TYPE A BEHAVIOUR PATTERN AND CORONARY HEART DISEASE IN THE SOUTH AFRICAN INDIAN POPULATION

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A thesis submitted in partial fulfilment of
the requirements for the degree of
Master of Arts (Clinical Psychology)
in the Department of Psychology
at the University of Natal

Pietermaritzburg
August, 1991

DECLARATION

I declare that the contents of this thesis, unless specified, represents my own original work.

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1991

ACKNOWLEDGEMENTS

I would like to express my appreciation to the following people:

Doug Wassenaar, my supervisor, for his guidance and active interest in the study.

Bruce Faulds, for his willing assistance and direction with statistical analysis of results.

Dr Dwarkapersad of Northdale Hospital for granting access to patients at Northdale Hospital and Dr Ranchod of Northdale Hospital for his advice.

The staff at C Ward, Northdale hospital for putting up with my repeated intrusions.

Alex, for her constant encouragement and for helping me keep this study in perspective, and my parents for their support throughout my studies.

The financial assistance of the Human Sciences Research Council is also appreciated.

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ABSTRACT

This study investigates the relationship between coronary heart disease and a number of established coronary risk factors among Indian males. The study also includes factors that are gaining recognition as coronary risk factors. The study gives particular focus to the Type A behaviour pattern (sometimes referred to as the coronary-prone behaviour pattern).

A group of patients (n = 25) with myocardial infarction from a local hospital was compared to (a) a control group of non-coronary patients (n = 25) at the same hospital, and (b) a control group of apparently healthy individuals (n = 25). The groups were compared to see whether the following risk factors were able to distinguish those with myocardial infarction from controls: Type A behaviour pattern, stress levels, anger levels, diabetes, previous coronary heart disease, hypertension, obesity, family history of coronary heart disease, physical exercise, cigarette smoking and alcohol consumption.

Subjects were matched for age, sex, marital status, ethnic grouping and employment status.

Statistical analysis indicated that none of the risk factors, except Type A behaviour pattern, were able to significantly distinguish the myocardial infarction group from controls. Type A behaviour pattern was the only factor able to significantly distinguish the myocardial infarction group from controls.

CHAPTER 1: INTRODUCTION

1.1. General Overview

Coronary heart disease (CHD) is the leading cause of death in most industrialised countries (Heaton, 1988). Modern civilisations are facing not only an increase in this rate, but younger and younger people are suffering from CHD (Bakal, 1979). These trends are also reflected in South Africa where CHD is seen to be a major physical problem, especially among the Indian population. Wentworth Hospital in Durban sees approximately 10 000 out-patients every year, 20% of whom are Indians (Mitha, 1980). South Africa has the highest CHD incidence in the world with 23.1 deaths per 100 000 people in the 25-34 age range as compared to 9.3 deaths per 100 000 in the United States (Mitha, 1980). Within South Africa, Asian men have the highest rates of CHD, (Wyndham, 1982; Balfe, Steinberg & Kustner, 1988) and hence they have the highest coronary mortality rates in the world (Balfe, et al., 1988). The high rate of CHD could be attributed to a number of factors as CHD has long been considered a disease with multifactorial origins (Jenkins, Zyzanski & Rosenman, 1979). There are a number of recognised risk factors that alert clinicians to individuals who may develop CHD. Some coronary risk factors (e.g. smoking) are well established (Epstein & Perkins, 1988), while others are becoming recognised with increasing support from research (Byrne, 1987b). One of the issues presently being investigated by a large number of researchers is the possibility of a coronary-prone behaviour pattern, often referred to as Type A behaviour pattern (TABP).

TABP is a set of behaviours that has been proposed as a coronary risk factor among Westerners and those living Western life styles (Cohen, 1978). The pattern is characterised by impatience, hostility and competitiveness in response to environmental demands (Friedman & Rosenman, 1974). Cohen, Matthews and Waldron (1978) claim that as non-industrialised cultures begin to assimilate Western values, their risk for CHD increases. As Indian males in South Africa appear to particularly vulnerable to CHD, it is of importance to identify the risk factors that best identify individuals at risk for CHD. As the weightings of different coronary risk factors has been found to vary between cultures (Rosenman, Swan & Carnelli, 1988), it is possible that certain risk factors are better able to discriminate between those with CHD and those relatively free of the condition. Observations of coronary patients at Northdale hospital in Pietermaritzburg would suggest that coronary patients present with predominantly classical risk factors and few appear to exhibit TABP.

1.2. Aim of This Study

The aim of this study is to investigate the incidence of the standard risk factors for CHD as well as non-standard coronary risk factors (such as TABP and stress) among Indian males. The study pays particular attention to TABP, and aims to assess the strength of the CHD-TABP association in this population group. The mechanisms that may mediate the TABP-CHD relationship are also investigated. Literature and findings are interpreted in the context of health psychology (Matarazzo & Leckliter, 1988) and the biopsychosocial model proposed by Engel (1977).

CHAPTER 2: LITERATURE REVIEW

2.1. Introduction:

This chapter reviews the context within which health psychology has arisen. Major factors that led to the emergence of the discipline are seen to be the call for a biopsychosocial approach to health issues (e.g. Engel, 1977). The vulnerability model (Zubin & Steinhauer, 1981) is seen as a working model that integrates biological, psychological and social components of illness in a way that assists in seeing chronic disease such as CHD as a disease of multifactorial pathogenesis (Jenkins, 1988). Issues surrounding CHD, TABP and stress are reviewed in a manner consistent with the vulnerability model.

2.2. The Need for a New Perspective on Health.

Engel (1977) has convincingly argued that medicine is in a crisis. He argues that both medicine and psychiatry have adhered to and been based on a model of disease that reduces illness to a biomedical phenomenon. He claims that the biomedical model has served as a useful guideline in the past, but has become a dogma that allows no room for social and psychological dimensions of the ill person. These psychological and social factors are, he argues, critical in influencing reported onset of illness as well as the variations in the course of illness. In addition, psychological and social factors may contribute to continued patienthood despite the correction of biochemical abnormalities. It is argued that treating illness is more than the removal of single causes of disease. There are a host of factors that affect

an ill person at a number of levels.

Engel (1980) proposes the biopsychosocial model as a scientific framework that takes into account the deficiencies in the biomedical model. This framework allows for the humanity of the patient and stresses the importance of the person as part of a larger system. Each system is made up of a number of organised levels which are separated by permeable boundaries. An individual in such a system will be sensitive to changes in other parts of the system as all parts are organised into a hierarchical relationship. In turn, the larger system (e.g. family) will be affected by the ill person.

This model is based on general systems theory advocated by von Bertalanffy (1975). Some of the levels of this organisation that need to be taken into account with an ill person are: Society, Culture, Community, Family, Person, Nervous system, Organ (System), Tissue, Cell and Molecule. By dealing with related events as specific levels that contribute to the collective system, the critical determinants of illness can be isolated. This counteracts the reductionalism of the biomedical model that often isolates determinants that are trivial when seen within a biopsychosocial approach. Engel claims that the model does not further burden the health professional, but provides a framework that enables the practitioner to intervene rationally with illness in a holistic manner.

2.3. The Emergence of Health Psychology

A function of the biopsychosocial model has been the increased contribution of psychologists in issues of health (Weiss, 1987). The term **health psychology** is however relatively new and the Division of Health Psychology of the American Psychological Association was convened in 1978 (Feuerstein, Labbe & Kuczmierczyk, 1986). Health psychology can be defined as

> any activity of psychology relating to any aspect of health, illness, the health care system, or health policy formation... (Feuerstein et al., 1986, p.3)

Hence, typical questions asked by the health psychologist are What factors contribute to illness and health behaviour in individuals? What is stress and how does it contribute to the development of illness? What role can the psychologist play in the health care system?

Health psychology focuses on the aetiology of health problems, the concurrence of psychological processes and medical problems, and the development of specifically psychological techniques that assist those who are ill (Broome, 1989).

2.3.1. Antecedents of Health Psychology

Historically, people have realised that they have some control over their health since about 500 B.C. (Feuerstein et al., 1986). Instead of magical causes for illness, the early Greek physicians stressed that a physical and emotional balance would maintain good health. Feuerstein et al. (1986) identify modern pioneers in this area as Wolff (1953) and Alexander (1950). Their studies

of how distress affects the development of illness emphasised the relationship between mind and body. Studies conducted by Cannon (1936) helped in the understanding of how repeated psychobiological responses could damage tissues in the body. Together these researchers promoted psychosomatic concepts such as the pathogenic potential of repeated psychophysiological responses to environmental stress. Feuerstein et al. (1986) also emphasise the contribution of Melzack and Wall (1965) in the development of health psychology. Their research on pain proposed that pain was influenced by both physiological and psychological processes. Other factors that have contributed to the establishment and acceptance of the role psychology can play in a medical setting include the decline in single cause infectious disease and the increase in multicausal chronic illness (Harvey, 1988). Psychologists have become involved in the treatment of psychological concomitants to such chronic illnesses. The increase in health care costs have also attributed to the focus on prevention of disease and promotion of health by modifying behavioral risks (Weiss & Schwartz, 1981).

2.3.2 Current Trends in Health Psychology

Concurrent with the emergence of health psychology has been the change in emphasis from specialised treatment to a more integrated health care system that acknowledges the importance of lifestyle (Feuerstein et al., 1986). Health care practitioners and patients have become more concerned with lifestyles that advance health. Health has in the past been seen as the absence of disease (Ford, 1983) and hence a passive phenomenon. However,

health is coming to be seen as an active promotion and maintenance of wellbeing of the person (Strumpfer, 1990). Health and illness can be seen on a continuum with a pathogenic emphasis (how people develop illness) at the one end and a salutogenic emphasis (how people remain healthy) at the other. Strumpfer (1990) emphasises how the two approaches compliment each other.

Salutogenesis has been proposed as a complementary paradigm to the predominant paradigm of pathogenesis by Strumpfer (1990). He claims that this paradigm balances the pathogenic focus on abnormality by looking at what factors keep individuals healthy. Holahan and Moos (1987) stress this in their model of illness. After studying a sample of 405 adults and their children, Holahan and Moos delineate self efficacy, self confidence, good social network resources and personal resources as some of the factors that may assist people remain healthy. Similarly Kritz and Moos (1974) see support, cohesion and affiliation (how close members of a community feel towards one another) as salutogenic factors.

