoga and bio-feedback in the management of hypertension. The Lancet, November 10, 1053-1055.

Patel, C.H. (1977). Biofeedback-aided relaxation and meditation in the management of hypertension. Biofeedback and Self-regulation, 2(1), 1-41.

Patel, C.H., Marmot, M.G., & Terry, D.J. (1981). Controlled trial of biofeedback-aided behavioural methods in reducing mild hypertension. British Medical Journal, 282, 2005-2008.

Patel, C.H., & Marmot, M.G. (1988). Efficacy versus effectiveness of relaxation therapy in hypertension. Stress medicine, 4(4), 283-289.

Polkinghorne, D.E. (1986). Conceptual validity in a nontheoretical human science. Journal of Phenomenological Psychology, 17, 129-150.

Props, A. (1990). Effects of relaxation, stress and personal variables on blood pressure in borderline hypertensives. Abstracts: European Health Psychology Society 4th annual conference, Oxford University, July 1990.

Rathus, S.A. (1973). A 30-Item schedule for assessing assertive behavior. Behavior Therapy, 4, 398-406.

Report of the task force on blood pressure control in children. Paediatrics (suppliment), 1977, 59, 797-820.

Rosendorff, C. (1989). How to manage mild hypertension. In: M.A. Seedat (Ed.). The challenge of hypertension in the 1990's: "How to" mini symposium, p52. Durban: 1989.

Rotter, J.B. (1966). Generalised expectancies for internal and external control of reinfircement. Psychological Monographs, 80, whole no 609.

Rouse, I.L., Beilin, L.J., Armstrong, B.K., & Vandongen, R. (1983). Blood pressure lowering effect of a vegetarian diet: controlled trial in normotensive subjects. The Lancet, January, 5-9.

Safran, C. (1987). New ways to lower your blood pressure. Singapore: Corgi.

Scholten, H., Maes, S., & van Elderen, T. (1990). Anger expression, anger control and blood pressure. Abstracts: Eurpoean Helath Psychology Society 4th annual conference, Oxford University, July 1990.

Scotch, N.A. (1960). A preliminary report on the relation of socio-cultural factors to hypertension among the Zulu. Annuals of the New York academy of science, 84, 1000.

Scotch, N.A. (1963). Sociocultural factors in the epidemiology of Zulu hypertension. American Journal of Public Health, 53(8), 1205-1213.

Seedat, Y.K., Hackland, D.B.T., & Mpontshane, J. (1981). South African Medical Journal, July, 7-10.

Seedat, Y.K., Seedat, M.A., & Hackland, D.B.T. (1982). Biosocial factors and hypertension in urban and rural Zulus. South African Medical Journal, June, 999-1002.

Seedat, M.A. (1989). The challenge of hypertension in the 1990's: "How To" Mini symposium. Durban, South Africa: June 1989.

Seedat, Y.K. (1990). Detection, evaluation and treatment of high blood pressure. In: Y.K. Seedat (Ed.). Hypertension: New perspectives. Craighall: Helm.

Seftel, H.C., Johnson, S., & Muller, E.A. (1980). Distribution and biosocial correlations of blood pressure levels in Johannesburg Blacks. South African Medical Journal, March, 313-320.

Sever, P.S., Gordon, D., Peart, W.S., & Beighton, P. (1980). Blood pressure and its correlates in urban and tribal Africa. The Lancet, July, 60-64.

Skrabal, F., Aubock, J., & Hortnagl, H. (1981). Low sodium/high potassium diet for prevention of hypertension: probable mechanisms of action. The Lancet, October, 1981.

Spiegler, M.D. (1983). Contemporary behavioral therapy. Palo Alto: Mayfield.

Spielberger, C.D. (1985). The Anger Expression scale. Unpublished

manuscript, Centre for Behavioral Medicine and Community Psychology, Human Resources Institute, University of South Florida, Tampa, Florida.

Surwit, R.S., & Shapiro, D. (1978). Comparison of biofeedback, neuromuscular biofeedback and meditation in the treatment of borderline essential hypertension. Journal of consulting and clinical psychology, 46(2), 252-263.

Swales, J.D. (1980). Dietary salt and hypertension. The Lancet, May, 1177-1179.

Taylor, J.A. (1953). A personality scale of manifest anxiety. Journal of Abnormal and social psychology, 48, 285-290.

Taylor, C.B., Agras, W.S., & Sevelius, G. (1977). Managing hypertension in the workplace. In: M.F. Cataldo (Ed.). Health and Industry. New York: Wiley.

Vinck, J. (1990). Coronary behaviour, job stress, anger and blood pressure. Abstracts: European Health Psychology Society 4th annual conference, Oxford University, July 1990.

Weiman, C.G. (1977). A study of the occupational stressor and the incidence of disease/risk. Journal of occupational medicine, 19, 119-122.

Wilcox, R.G., Bennet, T., Brown, A.M., & Macdonald, I.A. (1982). Is exercise good for high blood pressure? British medical journal, 285, 767-769.

APPENDIX 1

SCHEDULING STATUS: S4

PROPRIETARY NAME AND DOSAGE FORM:

TRITACE 1,25 mg Capsules
TRITACE 2,50 mg Capsules
TRITACE 5 mg Capsules
TRITACE 10 mg Capsules

COMPOSITION:
TRITACE 1,25 mg : Each capsule contains 1,25 mg Ramipril as active substance
TRITACE 2,50 mg : Each capsule contains 2,50 mg Ramipril as active substance
TRITACE 5 mg : Each capsule contains 5 mg Ramipril as active substance
TRITACE 10 mg : Each capsule contains 10 mg Ramipril as active substance

PHARMACOLOGICAL CLASSIFICATION:
Category A: 7.1.3 Hypotensives

PHARMACOLOGICAL ACTION:
Ramipril is a long acting inhibitor of the angiotensin converting enzyme (ACE).

Ramipril is a prodrug which is hydrolized in the liver after absorption from the gastrointestinal tract to form the active angiotensin converting enzyme inhibitor, ramiprilat.

Ramipril increases plasma renin activity and decreases plasma concentrations of angiotensin II and aldosterone. The beneficial haemodynamic effects are caused by ACE inhibition and the consequent reduction in angiotensin II results in dilatation of peripheral vessels and reduction in vascular resistance.

Angiotensin converting enzyme is identical to kininase II, one of the enzymes responsible for the degradation of bradykinin. There is evidence that ACE inhibition by ramipril appears to have some effects on the kallikrein-kinin-prostaglandin systems. It is assumed that these mechanisms contribute to the hypotensive activity of ramipril too and may be at least co-responsible for certain adverse reactions, e.g. dry cough.

Following oral administration ramipril is rapidly absorbed from the gastrointestinal tract, peak plasma concentrations of ramipril are reached within one hour. Ramipril is a prodrug, which is converted in the liver to its diacid metabolite, ramiprilat, by cleavage of an ester group. Peak plasma concentrations of ramiprilat are reached 2 – 4 hours after drug intake. Based on the urinary recovery of ramipril and its metabolites the extent of absorption is estimated to be 30 – 60%.