While this study will attempt to bear salutogenic factors in mind, the focus on CHD necessitates pathogenic emphasis. A biopsychosocial model advocated by Zubin and Steinhauer (1981) deals with such pathogens in an integrated framework.

Pathogenesis plays an integral role in the biopsychosocial approach to illness. However, Engel (1977) does not describe the workings of the model in detail (Feuerstein et al., 1986). Zubin and Steinhauer (1981) offer a application of the biopsychosocial

approach that has been an important contribution to the development of a working model of pathogenesis. While their paper focuses on schizophrenia, the concepts that they use can be usefully generalized to CHD. Both schizophrenia and CHD can be seen as episodic events that are precipitated when specific risks coincide.

An important contribution of the Zubin and Steinhauer model is the substitution of the linear concept of **cause** with the concept of **vulnerability.** A single event does not cause another event, but contributes to it. The nature of the resultant event is an interaction between person and environment that is mediated and moderated by a host of internal and external factors. When one generalises this way of looking at schizophrenia to medical conditions like CHD, the questions that one asks will be different. No longer does CHD appear to be a static condition, but the vulnerability to CHD changes as the health/illness threshold changes. Questions aimed at delineating cause will be replaced by questions that attempt to gauge vulnerability of an individual.

2.3.3. The Vulnerability Model

Zubin and Steinhauer (1981) begin their paper with a review of the advances in the understanding of schizophrenia prior to 1981. They acknowledge diagnostic progress, but call for an approach that will integrate different etiological models of schizophrenia. They state that the logjam of data and theory has impeded progress rather than contributed to the understanding of

schizophrenia. By themselves none of the models (genetic, ecological, learning theory, internal environment, neurophysiological) can account fully for the etiology of schizophrenia. Zubin and Steinhauer proceed to deal with the relative areas of competence of the different models. For example, they mention that it is widely accepted that a genotype for schizophrenia must be present in a person presenting with a schizophrenic episode. However, strict geneticists do not take enough notice of the environmental stressors that are needed to precipitate the phenotype. Zubin and Steinhauer argue that the interaction between various sources of vulnerability and specific stressors are essential factors in the development of a schizophrenic episode.

They use the concept of vulnerability to integrate the strengths of different approaches into an interactionalist model. They claim that

> The vulnerability model is a superordinate model that integrates the contributions of each of the others and provides for their interaction (p.481).

A parallel to this in the area of CHD, would be the need for a superordinate model that provides for the interaction of the factors that contribute to the onset of CHD. Jenkins, Zyzanski and Rosenman (1979) emphasise this when dealing with TABP:

> The Type A pattern is one of several important risk factors for a disease of multifactorial pathogenesis. None of these risk factors alone... is sufficiently sensitive to permit a clinician to predict with any assurance of accuracy whether or not a specific individual will incur clinical CHD (p8).

Therefore the clinician intent on predicting CHD in individuals will have to have an understanding of how different risks interact with each other.

Risk has been defined as the set of attributes (factors) that identify individuals that are likely to be diagnosed with a specific disease in the future (Matthews, 1982). The relationship between risk factor and illness is one of probability and should be distinguished from a causal agent (Zubin, 1979). Zubin and Steinhauer (1981) use the term **"marker"** to designate specific types of behaviour or performance that indicate onset, duration and offset of an illness episode. Hence a marker can be used to alert clinicians to the onset and duration of illness in a person already identified as at risk.

According to Jenkins (1988), the identification of risk factors has a twofold importance. Firstly, risk factors may be involved in the etiology of a condition and therefore reduction of the risk factor may reduce the future incidence of the condition. Secondly, should the risk factor be unalterable (as with gender and age in the case of CHD), or not involved in the etiology of the specific condition, the health practitioner is still alerted to a subgroup of people likely to incur the condition. Practitioners can then devote more energy to modifiable risk factors in these individuals and screen them on a more regular basis so that secondary intervention can be more immediate.

Zubin and Steinhauer (1981) see risk as a deficit in the person-environment interaction. A person with such a risk would be

considered vulnerable. When environmental demands (internal or external) strain the area(s) of deficit, the individual-environment balance is threatened. This threat may become illness when this balance is disturbed beyond a point where the person's immediate resources can reinstate equilibrium. A vulnerable individual need not develop a particular illness providing that stressors do not exceed their personal health/illness threshold.

This limit need not be stationary and is determined by:

a. the degree of vulnerability (ie risk): this risk may be acquired, inborn or elicited.
b. the life stresses needed to trigger the latent illness into actual illness.
c. moderating variables such as coping mechanisms, personality, and social networks.

The combination of the above needed to trigger an illness will differ from individual to individual. Triggers may not in themselves be very stressful, but they are stressful enough to exceed the illness threshold in an already weakened individual. Triggers tend to be

> undesirable, novel, unexpected, unanticipated, and uncontrollable; produce losses, and require considerable readjustment of routine (p.482).

Allied to vulnerability (the degree of risk) are the concepts of coping and competence (degree of resistance). Competence refers to the potential to deal with the environment (internal and external) and coping refers to the **extent** to which this potential is activated. Potentially resistant people may then develop an illness if they do not actively cope with the stresses that threaten to disturb their homeostasis.

Before dealing with these questions in more detail, coronary heart disease is outlined more fully below.

2.4. Coronary Heart Disease

Coronary heart disease, atherosclerotic (hardening of the arteries) heart disease and ischemic heart disease are terms used interchangeably to refer to the damage caused to the heart by an insufficient oxygen supply. All these are composite terms used to refer to a number of possible manifestations of lack of oxygen to the heart (Shepard, 1981). In this study, the term coronary heart disease (CHD) will be used throughout. CHD is produced by lesions of the coronary arteries. Excessive lesions of the arteries are referred to as coronary artery disease (CAD). Be cause CAD often develops into CHD, CAD is included here, with other forms of cardiac patterns.

2.4.1. Coronary Artery Disease

As the heart pumps blood through arteries, the blood presses against the artery walls and causes microscopic lesions. The artery generates new cells to repair damage and in so doing the artery walls become thicker. Blumenthal and Emery (1988) claim that this process begins in childhood and affects most people regardless of whether they develop CHD in the future.

However, when these repaired lesions (which consist of lipids) accumulate excessive amounts of lipids and cholesterol from passing blood, they can in time form plaques which constrict the

size of the lumen through which blood passes. If plaque growth is greater than the cellular blood supply, the artery may decompose and rupture. At this stage thrombi may occur and muscle tissue in need of oxygen is fed an inadequate supply. In the case of CHD, parts of the heart muscle receive an inadequate oxygen supply.

Exactly why occlusions develop is not certain, but focus is on the adhesiveness of blood platelets and the possible role of catecholamines (adrenaline and noradrenaline) in increasing platelet adhesiveness (Simpson, Olwine, Jenkins, Ramsey, Zyzanski, Thomas & Hames, 1974).

2.4.2. Angina Pectoris

Angina Pectoris occurs when the heart receives an inadequate supply of oxygen over a limited period of time. This results in anoxia and usually only occurs after the occlusion of at least one artery. Pain starts behind the sternum and spreads down the left arm. This typically occurs after psychological stress or physical exertion, and can be corrected by administering drugs such as beta blockers. This assists in bringing oxygenated blood to the heart and there is usually no permanent structural damage.

2.4.3 Myocardial Infarction

Myocardial infarction (the common heart attack or coronary thrombosis) occurs when there is a disruption of the oxygen supply to the heart muscle (myocardium) over an extended period of time. A thrombosis or clot gradually builds up in the coronary

arteries and typically restricts oxygen from the left ventricle of the heart. This happens independently of physical exertion and is more serious as the affected tissue dies. Pain in this case is similar to the pain experienced by an angina victim though longer in duration and more intense. Shortness of breath and weakness usually accompany the condition (Norton, 1982). There is a higher rate of mortality for those incurring an MI when compared to angina patients (Commerford, 1988). Drugs, such as betablockers and surgical techniques are some of the methods of medical intervention (Blumenthal & Emery, 1988)). However, once the cells in the heart muscle die, they are replaced by fibrous tissue. This further weakens the pumping action of the heart (Commerford, 1988).

Wyndham (1982) reviewed CHD trends in South Africa between 1968 and 1977 and found that in young Indians and whites, 59-77% of all deaths secondary to disease of the circulatory system were due to CHD. In a more recent study, Sewdarsen, Vythilingum, Jialal, Moodley and Mitha (1987) reported that 96% of their 108 Indian males with CHD had one or more of the standard coronary risk factors. These risk factors are now dealt with in more detail.

2.4.4. Coronary Risk Factors

Risk factors (often referred to as risk indicators or pathogens, Matarazzo & Leckliter, 1988) that identify those at risk for CHD have been well documented. Those referred to as traditional risk factors have been recognised for some time and consistent with

earlier references to the acquired/ inherited nature of vulnerability, the traditional or standard risk factors (based on Glass, 1977) can be separated into firstly those risks that are mostly considered behavioral or acquired risks and secondly those that are considered to be inherited. Some risks like obesity could be considered to be both a consequence of heredity and behavioral factors.

(a) Standard Coronary Risk Factors

Before a suspected risk factor is considered to be a valid risk factor, a number of criteria need to be addressed. These include

i. consistency (the association of the risk factor has been observed by different people in different locations over a time span)

ii. specificity (the association is limited to a specific disease)

iii. dose response (an increase in the risk dose is associated with an increase disease)

iv. plausibility (the link between the risk and the disease is biologically plausible) (Feuerstein et al., 1986).

The following have been accepted as standard coronary risks.

Inherited Risks

Gender (males have significantly higher rates of CHD throughout the lifecycle, Jenkins, 1988); **family history of CHD; hyperthyroidism** and **diabetes** are well documented as CHD risks.

Behavioural Risks

Dietary intake of animal fats (prospective studies have established a direct link between reported dietary intake and

subsequent CHD incidence [Shekelle, Shyrock, Paul, Lepper, Stamler, Lui & Raynor, 1981]); **cigarette smoking** (20 cigarettes a day may treble CHD risk; Kannel, in Krantz, Contrada, Hill & Friedler, 1988) and **physical inactivity** (physical inactivity reduces cardiac endurance and increases the percentage of body fat, Jenkins 1988). There is also evidence that excessive intake of alcohol may promote cardiac muscle deterioration (reviewed in Jenkins, 1988).