Food intake has no relevant influence on the extent of absorption.

Plasma concentrations of ramiprilat decline in a polyphasic manner. The effective half-life of ramiprilat after multiple once daily administration of ramipril is 13 – 17 hours for 5 – 10 mg ramipril and several times longer for lower doses, 1,25 – 2,5 mg ramipril. The prolonged half-life at low dosages is due to a high fraction of the metabolite being bound to the angiotensin converting enzyme at low plasma concentrations and thus a slow dissociation of this enzyme-inhibitor complex. However, with high dosages a shorter half-life due to a higher free fraction leading to easier dissociation is observed.

Steady-state plasma concentrations of ramiprilat after once daily dosing of the usual doses of ramipril are reached at about treatment day four.

Ramipril is almost completely metabolised and the metabolites are excreted mainly via the kidneys. Beside the bioactive metabolite ramiprilat, further inactive metabolites have been identified i.e. diketopiperazine ester, diketopiperazine acid and conjugates.

With impaired renal function the elimination of ramipril and ramiprilat from plasma is delayed and the urinary excretion reduced.

The protein binding of ramipril is about 73% and of ramiprilat about 56%.

INDICATION:
Mild to moderate hypertension.

CONTRA-INDICATION:
Hypersensitivity to ramipril.

History of angioneurotic oedema.

Ramipril is not recommended for use in children.

Safety in pregnancy and lactation has not been established.

WARNINGS:
The treatment of hypertension with this preparation must be carried out under regular medical supervision.

Treatment with TRITACE may impair the ability to drive or operate machinery, particularly at the start of treatment, when changing over from other preparations and during concomitant use of alcohol.

It is recommended to adjust the ramipril dose according to the degree of impairment of renal function.

In patients with impaired liver function, the metabolisation of the parent compound ramipril and therefore the formation of the bioactive metabolite ramiprilat is decelerated resulting in markedly elevated plasma ramipril levels due to a diminished activity of esterases in the liver. Use of ramipril in patients with impaired liver function is not recommended.

DOSAGE AND DIRECTIONS FOR USE:
Administration of TRITACE to hypertensive patients results in a reduction of both supine and erect blood pressure. The antihypertensive effect is evident within one to two hours after intake of the medicine, peak effect occurs 3 – 6 hours after intake, and has been shown to be maintained for at least 24 hours at recommended doses. The dose range is 2,5 – 10 mg TRITACE in a single daily dose.

The recommended initial dosage in patients not on diuretics is 2,5 mg TRITACE once a day. Dosage should be increased to 5 mg TRITACE and up to a maximum of 10 mg TRITACE once a day at intervals of 1 – 2 weeks based on patient response. A maximum dose of 10 mg should not be exceeded.

In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of TRITACE. The diuretic should, if possible, be discontinued for 2 – 3 days before beginning therapy with TRITACE to reduce the likelihood of hypotension. In case the diuretic therapy cannot be discontinued the initial dose of TRITACE should be 1,25 mg.

TRITACE should be taken with half a glass of liquid during or after the meal.

Dosage Adjustment in Renal Impairment
The usual dose of TRITACE is recommended for patients with a creatinine clearance > 30 ml/min (serum creatinine < 165 µmol/l). For patients with a creatinine clearance < 30 ml/min (serum creatinine > 165 µmol/l) the first dose is 1,25 mg TRITACE once daily and the maximum dose 5 mg TRITACE once daily.

In patients with severe renal impairment (creatinine clearance < 10 ml/min and serum creatinine of 400 – 650 µmol/l), the recommended initial dose is also 1,25 mg TRITACE once a day but the maintenance dosage should not exceed 2,5 mg TRITACE once a day.

SIDE-EFFECTS AND SPECIAL PRECAUTIONS:
The most frequently reported side-effects are nausea, dizziness and headache. A dry cough has been reported.

Cardiovascular System
Hypotension may occur after the initial dose of TRITACE as well as after increasing the dose of TRITACE. Symptomatic hypotension accompanied by dizziness, nausea and a feeling of weakness can be observed in salt volume depleted patients such as those treated with diuretics or patients on dialysis as well as in patients with severe congestive heart failure. Syncope has been observed.

In patients with concomitant congestive heart failure with or without renal insufficiency excessive hypotension has been observed and may be associated with oliguria or azotemia. In these patients therapy should be started under close medical supervision, and at a reduced starting dose.

If hypotension occurs, the patient should be placed in supine position and, if necessary, receive an intravenous infusion of physiological saline.

Renal Function
Treatment with TRITACE may impair renal function.

Patients with renal insufficiency may require reduced or less frequent doses of TRITACE and their renal function should be closely monitored. (See Dosage) There is a risk for impairment of renal function particularly in patients with renal insufficiency, congestive heart failure, bilateral renal artery stenosis and unilateral renal artery stenosis in the single kidney as well as after renal transplantation.

Some hypertensive patients with no apparent pre-existing renal disease may develop increases in blood urea and serum creatinine when TRITACE is given. In patients with renal insufficiency there is a risk of hyperkalaemia.

Liver Function
As TRITACE is a prodrug metabolised in the liver to its active moiety particular caution and close monitoring should be applied in patients with liver impairment. The metabolisation of the parent compound and therefore the formation of the bioactive metabolite ramiprilat may be decelerated resulting in markedly elevated plasma levels of the parent compound due to the diminished activity of esterases in the liver.

Surgery/Anaesthesia.
In patients undergoing surgery or during anaesthesia with agents producing hypotension, TRITACE may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism it can be corrected by volume expansion.

Neutropenia and Proteinures
Regular monitoring of white blood cell counts and protein levels in urine should be performed in patients with collagen vascular disease, like lupus erythematosus and systemic sclerosis, in particular where associated with impaired renal function and concomitant therapy with drugs like corticosteroids and antimetabolites.

Hyperkalaemia
Elevated serum potassium may occur in hypertensive patients. Risk factors for the development of hyperkalaemia include renal insufficiency and the concomitant use of agents to treat hyperkalaemia, as well as potassium sparing diuretics.

Gastrointestinal tract
Gastrointestinal disorders, e.g. nausea, diarrhoea or gastric pain may occur but these reactions are often transient. Taste disturbances may occur.

Angioneurotic oedema
Angioneurotic oedema may occur during therapy with ACE inhibitors including TRITACE. If laryngeal stridor or angio-oedema of the face, tongue or glottis occurs treatment with TRITACE must be discontinued and appropriate therapy instituted immediately.

Allergic reactions
Hypersensitivity reactions accompanied by pruritus, rash and sometimes fever may occur, but may resolve spontaneously after withdrawal of TRITACE.

Laboratory Values
Increases in blood urea and serum creatinine may occur, particularly in patients with renal insufficiency or in patients pretreated with a diuretic. Elevation of serum potassium may occur, since TRITACE decreases aldosterone secretion.

Potassium sparing diuretics such as spironolactone, amiloride, triamterene or potassium supplements should therefore be avoided.

Drug interactions
Combination with diuretics or other antihypertensives may potentiate the antihypertensive response to TRITACE. Potassium sparing diuretics such as spironolactone, amiloride, triamterene or potassium supplements increase the risk of hyperkalaemia. TRITACE may attenuate potassium loss caused by thiazide-type diuretics. Therefore, if concomitant use of these agents is indicated they should be given with caution and with frequent monitoring of serum potassium.

Concomitant therapy with lithium may increase the serum lithium concentration.

Interaction between ACE inhibitors and non-steroidal anti-inflammatory agents has been reported.

KNOWN SYMPTOMS OF OVERDOSAGE AND PARTICULARS OF ITS TREATMENT:
In case of overdosage excessive hypotension is to be expected. Treatment should include volume expanders.

IDENTIFICATION:
1,25 mg : yellow and white capsules
2,50 mg : medium orange and white capsules
5 mg : scarlet and white capsules
10 mg : blue and white capsules

PRESENTATION:
Cartons containing one or more blister packs with 10 capsules each.

STORAGE INSTRUCTIONS:
Store below 25°C.

The medicine must not be used after the expiry date printed on the pack.

Keep out of reach of children.

REGISTRATION NUMBERS:
TRITACE 1,25 mg : W/7.1.3/232
TRITACE 2,50 mg : W/7.1.3/233
TRITACE 5 mg : W/7.1.3/234
TRITACE 10 mg : W/7.1.3/235

NAME AND BUSINESS ADDRESS OF THE APPLICANT:
Hoechst Pharmaceuticals (Pty) Ltd.
(Reg. No. 58/01843/07)
3 Caxton Street
Industria 2092.

DATE OF PUBLICATION OF THIS PACKAGE INSERT:
08.09.1989.

H 54

Natrilix® TABLETS

SCHEDULING STATUS: S4

PROPRIETARY NAME:
NATRILIX® TABLETS

COMPOSITION:
Each tablet contains 2.5 mg indapamide [N-(3-sulphamoyl-4-chloro-benzamido) 2-methyl indoline.]

PHARMACOLOGICAL CLASSIFICATION:
A 7.1 (Hypotensive Drug)

PHARMACOLOGICAL ACTION:
Indapamide, an indole derivative of chlorosulphonamide, has an antihypertensive action causing a drop in systolic, diastolic and mean blood pressure. This antihypertensive action is maximal at a dose of 2.5 mg per day and the diuretic effect is slight, usually without clinical manifestation. At higher doses, the diuretic effect becomes more prominent. The extra-renal antihypertensive action of 2.5 mg per day is demonstrated as a decrease in vascular hyperreactivity and a reduction in total peripheral and arteriolar resistance. The extra-renal mechanism of action has also been demonstrated by the maintenance of anti-hypertensive effect in functionally anephric patients. The extra-renal action is thought to be due to the inhibition of transmembrane ionic influx, essentially calcic, and the stimulation of synthesis of the vasodilatory hypotensive prostaglandin PGE_2.

PHARMACOKINETICS
Indapamide is rapidly and completely absorbed after oral administration. Peak blood levels are reached after 1 to 2 hours. Indapamide is extensively metabolised and only 5-7% is found unchanged in the urine. The elimination half-life is 14-18 hours. Indapamide is 79% bound to plasma protein. The methyl-indoline portion of the molecule gives indapamide its lipophilic character, and indapamide's lipid solubility is 5 to 8 times that of the thiazides. It is as a result of this characteristic that indapamide localizes in smooth vascular muscle.

INDICATIONS:
NATRILIX (indapamide) is indicated in the management of mild to moderate hypertension.

CONTRA-INDICATIONS:
Severe hepatic insufficiency.
Safety in pregnancy has not been established.

DOSAGE AND DIRECTIONS FOR USE:
Adults: Maximum dose is 2.5 mg per day. The dosage is one tablet containing 2.5 mg indapamide hemihydrate, daily, to be taken in the morning with breakfast. In more severe cases, NATRILIX can be combined with other categories of antihypertensive agents.
Children: There is no experience of the use of this drug in children.

SIDE-EFFECTS AND SPECIAL PRECAUTIONS:
Side-effects include giddiness and headaches.
Indapamide given at 2.5 mg daily produces minimal diuresis. However, hyperuricaemia and hypokalaemia may occur, and potassium supplementation may be required.
Serum potassium should be monitored in patients prone or sensitive to hypokalaemia (such as patients treated concomitantly with steroids, digitalis or laxative drugs). Uric acid should be monitored, particularly in patients with a history of gout, who should continue to receive the appropriate treatment. Indapamide 2.5 mg (one tablet) daily can be administered to hypertensive patients with impaired renal function, and to those undergoing chronic haemodialysis, as there is no evidence of drug accumulation. However, the treatment should be discontinued if increasing azotemia and oliguria occur.

The co-administration of NATRILIX with diuretics, which may cause hypokalaemia, is not recommended.

KNOWN SYMPTOMS OF OVERDOSAGE AND PARTICULARS OF ITS TREATMENT:
Symptoms:
These could include: allergies, skin rashes, epigastric pain, nausea, photo-sensitivity, dizziness, weakness and paraesthesia.

Treatment:
Treatment is supportive and symptomatic.

IDENTIFICATION:
Pink, lens-shaped, sugar-coated tablets.

PRESENTATION:
Containers of 30 and 600 tablets.

STORAGE INSTRUCTIONS:
Store below 25 °C in a dry place. Keep out of reach of children.

REGISTRATION NUMBER: G/7.1/65.

NAME AND BUSINESS ADDRESS OF APPLICANT:
SERVIER LABORATORIES (S.A.) (PTY) LTD
P.O. Box 930, Rivonia, 2128.
1st Floor, Rivonia Gate, Cnr. 12th Ave/Rivonia Road, Rivonia, Transvaal.

DATE OF PUBLICATION OF THIS PACKAGE INSERT: June, 1988.
810 2/90

-139-

PLEASE PRINT

S.A. INSTITUTE FOR MEDICAL RESEARCH

SENDER AND ADDRESS: GRAHAMSTOWN

HOSPITAL: SETTLERS WARD: Spec Clinic NO: STAFF Sick bay.

PATIENT: Mrs HOSPITAL/PRIVATE CASE

ADDRESS:

AGE: 51yo. SEX: F RACE: B

MEDICAL AID:

ACCOUNT TO:

PREV. SPEC. No.:

SPECIMEN: Blood COLL. DATE AND TIME: 8h:00 5/9/90

INVESTIGATIONS: Fasting Cholesterol.