Inherited/Behavioral Risks

Increasing age; elevated serum cholesterol; elevated serum lipids; hypertension (levels of systolic and diastolic blood pressure have a linear relationship with CHD incidence, Jenkins, 1988) and **obesity** (a risk factor for total mortality as well as CHD, Jenkins, 1988).

Of these, smoking has been established as the single most predictive risk factor for CHD in Western countries (Bundy, 1989). South African research with Indian men has yielded results consistent with this finding. For example, 79% of the CHD patients in research conducted by Sewdarsen et al. (1987) smoked. The next highest indicator was elevated serum triglyceride levels (53%). The optimum combination of risk factors was a history of cigarette smoking, hypercholesterolaemia, hypertension, and abnormal glucose tolerance. Ninety six per cent of their patients had one or more of these risk factors. Combinations of risk factors in an individual also have a clear additive effect, although the exact manner in which these factors intensify risk is not known (Byrne, 1987a).

(b) Non-standard Coronary Risk Factors

While Glass (1977) claims that studies measuring marital status, occupation, income level, religion and ethnicity have not shown consistent associations with CHD mortality/ morbidity, some studies are claiming associations in these and other areas. For example, marital status may play a role in moderating CHD (Fisher, 1986).

For a long time, clinicians have suspected that there may be a set of personality factors or behaviours that may constitute a coronary risk factor. For example, in 1892 Osler stated that one of his MI patients was

> ...not the delicate neurotic person... but the robust, the vigorous in mind and body, the keen and ambitious man. (in Byrne, 1987b, p.122)

One such set of behaviours that may be such a risk factor is Type A behaviour pattern.

Type A Behaviour Pattern (TABP)

The term "Type A Behaviour Pattern" was first used by the cardiologists Friedman and Rosenman (1974). In the 1950's they noticed that the majority of their cardiac patients presented as hurried, anxious people who were time conscious. They began to investigate what they believed to be a coronary-prone behaviour pattern, and later formalised the set of behaviours that they had observed (such as time consciousness, hostility, competitiveness) into the Type A Coronary-Prone Behaviour Pattern. This became known simply as Type A Behaviour Pattern (TABP) or Type A. A

relative absence of the behaviours was referred to as the Type B Non-Coronary-Prone Behaviour Pattern (TBBP) or simply as Type B.

Friedman and Rosenman's (1974) early research indicated a significant association between TABP and CHD. This was later supported by Rosenman, Brand, Jenkins, Friedman, Straus and Wurm (1975) who carried out an extensive investigation into TABP and CHD incidence. They studied more than 3 000 men from the San Francisco area over 8.5 years. The study (known as the Western Collaborative Group Study) became the classic study that documented a significant association between TABP and CHD among middle aged-men. Corroboration for the TABP-CHD association has been consistently reported in the West. Siegal, Matthews, and Leitch (1983) claim that a coronary heart disease (CHD) risk factor is twice as likely in men exhibiting TABP when compared to those exhibiting TBBP. This is after the traditional risk factors such as smoking and a family history of CHD have been partialled out.

The dichotomy between standard risk factors and TABP is artificial from a biopsychosocial perspective as the interaction between different risk factors is of importance. However, literature still refers to the two as separate entities. This reflects the reductionist position that Zubin and Steinhauer (1981) oppose. The perspective in this study is to consider TABP as part of a larger biopsychosocial system.

Despite such a dichotomy in literature, in recent years official agencies and medical organisations have formally adopted psychosocial factors such as TABP as coronary risk indicators (e.g. the National Heart, Lung, and Blood Institute endorsed TABP as a valid coronary risk indicator in 1981, Jenkins, 1988). Heaton (1988) claims that it is becoming recognised in medical circles that the most important risk factors for the development of CHD are behavioral, and Jenkins (1988) adds that

> It is essential that psychologists, physicians, other health professionals and the general public begin to recognise that coronary heart disease is primarily a behavioral disease (p.330).

The United States National Health's Review Panel (1981) has formally acknowledged that

> the available body of scientific evidence demonstrates that Type A Behavior... is associated with a clinically apparent CHD in employed middle-aged US Citizens. This increased risk is greater than that imposed by age, elevated levels of systolic blood pressure, serum cholesterol, and smoking. (in Wright, 1988, p.2)

The next chapter deals with TABP, its genesis, its measurement and some of the difficulties associated with the construct.

2.5. Type A Behaviour Pattern

TABP has been defined as

> an action-emotion complex that can be observed in any person who is aggressively involved in a chronic, incessant struggle to achieve more and more in less and less time (Friedman & Rosenman, 1977, p.206).

Friedman and Rosenman (1977) add that the pattern is hard to recognise as it is often socially accepted and encouraged.

However, there are a number of characteristics that have been outlined to give a brief description of Type A's. Rosenman (1978, p. 62) provides one such profile:

The Type A individual can be recognised in an interview by :-

1. A general expression of energy and vigour, alertness, and confidence.
2. A firm handshake and brisk walking pace.
3. Loud and/or vigorous voice.
4. Terse speech, abbreviated responses.
5. Clipped speech.
6. Rapid speech and acceleration of speech at the end of a longer sentence.
7. Explosive speech (speech punctuated with certain words spoken emphatically and this is established as speaker's general pattern) that may contain swear words.
8. Interrupting by frequent rapid responses given before another speaker has completed his question or statement.
9. Speech hurrying in the form of saying "yes, yes" or "mm, mm" or "right, right" or by nodding his head in assent while another person speaks.
10. Vehement reaction to questions relating to impedance of time progress (i.e. driving slowly, waiting in lines).
11. Use of clenched fist or pointing his finger at you to emphasise his verbalizations.
12. Frequent sighing especially related to questions relating to his work. It is important to differentiate from the sighs of a depressed person.
13. Hostility directed at the interviewer or at topics of the interview.
14. Frequent, abrupt and emphatic one word responses to your questions. (i.e., Yes! Never! Definitely! Absolutely!).

The Type B is considered to be a relaxed, easy going and patient person who works at a steady pace without feeling pressurised by lack of time (Dembroski, Weiss, Shields, Haynes & Feinleib 1978). The Type B has a slower, smoother style of talking and moving (Friedman & Rosenman, 1977). The Type B is often defined negatively (an absence of TABP) and is less well characterised (Harvey, 1988) when compared to the Type A.

Friedman and Rosenman (1977) claim that behaviourial intensities of TABP differ from person to person and a perceived environmental challenge is needed to catalyse the pattern into being. The two most obvious core behaviours of the pattern are "hurry sickness" and hostility. Type A's are usually competitive and control orientated. In severe cases (Type A1) subjects become so hostile that a free floating hostility characterises many of their actions. Friedman and Rosenman emphasise that TABP is a pattern and not a personality type or syndrome. They assume TABP to be distributed on a continuum with extreme Type A (A1) at the one end and extreme Type B (B4) on the other. Type X is considered to be an equal mix of TABP and TBBP. Most research does not deal with the Type X.

Despite the efforts of Friedman and Rosenman to define the pattern in concrete terms, there is debate about how the construct should be defined.

2.5.1. Debate over the Definition of TABP

Much like the concept of IQ, TABP has been conceptualised as quantitatively continuous (Friedman & Rosenman, 1977). As with the concept of IQ there are a number of difficulties: there is no universally agreed definition of the pattern- TABP is what Type A tests measure. Construct validity for the concept is lacking (Keltikangas-Jarvinen, 1987) and empirical data has in many ways overtaken conceptual analysis of TABP (Harvey, 1988). Booth-Kewley and Friedman (1987) note that

> It appears that every researcher working in the Type A domain has his or her own conceptualization and accompanying operational definition of the Type A construct (p. 334).

Consequently there is active debate over the definition of TABP. Some authors extend TABP beyond being a constellation of behaviours in the face of perceived threat and ally it to personality syndromes and types (eg authoritarian personality; Byrne, Reinhart and Heaven, 1989) or a "loosely related constellation of attitudes" (Byrne, 1987a, p.663). Even the idea of continuous distribution throughout the population has polarised researchers. Some, like Matthews (1982) insist on the continuous nature of TABP, while others like Glass (1977) argue that Type B's are qualitatively different to Type A's and point to the absence of a linear association between the intensity of TABP and CHD as evidence of typology. Recent research (e.g. Pedersen, Lichtenstein, Plomin, DeFaire, McClearn & Matthews, 1989) does little to clear the confusion. This needs to be borne in mind in the sections that follow.

2.5.2. The Genesis of the Type A Behaviour Pattern

The factors that contribute to the establishment of TABP in the individual may be inherited or acquired.

Matthews and Krantz (1976) found a modest genetic component for hard driving, competitive aspects of TABP in pairs of monozygotic and dizygotic twins. Pedersen, Lichtenstein, Plomin, DeFaire, McClearn and Matthews (1989) conducted a similar study in which they compared separately raised monozygotic (n=99 pairs) and dizygotic (n=229 pairs) twins with similar numbers of corresponding twins who had been reared together. They found that sharing the same environment was not important in the development of the pattern and that a significant genetic influence was

inferred. This suggests that the genotype for the pattern may become a phenotype notwithstanding environmental stressors and may develop in a linear fashion.

Matthews (1982) proposes a linear progression from TABP in adolescents to TABP in adulthood. Using the work of Steinberger (1986) as evidence against this proposal, Wright (1988), claims that TABP is actualised in late adolescence and early adulthood "as a compensation for essentially non-Type A phenomena that have existed earlier" (p.5). He sees the following as crucial in the development of TABP:

a) a high need for achievement as a condition of good self-esteem
b) early success due to active striving
c) exposure to timed and/or competitive activities

This triad (experienced in early adulthood/adolescence) is necessary but not sufficient to generate TABP. The TABP ontogeny is completed when the triad occurs in persons with low self-esteem who compensate with increased striving for achievement. These individuals often achieved well when young and subsequent failures actualise a multimodal approach to life i.e. trying to achieve as much as is possible in many areas of life. Other theories for the genesis of TABP include the following (based on Wright, 1988):

Social learning theories use the work of Bandura (1969) to describe how the pattern is a response to social modelling. The individual learns through reinforcement and punishment that an aggressive, time conscious way of life is well accepted by those administering rewards- especially in the work place.