CLINICAL DETAILS:

09/5900

CHEMICAL PATHOLOGY

LABORATORY REF. No.
B 016498

J

DATE AND TIME RECEIVED 1990 -09- -5 DATE OF REPORT

	S — SODIUM 136 - 148	mmol/ℓ
	S — POTASSIUM 3,8 - 5,0	mmol/ℓ
	S — CHLORIDE 97 - 106	mmol/ℓ
	S — UREA 2,5 - 6,6	mmol/ℓ
	S — CREATININE 44 - 106	µmol/ℓ
	P — GLUCOSE 3,3 - 6,1	mmol/ℓ
	S — CALCIUM 2,25 - 2,75	mmol/ℓ
	S — PHOSPHORUS (Inorganic) 0,6 - 1,3	mmol/ℓ
	S — CHOLESTEROL 3,1 - 6,5 6,13	mmol/ℓ
	S — TRIGLYCERIDE 0,84 - 1,94	mmol/ℓ
	S — HDL M 0,9 - 1,4 / F 1,2 - 1,7	mmol/ℓ
	S — URATE M 0,190 - 0,430/F 0,140 - 0,330	mmol/ℓ
	S — MAGNESIUM 0,8 - 1,0	mmol/ℓ
	S - PROSTATIC ACID PHOSPHATASE 0 - 4	U/ℓ

	S — TOTAL PROTEIN 62-82	g/ℓ
	S — ALBUMIN 35 - 57	g/ℓ
	S — BILIRUBIN (TOTAL) 5 - 20	µmol/ℓ
	S — BILIRUBIN (CONJUGATED) 5 - 20	µmol/ℓ
	S — ALKALINE PHOSPHATASE 9 - 35	U/ℓ
	S — ASPARTATE TRANSAMINASE 0 - 26	U/ℓ
	S — ALANINE TRANSAMINASE 0 - 32	U/ℓ
	S — γ — GLUTAMYLTRANSFERASE M8 - 37 / F5 - 24	U/ℓ
	S — LACTATE DEHYDROGENASE 160 - 320	U/ℓ
	S — HBD 0 - 169	U/ℓ
	S — CREATINE KINASE (TOTAL) 15 - 125	U/ℓ
	S — CK — MB 0 - 14	U/ℓ
	S — AMYLASE 100 - 300	U/ℓ
	S - TOTAL ACID PHOSPHATASE 0 - 11	U/ℓ

Macroscopic Appearance ☐ Normal ☒ Slightly Haemolysed ☐ Icteric ☐ Lipaemic

COMMENT:

Phoned at ..... On..... for REGIONAL DIRE

umleys p.e. FORM 2 *Hierdie vorm is ook in Afrikaans beskikbaar*

DIET SCHEDULE

90/08/30 FRIDAY
Supper
2 slices white bread (no brown available) & bowl of soup
a big portion of pumkin
Mielie rice
Cooked Spinach with mince stew
Mug of milky coffee
Bedtime Snack - Unpeeled grated apple
90/08/31
½ cup chocolate pronutro & good helping stem milk
Tea Time. Cheese Sandwich & cup tea
Lunch - Fish and chips
Supper. soup bread and 1 banana downed & 2 cups coffee.

(1)

90/09/01 SATURDAY
Breakfast - Maltabella porridge & stem milk
Lunch - Chips
Pm Tea Buttered Sandwich and cup tea + 1 banana
Supper. 3 slices fresh brown bread slices not buttered Pap and Spinach & piece of meat and gravy
2 glasses orange drink
Sunday 90/09/02
Breakfast Pronutro & milk porridge
Tea time Cheese Sandwiches + cup tea
Lunch. One banana
Pm Tea Sandwich & Cheese & Pkng

(2)

Supper - Mielie rice & Spinach & pumkin & mutton Stew. Downed & 2 glasses mineral drink
Snack bed time - 1 Banana
90/09/03 Monday
overslept - late for breakfast- Quick cup of coffee
Tea time Cheese sandwich with pickled fish - 2 cups tea
Lunch - 2 bananas
Pm Tea time Slice cheese & Sandwich
Supper - Mielie rice & Spinach piece mutton rich gravy
Mineral drink 1 glass

(3)

90/09/04
Breakfast : Nothing
Tea Time Pickled fish Sandwiches
Pm Tea time Bowl Soup and 2 thick Slices unbuttered bread
Supper Mashed potatoes & Spinach and piece tenderised steak
Canned peaches & ice cream topping
Plenty of water to drink
90/09/05
No breakfast - nothing taken

(4)

Appendix A

NOVACO ANGER INVENTORY
BEFORE

For each of the following items, please rate the degree to which the incident described by the item would *anger or provoke you* by using the following scale:

1	2	3	4	5
not at all	a little	some-not much	much	very much

Use the same scale for all of the items. Please score your responses to the items on the answer sheet provided. Try to imagine the incident *actually happening to you*, and then indicate the extent to which it would have made you angry by scoring the answer sheet.

2 1. On your way to go somewhere, you discover that you have lost the keys to your car.
5 2. Going for a haircut and getting more cut off than you wanted.
4 3. Being overcharged by a repairman who has you over a barrel.
5 4. Being singled out for correction, when the actions of others go unnoticed.
5 5. You are walking along, minding your own business, when someone comes rushing past, knocking you out of his way.
5 6. Being called a liar.
5 7. You are in the midst of a dispute, and the other person calls you a "stupid jerk."
4 8. Hearing that a person has been deprived of his constitutional rights.
5 9. Someone borrows your car, consumes one-third of a tank of gas, and doesn't replace it or compensate you for it.
3 10. People who think that they are always right.
5 11. You unpack an appliance that you have just bought, plug it in, and discover that it doesn't work.
5 12. You are waiting to be served at a restaurant. Fifteen minutes have gone by, and you still haven't even received a glass of water.
5 13. Struggling to carry four cups of coffee to your table at a cafeteria, someone bumps into you, spilling the coffee.
4 14. Getting your car stuck in the mud or snow.
5 15. You are typing a term paper, hurrying to make the deadline, and the typewriter jams.
4 16. Employers who take advantage of their employees' need for work by demanding more than they have a right to.

5 17. Watching someone bully another person who is physically smaller than he is.
5 18. Professors who refuse to listen to your point of view.
5 19. You have hung up your clothes, but someone knocks them to the floor and fails to pick them up.
4 20. Being stood-up for a date.
5 21. Someone sneaks into your room and takes your wallet.
3 22. You are driving to pick up a friend at the airport and are forced to wait for a long freight train.
3 23. You are driving along at 45 mph, and the guy behind you is right on your bumper.
4 24. You are talking to someone, and he doesn't answer you.
4 25. Hitting your finger with a hammer.
5 26. Newspapers slanting the news against a man in political office to make him look bad to the public.
5 27. You have made arrangements to go somewhere with a person who backs off at the last minute and leaves you hanging.
3 28. People asking personal questions of you just for their own curiosity.
5 29. Your car is stalled at a traffic light, and the buy behind you keeps blowing his horn.
5 30. Watching someone berate another person to excess.
5 31. Being pushed or shoved by someone in an argument.
5 32. You accidentally make the wrong kind of a turn in a parking lot. As you get out of your car someone yells at you, "Where did you learn to drive?"
3 33. Someone who pretends to be something that he is not.
5 34. You walk out to the parking lot, and you discover that your car has been towed away by the campus police.
4 35. Working hard on a project and getting a poor grade.
5 36. Someone makes a mistake and blames it on you.
5 37. You get in your car to drive to work, and the car won't start.
5 38. Being hounded by a salesman from the moment that you walk into a store.
4 39. Being given an unnecessarily difficult exam when you need a good grade.
3 40. You are deprived of a promotion to which you are entitled because you haven't played up enough to the right people.
3 41. Someone who tries to make you feel guilty.
3 42. You are trying to concentrate, and a person near you is tapping his foot.
5 43. Someone else's dog routinely defecating in your front yard.
5 44. When you are criticized in front of others for something that you have done.

6 Acts of economic exploitation whereby businessmen take advantage
5 of need and demand an excessive profit.
5 77. Being punished for saying what you really believe.
5 78. You are in a theater ticket line, and someone cuts in front of you.
5 79. Being forced to do something in a way that someone else thinks that it should be done.
5 80. You use your last 10¢ to make a phone call, and you are disconnected before you finish dialing.
5 81. In a hurry to get somewhere, you tear a good pair of slacks on a sharp object.
4 82. Being misled or deceived by a man holding political office.
5 83. You are out on a date with someone who subtly or indirectly conveys to you that you just don't measure up to their standards.
5 84. You are at a shopping center, and two evangelistic people stop you and want to convert you to their religious ideas.
4 85. While washing your favorite cup, you drop it and it breaks.
5 86. Getting punched in the mouth.
5 87. Being falsely accused of cheating.
5 88. Someone ripping off your automobile antenna.
5 89. Discovering that you were deliberately sold defective merchandise.
5 90. People who are cruel to animals.

SCORE = 398

5 45. You lend someone an important book and they fail to return it.
5 46. In the parking lot where you have left your car, the person whose car is next to yours swings open his door, chipping the paint from your car.
5 47. Getting cold soup or vegetables in a restaurant.
4 48. Someone who is always trying to get "one-up" on you.
5 49. It's a cold morning and you have an 8 o'clock class. Begrudgingly, you get there on time, but the prof arrives 15 minutes late and announces that he is cancelling the class.
5 50. You are sitting next to someone who is smoking, and he is letting the smoke drift right into your face.
4 51. People who constantly brag about themselves.
5 52. Being thrown into a swimming pool with your clothes on.
3 53. Being joked about or teased.
4 54. Banging your shins against a piece of furniture.
3 55. Being on the receiving end of a practical joke.
4 56. Being forced to do something that you don't want to do.
5 57. You are in a discussion with someone who persists in arguing about a topic he knows very little about.
4 58. Losing a game that you wanted to win.
3 59. Being told to "go to hell."
5 60. Someone making fun of the clothes that you are wearing.
4 61. Someone sticking their nose into an argument between you and someone else.
3 62. Being forced to participate in psychological experiments.
5 63. You are walking along on a rainy day, and a car drives past, splashing you with water from the street.
5 64. Acts of prejudice against a minority or ethnic group.
5 65. Someone spits at you.
5 66. You need to get somewhere quickly but the car in front of you is going 25 mph in a 40 mph zone, and you can't pass.
5 67. Being talked about behind your back.
4 68. Stepping on a gob of chewing gum.
5 69. Hearing that a very wealthy man has paid zero income tax.
5 70. You have just cleaned up an area and organized the things in it, but someone comes along and messes it up.
5 71. Getting hit in the back of the head with a snowball.
5 72. You are involved in watching a TV program, and someone comes along and switches the channel.
3 73. Being told by an employer or professor that you have done poor work.
3 74. You are in a ball game, and one of your opponents is unnecessarily rough.
5 75. Being mocked by a small group of people as you pass them.

Appendix A

NOVACO ANGER INVENTORY
AFTER

For each of the following items, please rate the degree to which the incident described by the item would *anger or provoke you* by using the following scale:

1	2	3	4	5
not at all	a little	some-not much	much	very much

Use the same scale for all of the items. Please score your responses to the items on the answer sheet provided. Try to imagine the incident *actually happening to you*, and then indicate the extent to which it would have made you angry by scoring the answer sheet.

1. On your way to go somewhere, you discover that you have lost the keys to your car. 3
2. Going for a haircut and getting more cut off than you wanted. 4
3. Being overcharged by a repairman who has you over a barrel. 4
4. Being singled out for correction, when the actions of others go unnoticed. 4
5. You are walking along, minding your own business, when someone comes rushing past, knocking you out of his way. 5
6. Being called a liar. 5
7. You are in the midst of a dispute, and the other person calls you a "stupid jerk." 2
8. Hearing that a person has been deprived of his constitutional rights. 2
9. Someone borrows your car, consumes one-third of a tank of gas, and doesn't replace it or compensate you for it. 4
10. People who think that they are always right. 3
11. You unpack an appliance that you have just bought, plug it in, and discover that it doesn't work. 2
12. You are waiting to be served at a restaurant. Fifteen minutes have gone by, and you still haven't even received a glass of water. 3
13. Struggling to carry four cups of coffee to your table at a cafeteria, someone bumps into you, spilling the coffee. 4
14. Getting your car stuck in the mud or snow. 4
15. You are typing a term paper, hurrying to make the deadline, and the typewriter jams. 4
16. Employers who take advantage of their employees' need for work by demanding more than they have a right to. 3