Psychodynamic theories (based on the work of Freud, 1933) see TABP as a obsessive-compulsive defence against anxiety. This anxiety is defended against by increased activity so that few anxiety provoking thoughts are able to surface into consciousness.

Rogers' (1951) theory of self concept proposes that as parents and significant others evaluate the person, a self concept is forged. If this self concept is one of a "go getter" or "high achiever", the person acts in accordance with this concept.

If one sees TABP as a personality syndrome, the behaviour pattern may be the expression of underlying traits. Investigations by Irvine, Lyle and Allon (1982) reveal significant correlations between Neuroticism and TABP and Neuroticism and Speed and Impatience (TABP subscales) on the EPI. Furthermore TABP was associated with Dominance and Extrapunitiveness. The Extrapunitive scale was also associated with Speed and Impatience. However this could be because of common features that are shared by the two concepts. Fisher (1986) points out that the Neurotic Extrovert and the Type A have common features such as the speed with which they tackle tasks. Emara, El-Islam, Dagga, and Moussa (1986) also found an association between TABP and Neuroticism, but claim that the correlation between TABP and neuroticism is not surprising as both involve hyper-responsiveness to environmental events. The associations between TABP and elements of psychopathology may be distorted and Ivancevich and Matteson (1988) claim that many Type A components such as job involvement, emotional expressiveness and high energy

levels are healthy and socially desirable.

They argue that socially detrimental components such as hostility should be combated. It can be argued, however, that it is the excess of these behaviours in the Type A that is damaging not the presence of the behaviours per se. Many Type A behaviours in moderation can be seen as part of a healthy and balanced life. Jenkins (1988) states that Type A is a style of living characterized by extremes of competitiveness and hostility. It is these extremes of behaviour that are measured in assessment.

2.5.3 The Assessment of Type A Behaviour Pattern

The characteristics presented above (section 2.5) by Rosenman (1978) pertain to the structured interview (SI) which has been the primary TABP assessment device. Other methods to standardise the procedure have resulted in a number of self-administered questionnaires.

(a) Structured Interview

Early assessment of TABP was done on the basis of clinical judgement. This was considered to be an unreliable method of assessment. In an effort to standardise the criteria for clinical judgement, Rosenman, Friedman and their colleagues developed the Structured Interview (SI). In response to a number of questions, respondents are judged according to the content and process of their responses. Terseness of reply, volume, variability and speed of verbal style, facial and body movements all form part of the assessment (Jenkins et al., 1979).

(b) Jenkins Activity Survey.

Clinicians soon appreciated the need for a standardised assessment as the SI depends on the interviewer's skills and is too slow for use in large-scale studies (Jenkins, Rosenman, & Friedman, 1967). The SI is also time-consuming and costly: administration and interpretation of the SI cannot be conducted without previous training from an administrator versed in the procedure.

The Jenkins Activity Scale is an attempt to overcome these problems:

> The Jenkins Activity Survey was developed in an effort to duplicate the clinical assessment of Type A behavior by a more standard psychometric procedure and to make Type A assessment accessible both to individual practitioners and to researchers conducting large-scale industrial and epidemiological studies (Jenkins et al., 1979, p5).

The SI tends to measure the style of the respondent and the JAS tends to measure self-reported characteristics of the respondent (Rosenman, 1978). Jenkins et al. (1979) claim that Type A's are well known for their lack of insight concerning their own behaviour and often fake responses. For this reason, the development of the questionnaire was time-consuming and complicated. An initial questionnaire was developed in 1964. Validity of the questionnaire was assessed by selecting from an item pool (based on the SI) those items that were able to empirically discriminate those judged TABP or TBBP on the S.I. Items were optimally scaled according to the responses of half of a large sample of Type A men previously assessed by the SI. These weightings were retested on the remaining half of the

original sample and, with computational techniques, were transformed to have a mean of 0.0 and a standard deviation of 10. For all of those in the cross validation, there was 73% concordance. This became even higher when those scoring one standard deviation or more either side of the mean were compared: there was 100% agreement between the JAS and SI. The difference between the latter and former scores was due to the difficulty of both instruments in determining those close to the mean.

The JAS asks questions like "Have you given up any hobbies or leisure activities in the past 10 years primarily because of lack of time?". The Type A tends to answer 'yes' and the Type B 'no'. Different weightings for different answers culminate in a score. Type A's do not answer appropriately for all Type A indices and some Type B's have Type A attributes (Glass, 1977), but if the summation of the scores is above a certain level, then the person is judged to be Type A. Type B is a relative absence of Type A attributes reflected in a low score. Absence here refers to the absence of the same exaggeration of behaviours and not to the actual behaviours per se.

Despite the associations reported by many studies (see section 2.4.4. (b)), and in particular the Western Collaborative Group Study (Rosenman et al., 1975), this association does not establish a causal link between TABP and CHD. There are many difficulties with correlational research, some of which are dealt with in the next section.

2.5.4. Problems with the TABP-CHD Association

The problem with studies involving correlations or associations is that causation is not necessarily implied (Brand, 1978). Although this would be expected from a biopsychosocial perspective where a number of factors combine to produce an illness, it does cast doubt on the influence of TABP in the pathogenesis of CHD. Correlations are a statistically weak procedure and "confounding is inherent in correlational research ..." (Kantowitz & Roediger, 1984, p37). Any link between TABP and CHD may be spurious. Burish (1980) has also noted that, despite statistical significance, the association does not allow for confident prediction of CHD in the case of individuals. He adds that the connection between stress and TABP and their independent associations with CHD have still to be delineated. In addition, the mechanisms thought to mediate the CHD-TABP relationship are poorly understood.

Apart from the systematic difference between Type A's and Type B's that the TABP-CHD association could reflect, other factors that could explain the association are:

a. Another variable may produce CHD and TABP simultaneously, but separately. e.g. a Western life style may give rise to CHD and TABP independently (Cohen et al., 1978).

b. There may be a systematic difference between A and B types with respect to the traditional risk factors that each generates (Feinleib, Brand, Remington, & Zyzanski, 1978).

c. The association is a chance occurrence.

The exact relationship between CHD and TABP is hard to validate because ethical considerations preclude formal prospective experiments in which hypotheses could be tested. Formal experiments would have to purposely attempt to precipitate CHD in experimental groups, which would violate the principle of non-maleficence (Steere, 1984).

However, TABP remains the primary focus for debate and research in psychosomatic literature (Byrne, 1987a) and has survived a number of challenges concerning its usefulness as a coronary marker in the past (Feinleib et al., 1978). Yet the concept is still being challenged from a number of authors, for example Johnstone (1989) states:

> the topic of Type A behaviour and its links with coronary heart disease is currently in a state of some disarray and near crisis. (p.147)

Furthermore, much research subsequent to 1980 suggests little or no association between CHD and TABP in the USA and Britain (e.g. Mann & Brennan, 1987), although positive associations continue to be reported from Europe and other countries (Jenkins, 1988). Some authors have suggested that the concept represents a false trial and should be abandoned altogether (e.g. Ray, 1991).

Despite this, many studies have supported the claim that the person exhibiting TABP is twice as likely to suffer from CHD compared to those regarded as TBBP (Siegal et al., 1983, Feinleib et al., 1978).

This concludes the section on the first of Zubin and Steinhauer's (1981) factors in the vulnerability model (i.e. the degree of risk). The next section deals with Zubin and Steinhauer's second factor, namely the relationship between stress and illness (in particular CHD).

2.6. Life Stress and Illness

The role of stress in the development of illness, the maintenance of illness and the exacerbation of illness has been well documented. For example, stressful life events may precede the development of chronic diseases such as diabetes (Robinson & Fuller, 1985) and undesirable life events may also cause fluctuations in diabetics (Grant, Kyle, Teichman & Mendels 1974).

Selye (1956) is attributed as the first person to systematically describe responses to stress (Roskies, 1987). His view was that stress elicited an undifferentiated state of heightened arousal and hormonal change in response to noxious stimuli. These physiobiological changes were activated to overcome disturbances in the body's equilibrium cased by the noxious agent. He proposed the general adaption syndrome (GAS) to describe three core stages that constituted the syndrome. The first stage was referred to as the alarm reaction, followed by the stage of resistance and lastly the stage of exhaustion. The syndrome represents an attempt to adapt to the stressor and only the most extreme stressors result in death. Despite the adaptive nature of the response, Selye recognised that the attempt in itself may eventually cause more harm to the organism than the stimulus.

However, the stimuli Selye used in his studies were physical (e.g. cold, hunger) and not psychological. Selye also studied physiological reactions to stressors, and paid little attention to emotional and behavioural reactions. Nonetheless, Selye's studies linked environmental demands to physiological change and possible physical illness.

Building on this, Levi and Andersson (1975) argue that a change in a person's life circumstances can elicit a biological response that can become illness in a person who deals with the event in a maladaptive manner. Rahe, McLean and Arthur (1967) proposed that an individual's mental set determines how life changes will be interpreted. This perception in turn influences the psychological defenses the person uses and therefore the physiological level of arousal. Some defenses promote the normalisation of physiological arousal, while others increase and/or maintain the arousal. The way in which the individual manages these responses will determine whether illness could result. Those that are unaware of their arousal tend to develop symptoms which may become illness if not correctly managed.

Following a hypothesis that change in life events is stressful and that stress causes disease, Holmes and Rahe (1967) surmised that the incidence of disease would be proportional to the amount of life change in an individual's life. Their research supported this hypothesis and they found that medical patients reported more life changes than healthy people. A retrospective study with marines carried out by Rahe et al. (1967) supported this. Each

year marines filled out scales of life change. Prior to illness, there was a statistically significant rise in the amount of reported life changes. In a prospective study with a similar sample of marines, Rahe, Biersner, Ryman and Arthur (1972) claim an association between life events and visits to the dispensary.