17. Watching someone bully another person who is physically smaller than he is. 5
18. Professors who refuse to listen to your point of view. 4
19. You have hung up your clothes, but someone knocks them to the floor and fails to pick them up. 4
20. Being stood-up for a date. 3
21. Someone sneaks into your room and takes your wallet. 5
22. You are driving to pick up a friend at the airport and are forced to wait for a long freight train. 4
23. You are driving along at 45 mph, and the guy behind you is right on your bumper. 3
24. You are talking to someone, and he doesn't answer you. 3
25. Hitting your finger with a hammer. 4
26. Newspapers slanting the news against a man in political office to make him look bad to the public. 3
27. You have made arrangements to go somewhere with a person who backs off at the last minute and leaves you hanging. 4
28. People asking personal questions of you just for their own curiosity. 3
29. Your car is stalled at a traffic light, and the buy behind you keeps blowing his horn. 3
30. Watching someone berate another person to excess. 5
31. Being pushed or shoved by someone in an argument. 4
32. You accidentally make the wrong kind of a turn in a parking lot. As you get out of your car someone yells at you, "Where did you learn to drive?" 4
33. Someone who pretends to be something that he is not. 3
34. You walk out to the parking lot, and you discover that your car has been towed away by the campus police. 5
35. Working hard on a project and getting a poor grade. 3
36. Someone makes a mistake and blames it on you. 4
37. You get in your car to drive to work, and the car won't start. 4
38. Being hounded by a salesman from the moment that you walk into a store. 4
39. Being given an unnecessarily difficult exam when you need a good grade. 4
40. You are deprived of a promotion to which you are entitled because you haven't played up enough to the right people. 4
41. Someone who tries to make you feel guilty. 3
42. You are trying to concentrate, and a person near you is tapping his foot. 3
43. Someone else's dog routinely defecating in your front yard. 5
44. When you are criticized in front of others for something that you have done. 4
45. You lend someone an important book and they fail to return it. 3
46. In the parking lot where you have left your car, the person whose car is next to yours swings open his door, chipping the paint from your car. 5
47. Getting cold soup or vegetables in a restaurant. 4
48. Someone who is always trying to get "one-up" on you. 4
49. It's a cold morning and you have an 8 o'clock class. Begrudgingly, you get there on time, but the prof arrives 15 minutes late and announces that he is cancelling the class. 5
50. You are sitting next to someone who is smoking, and he is letting the smoke drift right into your face. 5
51. People who constantly brag about themselves. 4
52. Being thrown into a swimming pool with your clothes on. 5
53. Being joked about or teased. 4
54. Banging your shins against a piece of furniture. 4
55. Being on the receiving end of a practical joke. 3
56. Being forced to do something that you don't want to do. 5
57. You are in a discussion with someone who persists in arguing about a topic he knows very little about. 4
58. Losing a game that you wanted to win. 3
59. Being told to "go to hell." 3
60. Someone making fun of the clothes that you are wearing. 3
61. Someone sticking their nose into an argument between you and someone else. 3
62. Being forced to participate in psychological experiments. 4
63. You are walking along on a rainy day, and a car drives past, splashing you with water from the street. 5
64. Acts of prejudice against a minority or ethnic group. 5
65. Someone spits at you. 5
66. You need to get somewhere quickly but the car in front of you is going 25 mph in a 40 mph zone, and you can't pass. 4
67. Being talked about behind your back. 4
68. Stepping on a gob of chewing gum. 4
69. Hearing that a very wealthy man has paid zero income tax. 4
70. You have just cleaned up an area and organized the things in it, but someone comes along and messes it up. 4
71. Getting hit in the back of the head with a snowball. 5
72. You are involved in watching a TV program, and someone comes along and switches the channel. 4
73. Being told by an employer or professor that you have done poor work. 3
74. You are in a ball game, and one of your opponents is unnecessarily rough. 3
75. Being mocked by a small group of people as you pass them. 3

76. Acts of economic exploitation whereby businessmen take advantage of need and demand an excessive profit. 4
77. Being punished for saying what you really believe. 5
78. You are in a theater ticket line, and someone cuts in front of you. 4
79. Being forced to do something in a way that someone else thinks that it should be done. 4
80. You use your last 10¢ to make a phone call, and you are disconnected before you finish dialing. 5
81. In a hurry to get somewhere, you tear a good pair of slacks on a sharp object. 4
82. Being misled or deceived by a man holding political office. 4
83. You are out on a date with someone who subtly or indirectly conveys to you that you just don't measure up to their standards. 5
84. You are at a shopping center, and two evangelistic people stop you and want to convert you to their religious ideas. 5
85. While washing your favorite cup, you drop it and it breaks. 4
86. Getting punched in the mouth. 5
87. Being falsely accused of cheating. 5
88. Someone ripping off your automobile antenna. 4
89. Discovering that you were deliberately sold defective merchandise. 5
90. People who are cruel to animals. 5

SCORE = 360

Beside each below, indicate whether that item is true or false for you:

1 1. I do not tire quickly. F

___ 2. I am troubled by attacks of nausea. F

1 3. I believe I am no more nervous than most people. F

1 4. I have very few headaches. F

1 5. I work under a great deal of tension. T

1 6. I cannot keep my mind on one thing. T

1 7. I worry over money and business. T

___ 8. I frequently notice my hands shake when I try to do something. F

___ 9. I blush no more often than others. T

___ 10. I have diarrhea once a month or more. F

___ 11. I worry quite a bit over possible misfortunes. F

1 12. I practically never blush. F

___ 13. I am often afraid I am going to blush. F

___ 14. I have nightmares every few nights. F

___ 15. My hands and feet are usually warm enough. T

___ 16. I sweat very easily even on cool days. F

1 17. Sometimes when embarrassed, I break out in a sweat, which annoys me greatly. T

___ 18. I hardly ever notice my heart pounding, and I am seldom short of breath. T

___ 19. I feel hungry almost all the time. F

___ 20. I am very seldom troubled by constipation. T

___ 21. I have a great deal of stomach trouble. F

1 22. I have had periods in which I lost sleep over worry. T

___ 23. My sleep is fitful and disturbed. F

1 24. I deram frequently about things that are best kept to myself. T

___ 25. I am easily embarrassed. F

1 26. I am more sensitive than most other people. T

1 27. I frequently find myself worrying about something. T

____ 28. I wish I could be as happy as others seem to be. F

1 29. I am usually calm and not easily upset. F

1 30. I cry easily. T

____ 31. I feel anxiety about something or someone almost all the time. F

____ 32. I am happy most of the time. T

1 33. It makes me nervous to have to wait. T

____ 34. I have periods of such great restlessness that I cannot sit long in a chair. F

1 35. Sometimes I become so excited that I find it hard to get to sleep. T

____ 36. I have sometimes felt that difficulties were piling up so high that I could not overcome them. F

____ 37. I must admit that I have at times been worried beyond reason over something that really did not matter. F

____ 38. I have very few fears compared to my friends. T

____ 39. I have been afraid of things or people that I know could not hurt me. F

____ 40. I certainly feel useless at times. F

____ 41. I find it hard to keep my mind on a task or job. F

1 42. I am usually self-conscious. T

1 43. I am inclined to take things hard. T

____ 44. I am a high-strung person. F

____ 45. Life is a strain for me much of the time. F

____ 46. At times I think that I am no good at all. F

____ 47. I am certainly lacking in self-confidence. F

____ 48. I sometimes feel that I am going to pieces. F

____ 49. I shrink from facing a crisis or difficulty. F

____ 50. I am entirely self-confident. T

18 = [illegible]

[illegible]

ASSERTIVE BEHAVIOR

To determine your general behaviour, indicate how characteristic or descriptive each of the following statements is of you by using the code that follows:

+3 = very characteristic of me, extremely descriptive
+2 = rather characteristic of me, quite descriptive
+1 = somewhat characteristic of me, slightly descriptive
-1 = somewhat uncharacteristic of me, slightly nondescriptive
-2 = rather uncharacteristic of me, quite nondescriptive
-3 = very uncharacteristic of me, extremely nondescriptive

-1 1. Most people seem to be more aggressive and assertive than I am. +1

+1 2. I have hesitated to make or accept dates because of "shyness". -1

-3 3. When the food done at a restaurant is not done to my satisfaction, I complain about it to the waiter or waitress. -3

+2 4. I am careful to avoid hurting other people's feelings, even when I feel that I have been injured. -2

+3 5. If a salesperson has gone to considerable trouble to show me merchandise that is not quite suitable, I have a difficult time in saying no. -3

+3 6. When I am asked to do something, I insist upon knowing why. +3

+2 7. There are times when I look for a good, vigorous arguement. +2

+2 8. I strive to get ahead as well as most people in my position. +2

+2 9. To be honest, people often take advantage of me. -2

+3 10. I enjoy starting conversations with new acquaintances and strangers. +3

+3 11. I often don't know what to say to attractive persons of the opposite sex. -3

+3 12. I will hesitate to make phone calls to business establishments and institutions. − 3

+3 13. I would rather apply for a job or for admission to a college by writing letters than by going through with personal interviews. − 3

+3 14. I find it embarrassing to return merchandise. − 3

+2 15. If a close and respected relative were annoying me, I would smother my feelings rather than accept my annoyance. − 2

+2 16. I have avoided asking questions for fear of sounding stupid. − 2

−3 17. During an arguement I am sometimes afraid that I will get so upset that I will shake all over. + 3

+2 18. If a famed and respected lecturer makes a statement that I think is incorrect, I will have the audience hear my point of view as well. + 2

+2 19. I avoid arguing over prices with clerks and salespeople. − 2

+3 20. When I have something important or worthwhile, I manage to let others know about it. + 3

+3 21. I am open and frank about my feelings. + 3

+2 22. If someone has been spreading false and bad stories about me, I see him/her as soon as possible to "have a talk" about it. + 3

−2 23. I often have a hard time saying no. + 2

−1 24. I tend to bottle up my emotions rather than make a scene. + 1

____ 25. I complain about poor service in a restaurant and elsewhere. + 2

−3 26. When I am given a compliment, I sometimes just don't know what to say. + 3

+1 27. If a couple near me ina theatre or at a lecture were conversing rather loudly, I would ask them to be quiet or to take their conversation elsewhere. + 1

+2 28. Anyone attempting to push ahead of me in line is in for a good battle. + 2

+3 29. I am quick to express an opinion. +3

-1 30. There are times when I just can't say anything. +1

31 [illegible]

LOCUS OF CONTROL

Circle the answers below that best describe your beliefs:

1. (a) Grades are a function of the amount of work students do.
   b. Grades depend on the kindness of the instructor.
2. (a) Promotions are earned by hard work.
   b. Promotions are the result of being in the right place at the right time.
3. a. Meeting someone to love is a matter of luck.
   (b) Meeting someone to love depends on going out often so as to meet many people.
4. a. Living a long life is a function of heredity.
   (b) Living a long life is a function of adopting healthy habits.
5. a. Being overweight is determined by the number of fat cells you were born with or developed early in life.
   (b) Being overweight depends on what and how much food you eat.
6. (a) People who exercise regularly set up their schedules to do so.
   b. Some people just don't have the time for regular exercise.
7. a. Winning at poker depends on betting correctly.
   (b) Winning at poker is a matter of being lucky.
8. (a) Staying married depends on working at the marriage.
   b. Marital breakup is a matter of being unlucky in choosing the wrong marriage partner.
9. (a) Citizens can have some influence on their governments.
   b. There is nothing an individual can do to affect government function.
10. a. Being skilled at sports depends on being born well coordinated.
    (b) Those skilled at sports work hard at learning those skills.
11. (a) People with close friends are lucky to have met someone to be intimite with.

b. Developing close friends takes hard work.

12. a. Your future depends on who you meet and on chance.

(b) Your future is up to you.

13. a. Most people are sure of their opinions that their minds cannot be changed.

(b) A logical arguement can convince most people.

14. (a) People decide the direction of their lives.

b. For the most part we have little control of our futures.

15. (a) People who don't like you just don't understand you.

b. You can be liked by anyone you choose to like you.

16. (a) You can make your life a happy one.

b. Happiness is a matter of fate.

17. (a) You evaluate feedback and make decisions based upon it.

b. You tend to be easily influenced by others.

18. a. If voters studied nominees' records, they could elect honest politicians.

(b) Politicians and politics are corrupt by nature.

19. a. Parents, teachers and bosses have a great deal to say about one's hapiness and self-satisfaction.

20. a. Air pollution can be controlled if citizens would get angry about it.

(b) Air pollution is an inevitable result of technological progress.

15 = TOTAL [illegible] SCALE [illegible]

5/23/79

DWIGHT TERRY "A SOCIAL SUPPORT SCHEDULE"

SOCIAL SUPPORT SCHEDULE

Complete the following scale by carefully reading each of the statements and placing one of the following letters in the blank spaces provided:

A = strongly agree

B = agree

C = uncertain

D = disagree

E = strongly disagree

0 1. Sometimes I feel all alone in the world. E

___ 2. I worry about the future facing today's children. A

3 3. I don't get invited out by friends as often as I'd really like. B

___ 4. The end often justifies the means. B

3 5. Most people today seldom feel lonely. D

___ 6. Sometimes I have the feeling other people are using me. B

___ 7. People's ideas change so much that I wonder if we'll ever have anything to depend on. D

1 8. Real friends are as easy as ever to find. B

___ 9. It is frightening to be responsible for the development of a little child. D

___ 10. Everything is relative, and there just aren't any definite rules to live by. E

0 11. One can always find friends, if one is friendly. A

___ 12. I often wonder what the meaning of life really is. E

___ 13. There is little or nothing I can do towards preventing a major "shooting" war. B

3 14. The world in which we live is basically a friendly place. D

____ 15. There are so many decisions that have to be made today that sometimes I could just blow up. A

____ 16. The only thing that one can be sure of today is that one can be sure of nothing. D

1 17. There are few dependable ties between people anymore. D

____ 18. There is little chance for promotion on the job unless a person gets a break. A

____ 19. With so many religions abroad, one doesn't really know which to believe. D

____ 20. We're so regimented today that there's not much room for choice even in personal matters. D

____ 21. We are just cogs in the machinery of life. E

1 22. People are just naturally friendly. B

____ 23. The future looks very dismal. D

3 24. I don't get to visit friends as often as I'd like. B

15 = Total Score

Score 0 – 36. The higher the score, the more socially isolated you believe you are to be.