Stress may also be experienced as the result of illness. Cohen and Lazarus (1973) see this stress in terms of threats such as the threat of dying, threats to one's future plans, threats to one's sense of self, and threats involving adjusting to a new way of life or setting (such as the hospital). Any cognitive or behavioral attempts to master the new internal and external demands on the person will strain personal resources. This maintains or exacerbates the person's physiological condition and illness.

Before dealing with life stress and coronary heart disease in particular, stress is defined in a manner consistent with the vulnerability model of Zubin and Steinhauer (1981).

2.6.1 A Definition Of Stress

The widespread use of the term "stress" and the many contexts in which the term is used with different meanings has led to difficulties in reaching a consistent definition. Fisher (1986) cautions against the tendency to see stress as a unitary phenomenon and Harvey (1988) notes that the term has been used to mean a stimulus, a response and a subjective experience. Lazarus, Coyne and Folkman (1982) state that stress is a

constellation of many acts and thoughts elicited by a complex set of demands rather than a single act. Stress may best be understood in terms of an individual's interaction with the environment (Harvey, 1988). Both the set of environmental demands and the person responding to those demands need to be incorporated into a definition.

In the past, definitions of stress have tended to be one-sided. Stress has been seen as an external demand pressurising an organism. This however allows no room for the subjective experience of stress e.g. different people find different levels of rock music stressful. On the other hand, one can see stress as the particular response of an individual. One person may find a roller-coaster ride stressful and another may find it enjoyable. On combining the two models, stress becomes the particular way in which a person's mental state determines how external or internal demands will be interpreted, or more simply the interaction between a person's coping mechanisms and the demands of the environment (Fisher, 1986). Fisher uses Cox's (1978) model to provide a more complete picture.

The model starts with an environmental demand (which may be internal or external; physical or psychological). The person perceives the demand and compares it to their perceived coping ability. If the perceived demand exceeds the perceived coping ability, stress is experienced. The model also includes how one deals with the resultant success or failure in dealing with the demand. There are feedback loops at each level of the process.

Fisher (1986) provides the following definition of stress:

> stress exists when conditions (internal or external) deemed unpleasant cannot be changed (p.12).

In other words stress occurs when personal resources are perceived to be insufficient to deal with environmental demands. Implicit in her definition is the concept of control over the environment. According to Fisher, when a person does have control but thinks that they do not, they are in a state of 'no control'. Actual control can never exceed perceived control. Fisher's definition of stress will be adopted in this study as her emphasis on the interactional nature of stress is consistent with the biopsychosocial model as elaborated below.

2.6.2. Stress from a Biopsychosocial Perspective

A systems perspective of stress emphasises that an individual at any one time is a product of external (environmental and cultural) and internal (biological and psychological) forces (Marsella, 1984). Stress is seen as the interaction of stressors and supports in the individual's world. A **stressor** here is seen as any process, event or object that elicits a state of change in the person's system. Consequently a disturbance in a seemingly distant area can exert an influence on the individual's internal functioning. Stress can occur at a number of levels. At the biological level stress can be experienced when the body cannot cope with the demands placed upon it, as is the case with malnutrition. Such reactions and behaviours that occur in the individual experiencing stress will be referred to as **stress responses.**

At a psychological level, the way in which cognitive state mediates stress is important as has been outlined in the previous section (e.g. perceptions of control). At a cultural and social level family network and cultural transition may disturb the system within which the individual lives. In order to deal with the different levels of stress, Marsella (1984) proposes the following framework to assess the stress responses of any particular individual:

1. Somatic: reproduction, repair, rest, nourishment
2. Sensory: information, acquisition and processing
3. Perceptual: information sorting, interpreting, judging
4. Motor: co-ordination, movement
5. Affective: arousal, emotion
6. Cognitive: symbolic behaviour, language, memory
7. Interpersonal: sociability, appearance
8. Self: purpose, meaning, coherence.

If a stressor is severe, the whole system may be disturbed, and act in unpredictable ways (Green, 1985). This disturbance may have pronounced effects on the physiological processes of a person, which in turn can increase the likelihood of disease (Kritz & Moos, 1974). Predicting the manner in which the person may behave in response to stress will be a function of which level of the person is most affected (Janoski, 1984).

Disease and treatment also affect the whole person at a number of levels (Harvey, 1988). Hence the interaction between the individual and the environment needs to be understood if intervention is to address the factors that precipitated illness. While this will be different for each individual, the kinds of

stress a person may experience have been collated under three broad headings that reflect intensity and duration. These are life dissatisfaction, chronic stress, and acute stress (Glass, 1977). The DSM III (American Psychiatric Association, 1980) reflects similar differences in its definition of post traumatic stress disorder and reactive psychosis definitions. Straker and the Sanctuary Counselling Team (1987) claim that 'continuous stress disorder' is another such clinical entity which is appropriate to refer to those living in constant states of abnormal stress.

Fried (1983) divides stress into similar categories of catastrophic stress (e.g. earthquakes), acute stress (e.g. the death of a loved one), and endemic stress (e.g. the continuous, daily stresses that most people are exposed to).

2.6.3. Stress and Coronary Heart Disease

Different kinds of stress may trigger CHD in vulnerable individuals in different ways. Glass (1977) claims that life dissatisfaction, chronic stress, and acute stress have all been independently implicated in the onset of CHD. Each will be dealt with here separately.

(a) Life Dissatisfaction and Coronary Heart Disease

The Sisyphus reaction (Bruhn, Paredes, Adsett & Wolf, 1974) is a term used to describe people who strive persistently with little success. They tend to approach life with a general sense

of dissatisfaction (Liljefors & Rahe, 1972). This phenomenon has been associated with those suffering CHD and is claimed to be a variable on which monozygotic twins discordant for CHD can be discriminated. Coronary patients report being more dissatisfied with their jobs when compared to controls (Theorell & Rahe, 1972) as well as more dissatisfied with life in general (Romo, Siltanen, Theorell & Rahe, 1974). It is as though the victim has been living close to their health/illness threshold as a way of life and no acute stress is needed to catalyse CHD. However, many studies in this area are retrospective and thus of doubtful validity.

(b) Chronic Stress and Coronary Heart Disease

More extreme is the recent association of major depression and cardiac events in people suffering from coronary artery disease. Carney, Rich, Freedland, Saini, Tevelde, Simeone and Clark (1988), while conceding their moderate sample size (52 subjects), see Major Depressive Disorder as an independent marker in the prediction of coronary events in those diagnosed with coronary angiography. Major depressive disorder was found in 9 of the 52 subjects and major depressive disorder was found to be the best predictor of cardiac events in the 12 months following assessment. Byrne's (1987a) review of initial studies (n = 5) suggests that the accumulation of stressful life events over time was associated with CHD. However, when compared to later studies (n = 4) this relationship was not as evident.

Byrne and Whyte (1980) suggest that it may not be the frequency of life events that is important, but the significance that CHD patients attribute to events. In comparing 120 CHD patients with 40 patients who were discharged from hospital soon after admission without diagnosis of MI or serious illness, they found that the two groups could not be discriminated on the basis of life event frequency or life change/impact scales. Only impact scales of emotional distress could significantly distinguish the two groups. Byrne and Whyte (1980) suggest that MI patients experience the year prior to MI as particularly distressing. This is consistent with Lazarus' (1966) contention that life events are only stressful to the extent to which individuals perceive them as such. In view of this, one can see how the Type A's perceptual set may facilitate the experience of stress in circumstances where others may not experience stress.

(c) Acute Stress and Coronary Heart Disease

It also appears that single events are enough to both weaken an individual and trigger illness at the same time. An apparent increase in stressful events in the 6 months prior to cardiac death has been used as support for a link between acute stress and CHD (Rahe & Lind, 1971). This appears to be the case especially where the life stress has included bereavement (Byrne, 1987a). Single traumatic events are also associated with the onset of other chronic illnesses such as diabetes (Robinson & Fuller, 1985). However, Tennant, Langeluddecke, Fulcher and Wilby (1988) offer contradictory evidence. After variables such as age were controlled for, they could find no association between

chronic or acute life events and CAD in their sample of nearly 500 adults.

Because of the complex nature of stress, the associations of stress and coronary heart disease are not easy to study. Some of the difficulties affecting research of this kind is discussed more fully below.

2.6.4. Problems With Stress-CHD Associations

Because CHD is a chronic disease that develops over a long period of time, linking life events to the onset of disease ignores the preceding chronic life situations. For this reason, the association between acute stress and CHD may be spurious. There may also be problems with using other forms of stress (life dissatisfaction, chronic stress) as explanations for the onset and development of illness. Lazarus (1966) warns that human maladaption cannot be simplistically attributed to stress. Stress generates a range of coping mechanisms and is as much a product of inept coping as it is of environmental demands.

Bruhn et al. (1974) also comment that psychosocial circumstances cannot be replicated. Studies that attempt to link disease and psychosocial events are therefore difficult to validate. The retrospective nature of many studies may also compromise validity as patients may reinterpret the significance of events in the light of their illness.

Some authors prefer to add these types of stress into a larger category simply termed chronic troubling emotions. Jenkins (1988) asserts that depression, hypochondriasis, anxiety, life dissatisfaction and interpersonal discord all have well documented associations with CHD. Krantz and Raisen (1988) add that these emotions are usually associated with the "soft" cardiac events (angina) rather than the "hard" events (myocardial infarction). Rather than triggering MI, they seem to be events that precipitate angina, a less serious disorder. More recent research has failed to reproduce associations of this "negative affectivity" and CHD (e.g. Watson & Pennebaker, 1989). Watson and Pennebaker (1989) claim that negative affectivity should be seen as a nuisance factor as it is correlated with both health and stress measures. Lastly the mechanisms that may mediate the stress-CHD association are not fully understood. However, research findings are suggesting mechanisms that may mediate stress and illness.

2.6.5. Stress and the Mechanisms of Illness

The body as an open system constantly maintains an import and export of matter and energy with the environment (von Bertalanffy, 1975). While it is acknowledged that equilibrium is never fully attained and the healthy organism moves towards a state of equilibrium (Lachenicht, 1988), it is useful to assume that equilibrium can be achieved by the healthy organism. Disease can be seen as the disruption of autoregulatory processes beyond a point where those same mechanisms can reinstate this balance. Without outside intervention, the state of disequilibrium worsens and illness becomes more extreme.