"Occupational Stress Scale"

. For each question below, place the number that represents your answer in the space provided. These questions were developed by Weiman.[1]

1 = never
2 = seldom
3 = sometimes
4 = frequently
5 = nearly all the time

4 1. How often do you feel that you have too little authority to carry out your responsibilities? 4

4 2. How often do you feel unclear about just what the scope and responsibilities of your job are? 4

5 3. How often do you not know what opportunities for advancement or promotion exist for you? 5

3 4. How often do you feel that you have too heavy a work load, one that you could not possibly finish during an ordinary workday? 3

4 5. How often do you think that you will not be able to satisfy the conflicting demands of various people around you? 4

2 6. How often do you feel that you are not fully qualified to handle your job? 2

3 7. How often do you not know what your superior thinks of you, how he or she evaluates your performance? 3

1 8. How often do you find yourself unable to get information needed to carry out your job? 1

3 9. How often do you worry about decisions that affect the lives of people that you know? 3

2 10. How often do you feel that you may not be liked and accepted by people at work? 2

4 11. How often do you feel unable to influence your immediate supervisor's decisions and actions that affect you? 4

2 12. How often do you not know just what the people you work with expect of you? 2

3 13. How often do you think that the amount of work you have to do may interfere with how well it is done? 3

3 14. How often do you feel that you have to do things on the job that are against your better judgment? 3

5 15. How often do you feel that your job interferes with your family life? 5

= Total Score ÷ 15 = 3,2

Specific applications Score 0-5

[redacted]

APPENDIX 2

GUIDELINES TO BENSON'S RELAXATION RESPONSE

(1) Sit quiety in a comfortable position.

(2) Close your eyes.

(3) Deeply relax all your muscles, beginning at your feet and progressing up to your face. Keep them relaxed.

(4) Breathe through your nose. Become aware of your breathing. As you breathe out, say the word, "ONE", silently to yourself. For example, breathe IN...OUT, "ONE"; IN...OUT, "ONE"; etc. Breathe easily and naturally.

(5) Continue for 10 to 20 minutes. You may open your eyes to check the time, but do not use an alarm. When you finish, sit quiety for several minutes, at first with your eyes closed and later with your eyes opened. Do not stand up for a few minutes.

(6) Do not worry about whether you are successful in achieving a deep level of relaxation. Maintain a passive attitude and permit relaxation to occur at its own pace. When distracting thoughts occur, try to ignore them by not dweling upon them and return to repeating "ONE". With practice, the response should come with little effort. Practice the technique once or twice daily, but not within two hours after any meal, since the digestive processes seem to interfere with the elicitation of the Relaxation Response.

From: The Relaxation Response by Herbert Benson and Miriam Klipper

Synopsis of Anger Management Principles Given to Clients

The following is a version of the anger management principles that were given to clients in a printed form as part of the educational phase of treatment.

1. Some of the time and maybe a lot of the time, becoming angry has something to do with doubting yourself, being unsure, or feeling threatened by someone else. It's always important to *remember that you are a worthy person* and that you have many good qualities.

2. Sometimes you get angry because you take things personally when there is no need to do that. But even when someone is being directly offensive to you, you can control and contain your anger by staying task oriented—that is, the most important thing to do is stay focused and *stick to what must be done in the situation to get the outcome you want*. When you begin taking insults personally, you get distracted from your task and get caught up in an unnecessary combat. Don't let yourself get sidetracked and get baited into a quarrel. Recognize what the other person is doing as a provocation but stay task oriented and issue focused.

3. Sometimes you get angry simply because it is the one thing you have always done in a certain kind of situation. As you learn alternative ways of reacting to provocations that don't involve anger, you will be less inclined to react with anger.

4. One of the most important things you must do to control your anger is to recognize the signs of arousal as soon as they occur. As you become more and more sharply tuned to the signs of tension and upset inside you, you will achieve greater ability to short circuit the anger process. Heightened anger makes you agitated and impulsive. As you learn to relax more easily, your ability to regulate anger will improve.

5. Your anger can serve a very useful function and that is it can be an alerting signal for you that you are becoming upset and that effective action is called for, if a positive outcome is to result. *Use your anger to work to your advantage.* Remember, getting angry makes you agitated and impulsive; and *impulsive, antagonistic acts get you into trouble. Stay task oriented and instruct yourself.*

6. Sometimes you get angry because things look like they are getting out of hand and you want to take charge. Sometimes you are afraid that things will not go the way you want them, so you get angry to control them. You will learn that when you self-instruct and manage your anger, you *are* in control of the situation. *The best way to take charge of a situation can be not to get angry when most people would expect or even want you to be upset.*

7. As you learn to break down provocation experiences into chunks or stages, you will have a better handle on things, which is another way of putting you on top of the situation. You will also learn how to instruct yourself in ways that correspond to these stages.

8. Sometimes you get annoyed, upset, and angry because you have spent more time being problem-conscious that you have been accomplishment-conscious. *You often forget or dismiss the good things* that you do, but you don't let yourself get away with the mistakes and failings. You must *remember to congratulate yourself* when you have succeeded in managing your anger and let yourself feel good about it.

Examples of Self-Statements for Various Provocation Stages

Preparing for a Provocation:
What is it that I have to do?
I can work out a plan to handle this.
I can manage this situation. I know how to regulate my anger.
If I find myself getting upset, I'll know what to do.
There won't be any need for an argument.
Time for a few deep breaths of relaxation. Feel comfortable, relaxed, and at ease.
This could be a testy situation, but I believe in myself.

Confronting the Provocation:
Stay calm. Just continue to relax.
As long as I keep my cool, I'm in control here.
Don't get all bent out of shape; just think of what to do here.
You don't need to prove yourself.
There is no point in getting mad.
I'm not going to let him get to me.
Don't assume the worst or jump to conclusions. Look for the positives.
It's really a shame that this person is acting the way she is.
For a person to be that irritable, he must be awfully unhappy.
If I start to get mad, I'll just be banging my head against the wall. So I might as well just relax.
There's no need to doubt myself. What he says doesn't matter.

Coping with Arousal and Agitation:
My muscles are starting to feel tight. Time to relax and slow things down.
Getting upset won't help.
It's just not worth it to get so angry.
I'll let him make a fool of himself.
It's reasonable to get annoyed, but let's keep the lid on.
Time to take a deep breath.
My anger is a signal of what I need to do. Time to talk to myself.
I'm not going to get pushed around, but I'm not going haywire either.
Let's try a cooperative approach. Maybe we are both right.
He'd probably like me to get really angry. Well, I'm going to disappoint him.
I can't expect people to act the way I want them to.

Self-Reward:
It worked!
That wasn't as hard as I thought.
I could have gotten more upset than it was worth.
My ego can sure get me in trouble, but when I watch that ego stuff I'm better off.
I'm doing better at this all the time.
I actually got through that without getting angry.
I guess I've been getting upset for too long when it wasn't even necessary.