In order to attain equilibrium, the individual needs to deal with environmental demands. These demands can be regarded as internal (such as resisting a viral infection within the body) or external (such as dealing with the loss of a spouse). In order to be prepared for these demands, the body can anticipate an environmental demand (Sterling & Eyer, 1981). Part of this adaptation necessitates the brain overruling autoregulatory processes until the demand has passed or been dealt with. The central nervous system makes little differentiation between perceived threat and real threat (Wright, 1988). Hence, constant perceptions of demands mean a constant overruling of autoregulatory processes. This adaptive function can become maladaptive (Fisher, 1986) over time as the body experiences continued strain. As not all anticipated threats materialise into actual threats, it is the perception of threat rather than the reality of threat that is important in the maintenance of arousal.

With each perception of threat, internal mechanisms prepare the body for action via the sympathetic nervous system. Hormones that raise blood pressure and release sugars and fatty acids are secreted. With prolonged anticipation of threat, these processes start to alter normal bodily functions such as insulin regulation and renal activity (Fisher, 1986). A functional change in the bodily organs can result. In extreme cases the functional change may eventually become an anatomical change. This is the arousal pathology model that Fisher (1986) uses in discussing stress and coping, and one that will be used in discussing stress and TABP.

Before doing so, the last of Zubin and Steinhauer's (1981) 3 critical factors in the vulnerability model will be dealt with: moderating variables.

2.7. Moderating Variables and Coronary Heart Disease

Comparatively little research has been devoted to the variables that may moderate the onset of CHD in those at risk. There may be variables that moderate both the degree of vulnerability and the amount of stress needed to catalyse latent illness.

Marsella (1984) states that the experienced stress of a person is mediated by the stressor and the support available to that system. Like the demands in Fisher's model of stress, the supports that mediate how individuals will experience the demands exist on a number of levels. On a **biological level**, the health of the person will mediate the experience of the demand. Poor nutrition and fatigue are just two examples of biological factors that can increase the malignant potential of a stressor. On a **psychological level**, it has already been pointed out how the cognitive appraisal of events influences the experience of stress. Psychodynamic defence mechanisms such as projection are examples of how stressful experiences are diffused by distorting the world to be less threatening. On a **family level**, the support of family has been documented in mediating illness as has the availability of supports at a **social level** (Gottleib, 1981). With specific reference to individuals exhibiting TABP, there is evidence to suggest that Type A's exacerbate their coronary risk by avoiding social support (Hart, 1988). Blumenthal, Burg,

Barefoot, Williams, Haney and Zimet (1987) also consider social support as a specific moderator of long term CHD prognosis in men exhibiting TABP.

It appears that those with limited resources would be more at risk for CHD. However, CHD appears to be a disease of the first world and often affects those who are socially mobile (Cohen, 1978), and who have access to good support networks and medical services. As will be investigated later in this study, Cohen (1978) claims that coronary risk in non-Western populations increases when social mobility is accompanied by TABP.

The vulnerability model has been used to describe some of the biopsychosocial factors that influence the development of CHD. The next section attempts an integration between stress and TABP as there are elements common to both concepts.

2.8. Integration: The relationship between stress and TABP

It has been established that both stressful life events and TABP may be precursors to CHD (Byrne & Whyte, 1980; Rosenman et al., 1975). TABP and stressful life events are often studied separately, but the two factors may be related (Byrne, 1981). Byrne and Rosenman (1986) used a sample of nearly 600 males to reveal a significant, but modest association between TABP and reported levels of life stress. They conclude that stress and TABP form a confluence of coronary risk. Friedman and Rosenman (1977) imply a link between TABP and stressful life events when they claim that TABP is not a set of attributes existing within

an individual, but as a characteristic manner in which a person interacts with the environment. They see TABP as a way of responding with the environment that increases the likelihood of stress. The two are considered to be nonetheless separate. Jenkins (1988) claims that

> Type A behavior is clearly not the same as stress or distress because Type A is neither an unpleasant stimulus nor a reaction to discomfort. Rather it is a pattern of intense and sustained behavioral activation that is usually self-initiated (p.328).

It is, however, this self-initiated behaviour that increases the perception of the environment as full of demands (Glass, 1978). Like stress, TABP can be seen as a person-environment interaction that becomes more evident in circumstances where the environment is perceived as challenging. Matthews (1982) sees TABP as

> an overt behavior elicited from susceptible individuals by an appropriately challenging environment (p.293).

Fisher (1986) points out that perceived control may help in understanding this. If Type A's have a greater need to exert control over their environments, then they may perceive more events as threatening to their control and thus experience more stress (cf. Fisher's emphasis on the role of perceived control and stress).

2.8.1. TABP and the Need for Control

Glass (1977) maintains that the TABP individual has a need to be in control (i.e. have a sense of mastery over the environment [Fisher, 1986]). People in general prefer activities in which

they have some control over the process and outcome of events (Greer, Davidson & Gatchel, 1970), and it has been established that a sense of control is important to well being in daily events (Larson, 1989). However, for the TABP person, the need for a sense of control is excessive. Glass also claims that TABP people guard their control and exert greater effort to maintain control than do 'normals'. Glass (1978) sees this control as a mechanism to maintain a good self image in those showing TABP. He supports this view by showing how people displaying TABP work harder to succeed, suppress states that affect task performance (e.g. hunger and fatigue), have a rapid pacing of activities and become hostile if their efforts are frustrated (Glass, Synder & Hollis, 1974). If faced with an environment that they cannot control (i.e. their responses do not have any effect on the environment) they may either handicap themselves so that they may blame failure on other sources (Weidner, 1980), or they may become helpless or hyporesponsive. Glass emphasises the oscillation between hyperresponsive and hyporesponsive states as important in the onset of CHD.

Contrary to expectation, the hyporesponsive state does not enable the body to recuperate and repair damage from the hyperresponsive state. This state of "psychic exhaustion" mobilizes other processes that strain the body further. It is more akin to "suspended suspense" (Wright, 1988) in which the person takes up a helpless position. During this time noradrenaline levels rise (Glass, 1977) along with cholesterol levels, serum lipids and corticosteroids (often found in those who experience overwhelming

circumstances [Rahe, Rubin, Gunderson & Arthur, 1971]). It is not a state of rest, but one of distress that further burdens the sympathetic/parasympathetic nervous system. It appears then that this need for control and success plays a pivotal role in keeping the Type A aroused. It further appears that this need to achieve is insatiable.

Friedman and Ulmer (1985) refer to the need for control as the "insecurity of status". They use William James' (1890) formula to understand the phenomenon:

$$\text{Self-Esteem} = \frac{\text{Achievements}}{\text{Expectations}}$$

They add that

> regardless of the opinions of strangers or friends, if a person's expectations are in excess of his achievements, his self-esteem remains inadequate... it may in itself be largely responsible for other aspects of Type A behavior (p19).

This is especially so in view of the research presented by Burnham, Pennebaker and Glass (1973) which demonstrated that Type A's tend to deny or be unaware of their own limitations. They also tend to set unrealistic personal goals (Ward & Eisler, 1987) i.e. Expectations are greater than Achievements. To exacerbate this state, Type A's may down-play their achievements: Henley and Furnham (1989) show how people displaying TABP see themselves in a negative light and proceed to emphasise that James' equation may play an important role not only in understanding etiology, but in maintaining TABP.

Carver, Coleman and Glass (1976) claim that any achievement is likely to be followed by the allocation of more responsibility and demands to the achiever. This sets in motion the sequence again.

This need for control usually has certain emotions associated with it (e.g. hostility, Glass, 1978). In turn, these emotions have physiological correlates (such as increased blood pressure) that are believed to be implicated in the pathogenesis of CHD. The physiological reactivity of the Type A forms the basis of Fisher's (1986) arousal pathology model, and is discussed in the next section.

2.8.2. Physiological Reactivity of the Type A Person

TABP can be seen as a characteristic and maladaptive attempt at coping with environmental demands (Fisher, 1986). As the individual exhibiting TABP interacts with the environment, a number of levels and corresponding processes (behavioural, hormonal and metabolic) are initiated (Herd, 1978). As Herd points out, TABP is more than a score on the JAS and it is more than a particular speech manner exhibited in the SI. Each dimension of the pattern has physiological correlates that are believed to facilitate CHD as they in turn are adjusted, started and stopped according to the behavioural elements of the pattern. The anticipation of threat activates these processes on a daily basis. However, should these be activated for long periods of time in response to an overanticipation of threat, regulatory processes may be strained (cf. section 2.6.5. on

stress and the mechanisms of illness). This is exactly what appears to happen with those exhibiting TABP. The need for control facilitates emotional responses which strain regulatory processes in the body.

As TABP is an action-emotion complex (see Rosenman & Friedman's definition on page 19), the emotional component is important. Emotional arousal significantly increases systolic and diastolic blood pressures (James, Yee, Harsfiels, Blank & Pikering, 1986). TABP is associated with systolic blood pressure variability and peak systolic pressure ranges. In other words, Type A's appear to react more intensely to situations than do normals. This would be consistent with the Type A's perception of an event as more threatening than the Type B's perception of that same event. This may not be all situations, but merely those perceived as threatening to their sense of control (Siegal, Matthews & Leitch, 1983). There would appear to be many such situations. Newlin and Levenson's (1982) research suggests that this abnormally high arousal is expended over different situations in different settings.

Evidence suggests that different emotions mediate different kinds of physiological response. In contrast to the Schachter and Singer (1962) theory of emotion (which claims a single state of arousal for all emotions), James et al. (1986) claim that angry or anxious states correlate with higher blood pressures than those of happiness (in borderline hypertensives). Furthermore, happiness was inversely related to systolic blood pressure,

whereas anxiousness was positively associated with diastolic pressure. They suggest that emotional effects are greater in individuals with more labile blood pressures (i.e. Type A's - Siegal et al., 1983). Because they are more prone to exhibit hostility/anger than happiness (hostility is one of the 3 core features of Type A's), not only do Type A's forego opportunities to normalise their blood pressure by not being happy, but their blood pressure levels in response to anxiety or anger are higher when compared to a Type B experiencing the same emotions.

2.8.3. Somatization and TABP

Subtle metabolic processes influence this rise in blood pressure. During arousal, hormones promoting synthesis and anabolic processes (that need energy) are suppressed and hormones linked to catabolic activity (energy out) are released (Fisher, 1986). These include glycogen and, importantly, catecholamines. Cortisol acts against effects of insulin and blood sugars rise. Fatty acids are freed and cholesterol levels rise. When this arousal is excessive and damaging as is thought to be the case with the Type A, it is referred to as "somatization" (Fisher, 1986). Somatization is the overreacting of bodily processes and can be seen as a "tendency to experience and communicate somatic distress in response to psychosocial stress..." (Lipowski, 1988, p.1358).

The continued release of catecholamines together with the associated release of sugars, fatty acids and cholesterol are thought to engender CHD somatization.

2.8.3.1. Prerequisites for Somatization

Fisher (1986) states that the prerequisites for somatization are stress hormone (in this case catecholamine) secretion at (1) a high intensity (2) for a long time (3) on a frequent basis. This is exactly what Type A's undergo:

(1) they are aroused to greater intensities (Stoney, Langer, Sutterer & Gelling, 1987)
(2) over long periods of time (Bergman and Magnusson, 1986; Abbott, Peters & Vogel, 1988)
(3) on a frequent basis (Stoney et al., 1987).

Contrary to expectation, high catecholamine levels (usually measured in the urine) are not a good indication of those somatising specifically CHD. Firstly the TABP-CHD association is not a one-to-one relationship. TABP has been implicated in carotid arteriosclerosis, peripheral arterial disease, vascular migraine, accidents and suicides (Emara et al., 1986). A further moderating variable may be individual stress response. This depends not only on particular cognitive appraisal of events (Schachter & Singer, 1962), but also upon prevailing bodily response modes or preferred somatisation pathways of the individual. Malmo and Shagrass (1949) found that people tend to have a prevailing manner in which their bodies respond to stress. For example, patients from a high somatic muscle tension group and a cardiac disease group tended to respond to stress in their own respective mode i.e. either with muscle tension or raised heart activity. Hence disorders such as gastrointestinal ulcers and migraine headaches reflect different somatization paths (Fisher, 1986). Type A's tend to exhibit enhanced cardiovascular reactivity (Williams, Lane, Kuhn, Melosh, White & Schanberg,

1982), and so may have a particular somatization pathway that favours CHD. However, those exhibiting TABP may not solely respond to stress in a way that facilitates CHD. For example, Keltikangas-Jarvinen (1987) reports that patients suffering duodenal ulcers are more likely to be Type A when compared to patients with ulcerative colitis, irritable colon syndrome, gallstone disease and varicose veins. Others generalise this further and claim that TABP is a general disease-prone condition (e.g. Rime, Ucros, Bestgen & Jeanjean, 1989).

2.8.3.2. Somatization Pathways

The manner in which behaviour and state of mind affects arousal has been mentioned. In addition, the state of mind and behaviour of a person may influence different hormones that are secreted in response to perceived threat. Frankenhaeuser and Johansson (in Fisher, 1986) have correlated two of these hormones with different psychological states. Individuals that respond to environmental demands with a sense of control over the situation tend to secrete only catecholamines. Those that respond to the environmental demand from a position of helplessness (low perceived control), tend to secrete catecholamines as well as corticosteroids. When an individual is in a state of helplessness, the corticosteroid increase promotes the breakdown of the immune system (Fisher, 1986). Therefore, Type A's who are mostly helpless would be more susceptible to illness that involves immuno-logical breakdown (e.g. cancer). Type A's that tend to remain in control states may be more likely to suffer organ strain (e.g. MI). The particular somatization pathway that

the TABP engenders through increased catecholamine levels appears nonetheless to favour CHD.

Summary

Somatization is the process whereby damaging levels of arousal occur in the body. If this happens on a frequent basis for prolonged periods of time at a sufficiently intense level, the body may begin to malfunction. Individuals who deal with stress in a particular manner may expect to see a specific manifestation of somatization that targets and reflects specific bodily organ(s) used most in such a response. Looking closely at the hormones that are secreted in stressful conditions indicates which particular somatization pathway is being favoured. The CHD somatization pathway appears to have at its core the over secre tion of catecholamines. This oversecretion of catecholamines is evident in those exhibiting TABP.

The arousal pathology model assists in understanding the mechanisms that strain the heart. The possible manner in which this arousal pathology is facilitated by TABP has also been described. However, there is also evidence that TABP may facilitate other mechanisms and pathways that are also believed to contribute to the development of CHD. One of these pathways is via increased cholesterol levels.

2.8.3.3. TABP and Cholesterol

Burnham, Pennebaker and Glass (1975) studied time urgency in 33 students judged to be Type A and compared them to 29 Type B

students. They found that Type A's tend to judge the lapse of one minute sooner than Type B's. They also work at near maximum capacity irrespective of a deadline. Friedman (1978) claims that such a chronic sense of urgency in rats is associated with blood clotting time and an increase in serum cholesterol levels. Other important findings on cholesterol levels come from Rahe, Rubin, Gunderson and Arthur (1971). They researched a Navy underwater demolition team undergoing training and found that the increased level of serum cholesterol was consistently and frequently associated with moods and feelings of anger, fear, lethargy, and depression. These are the experiences expected from a Type A when hyperresponsive and hyporesponsive respectively. Rahe et al. report negative associations between levels of cholesterol and emotions of motivation and happiness. This mirrors the findings on blood pressure and 'positive' versus 'negative' emotional states from James et al. (1986) i.e. that states of happiness and motivation may reduce cholesterol and blood pressure levels. The 'negative' states used as examples above were associated with stress situations that were perceived as "no control" events.

It appears that TABP may facilitate the increase in cholesterol levels- a traditional coronary risk factor. Friedman and Ulmer (1985) state that

> there is no question about the fact that serum cholesterol level may vary directly with the intensity of Type A Behavior Pattern (p.11).

This suggests that TABP is implicated in both coronary somatization pathways and an increase in some traditional risk

factors. Clearly there is a degree of overlap between biochemical explanations of CHD development and somatization pathways. One can also conclude that the

> Type A's reactivity may be a common physiologic pathway through which other psychosocial risk factors operate (Krantz & Raisen, 1988, p.338).

2.8.3.4. Overlap of Biochemical and Somatization Explanations of Coronary Heart Disease

The somatisation pathway primarily concerns the role of catecholamines in CHD pathogenesis. In addition to this, lipid levels and cholesterol levels are known to influence CHD. The former argument claims that raised catecholamines strain the heart directly. Biochemical explanations argue that raised cholesterol/ serum lipid levels and raised levels of catecholamines are associated with coronary plaques which then cause occlusion of an artery resulting in CHD. The following model attempts to combine the two.

When the Type A experiences stress he meets the challenge with catabolic activity. This entails the secretion of catecholamines, cholesterol and serum lipids. The catecholamines directly stimulate the heart to beat faster and more forcefully. Blood pressure is increased (somatization pathway) for long periods of time. With this increase in blood pressure, there is an associated increase in the number of lesions that occur in the artery walls due to passing platelets. As there is an elevated level of lipids and cholesterol, the lesions accumulate large amounts of these substances to form plaques. Over time, the

artery lumens constrict as plaques build up. In addition, catecholamines (especially noradrenaline) are associated with the aggregation of thrombosites (Glass,1977) and platelets (Williams, 1978; Simpson, Olwine, Jenkins, Ramsey, Zyzanski, Thomas & Hames, 1975). The already constricted blood supply to the heart may be further slowed down by the aggregation of these blood cells in the presence of catecholamines. The strained heart struggles to pump blood thick with hormones and platelets through arteries that are being constricted by plaque accumulation. Eventually the heart's own oxygen supply is restricted and results in either temporary damage (angina), permanent damage (myocardial infarction) to the myocardium or sudden death.

In terms of the vulnerability model (Zubin & Steinhauer, 1981), the overlap of etiological models is evident. Environmental field forces interact with the social-psychological framework of the person (e.g. need for control, achievement striving) and are in turn translated into internal biological forces that strain the body.

While this summary depicts what may occur in the typical Type A individual, recent research (e.g. Dembroski, MacDougal, Williams, Haney & Blumenthal, 1985) suggests that not all Type A's are at risk for CHD. There may be parts of the pattern that are particularly pathogenic (as the following section indicates).

2.9. Type A Behaviour Pattern: The search for active ingredients

The evidence presented so far would indicate that the TABP concept has been and should continue to be a useful factor in the prediction of CHD. However, the concept as a global collection of behaviours has been challenged as it is believed that not all of the pattern's components (e.g. job involvement) are associated with the endpoint of CHD (Dembroski et al., 1978). Attention is now being focused on isolating the malignant or active ingredients of the pattern. Hostility/anger has been delineated as one potential active ingredient. The issue of hostility/anger has received much attention in recent publications (e.g.Dembroski, MacDougal, Costa & Grandits, 1989) and is dealt with below.

2.9.1. Hostility, TABP and CHD

There is confusion about the relationship between hostility and TABP. Friedman and Rosenman (1977) mention that a high hostility level alone is enough to classify one as a Type A candidate. Some recent journals have dealt with the independent CHD associations of hostility/anger and TABP quite separately (e.g, Barefoot, Dahlstroom, & Williams, 1983), while not attempting to clarify the relationship between the two. Other researchers (e.g Jenkins, Zyzanski & Rosenman, 1978) have called for a refining of the TABP concept as they believe that the pattern may be made up of several patterns, only some of which are coronary prone.

2.9.2. Research Findings

Roksies (1987) cites the Menningers (1936) as the first psychiatrists to study patients with CHD. Part of their description of these patients emphasised the aggressive tendencies of these patients. Williams, Haney, Lee, Yi-Hong Kong, Blumenthal and Whalen (1980) found that both hostility and TABP correlated with the degree of coronary occlusion in a sample of patients with differing severity of CAD. Their results were based on a cross-sectional study that included a restricted clinical population. This limitation was partly countered by Barefoot et al. (1983) who used a sample of 225 physicians who had filled out the MMPI as part of their medical training. The Ho (Hostility) scale (Cook & Medley, 1954) was used to determine hostility levels and these physicians were sent health questionnaires. Barefoot et al. found that hostility was significantly related to both CHD and total mortality in the 25 year follow-up period.

Dembroski et al. (1985) investigated which elements of the multidimensional A pattern related to CAD. After controlling for the major traditional risk factors (such as smoking and age), they found no relationship between CAD and global TABP. Only some Type A's were CAD prone. Only "Potential for Hostility" and "Anger" (two of the Structured Interview measures) were positively associated with disease severity (and hence a linear relationship between CHD and Anger/Hostility may exist in contrast to the TABP-CHD relationship). Dembroski et al. (1989) repeated these findings in a more recent study that matched 192 MI cases against 384 controls. The two groups were compared on

components of hostility and TABP. Again these researchers concluded that hostility as opposed to TABP was an independent risk factor for CHD.

A similar study conducted by Hecker, Chesney, Black and Frautschi (1988) compared 250 CHD cases and 500 healthy controls. Subjects were evaluated on global TABP and separate components of the pattern. After statistical analysis, they concluded that TABP consisted of a number of benign and pathogenic factors. The most pathogenic factor identified was hostility, which they claim was the most significant single risk factor when compared to the other TABP indices such as time consciousness and competitiveness.

These findings need to be moderated by the findings of Koskenvuo, Kaprio, Rose, Kesaniemi, Sarna, Heikkila and Lankinvainio (1988). They investigated the relationship between self-reported levels of hostility and CHD in a large sample (n = 3 750) of Finnish men. Follow-up after 3 years revealed that extreme hostility was a significant determinant of CHD in hypertensive men. The relationship was not evident among healthy men. Suarez and Williams (1989) also report qualifications of the hostility-CHD association. After studying hostility and cardiovascular arousal in 154 students, they claim that this relationship is mediated by situational characteristics such as interpersonal challenges. They conclude that people who rate high on the Ho scale and who have frequent interpersonal challenges are more at risk than those with high Ho scores who live in a less interpersonally challenging environment.

As assessment of the TABP incorporates levels of hostility as one of its scales (Jenkins, 1978), the validity of the pattern may be due to its ability to discriminate between individuals with high and low hostility levels.

The significance of hostility in TABP can be theoretically tied to the important role that goal achievement plays in the pattern (Friedman & Ulmer, 1985). Type A's tend to become more hostile than others if frustrated in their goals (Carver, Coleman & Glass, 1976).

The most important finding from the study conducted by Dembroski et al. (1985) was that "Potential for Hostility" was associated with the end points of CHD only if subjects were high on the "Anger-In" dimension. It was this particular style of hostility/anger that predisposed individuals to CHD.

Anger-In is not a new concept. In 1954 Funkenstein, King and Drolette hypothesised that the physical component of anger is mediated by the direction of expression i.e. anger towards oneself (Anger-In) or anger towards others (Anger-Out). They found that anxiety and Anger-In groups were physiologically similar (elevated systolic blood pressure and heart rate increases). Anger-Out, on the other hand, was associated with a larger diastolic blood pressure while heart rate remained the same. In addition to the direction of anger expression, failure to express anger may also be related to high blood pressure in hypertensive subjects (Matteson, in Fisher, 1986).

2.9.3. Anger and CHD

Dimsdale, Pierce, Schoenfield, Brown, Zaiman, and Graham (1986) found that across their closely matched subjects (n = 572), systolic blood pressure was significantly related to suppressed anger. Harburg, Erfurt, Hauenstein, Chape, Schull and Schork (1973) found that high stress subjects who suppressed their anger had higher blood pressures when compared to those who expressed their anger. This constitutes something of a hydraulic model that may explain why those that suppress anger may be more at risk for CHD. The anger mounts up and is expressed in hypertension (itself a risk factor for CHD) and elevated blood pressure. However, if one discharges hormones in a burst of expression (Anger-Out), the caustic nature of the hormones (due to the chronic nature of secretion) may be reduced and the body can institute recuperation processes (Wright, 1988).

Anger has a close relationship with hostility. Many publications (e.g. Booth-Kewley & Friedman, 1987) deal with the two as interchangeable. Saul (1976) sees hostility as a motivational force aimed at injuring or destroying some object. Anger can be seen as the expression of this force. It may be that anger generates a somatic response pattern that may be more damaging than response patterns generated by other emotions. There is some evidence that supports this contention. For example, Schwartz, Weinberger and Singer (1981) found that anger produced the greatest increases in cardiovascular reactivity (blood pressure and heart rate) in 32 subjects when compared to emotional states of happiness, sadness, and fear. The evidence presented by

Funkenstein et al. (1954) and Matteson (in Fisher, 1986) suggests that people who use Anger-Out styles may be less at risk compared to those who use Anger-In. The exact physiological pathways that account for this discrepancy are uncertain.

Before proceeding to studies that investigate TABP as a specifically Western phenomenon, the mechanisms that have been dealt with in this chapter are summarised.

Summary

The framework of stress manifesting at a number of levels proposed by Marsella (1984) (section 2.6.2.) can be used to summarise the Type A individual as follows:

Somatic: It has been shown that at a somatic level, the Type A does not allow time for effective regeneration of the body. The excessive catecholamine and cholesterol secretions constitute a particular somatic disturbance which predispose the individual to CHD.

Perceptual: The perception of the environment as full of demands is excessive in the Type A. Where others would tend not to see the environment as stressful, the Type A judges the situation as threatening and may not interpret events in a realistic manner. Unrealistic social comparison may further distort perceptual processes.

Motor: Type A's tend to have more rapid movement than Type B's, and rapid pacing over tasks, which in turn elicits certain

somatic correlates that are believed to facilitate coronary somatization pathways.

Affective: Type A's appear to react more intensely on an emotional level. The hostility of the Type A has been well documented as has the particularly high arousal levels Type A's tend to exhibit when angry/hostile. Again this has a close association with the somatic disturbances experienced by an individual at risk for CHD. The direction of anger expression may also influence somatic disturbance. Those using the Anger-in style may be more at risk for CHD.

Interpersonal: Competitiveness at an interpersonal level and constant self comparison to others may precipitate conflict between the Type A and others. This maintains the perceived need to be alert and on guard for environmental challenges.

Self: Friedman (in Rodgers, 1989) claims that Type A's tend to see the purpose of life as material i.e. the acquisition of material possessions. Friedman claims that Type A's lack a "spirituality" in the sense that they do not see the importance of life enriching activities like music and reading.

At a **family level**, there is evidence (e.g. Hart, 1988) to suggest that Type A's fail to utilise social supports and networks that mediate and resist CHD. After studying 113 coronary patients, Blumenthal et al. (1987) found a TABP/perceived social support interaction. Using the SI and Perceived Social Support Scale as part of their battery of tests administered to 113 coronary patients, they found that CAD was inversely related to the level

of social support for those exhibiting TABP. The relationship did not hold for Type B's. Blumenthal et al. conclude by claiming that social support moderates the health of Type A's.

At a **cultural** level, there is evidence to suggest that TABP is a specifically western phenomenon that can only be understood in the context of western values (Cohen et al., 1978). The manner in which the individual fits into this larger system is central to the biopsychosocial framework. This forms the theme for the next chapter on TABP as a western phenomenon.

2.10. Type A Behaviour Pattern as a Western Phenomenon

The three core factors of TABP are Factor H (hard driving and competitive), Factor J (job involvement) and Factor S (speed importance). These factors are often socially rewarded and

> In most industrial Western societies, competition, aggression and meeting performance or time deadlines are considered to be requirements to receive rewards (Ivancevich & Matteson, 1988, p.45).

Not only do the three core factors of TABP have much in common with the western lifestyle (Cohen, 1978), but Ivancevich and Matteson (1988) also claim that these social and cultural antecedents of TABP serve to maintain and reinforce the pattern. However, it still needs to be determined whether TABP constitutes a high coronary risk independent of the prevailing cultural conditions or whether it is merely a reflection of the typical life style of middle-aged American men (Cohen et al., 1978). The immigration of Eastern cultures to the West provides a population that reflects a change in cultural setting. Japanese immigrants

to America provide researchers with one such opportunity to study the relationship between culture and TABP.

Cohen (1978) studied Japanese Americans, who despite valuing efficiency, tended to place highest value on the wellbeing of the group rather than the individual, and tended to value co-operation above competition. Her findings indicate a culture-free core of items which constitute CHD risk irrespective of cultural setting. There is a nonsignificant but modest association between American Japanese who exhibit TABP (JAS) and CHD (Cohen et al., 1978). However, when TABP was coupled with those who were culturally mobile, the risk was similar to that of white American males i.e. those who were both Type A and culturally mobile were two to three times as likely to develop CHD when compared to those who had only one of the two factors. Friedman and Ulmer (1985) similarly note that the Japanese female's chances of CHD have quadrupled since the second World War as industrialisation and cultural mobility have increased. But in those communities where industrialisation has not been as pronounced or rapid, the predictive validity of TABP is reduced. This appears to be the case with other cross-cultural studies.

2.10.1. Cross Cultural Studies of Type A Behaviour Pattern

Emara et al. (1986) studied TABP in 60 Arab M.I. patients and compared this group to a control of 60 Arab non-CHD patients. They found no difference between the two groups and suggest that TABP may be a set of behaviours that is culture-bound. They cite Dressler (1981) in claiming positive associations of CHD and TABP in a number of countries: Belgium, Finland, Sweden, the Nether-

lands and Greece. They add that no studies have been carried out in cultures that do not reward the core TABP characteristics as points of strength. Their results indicate no association between TABP and CHD in their Arab patients. They add that mechanisms that mediate CHD via high catecholamine levels in individuals living in industrial settings may not be peculiar to TABP. The same mechanisms may be actualised in different ways in noncompetitive societies.

2.10.2. Coronary Heart Disease in the South African Indian Population

Investigations of the coronary risks among Indians in South Africa have isolated a number of standard risk factors that are associated with CHD. However, some studies (e.g. Sewdarsen et al., 1987; Walker, 1980) have investigated these risk factors in the absence of control groups. Therefore it is not known how well these risk factors are able to discriminate between those who incur CHD and those that remain free of the disease. For example, it may appear that smoking identifies those likely to develop CHD because 79% of coronary patients smoke. But if comparison to a hypothetical control group of "normals" reveals that 79% of this control group smoke, smoking is not able to distinguish between the two groups. In this case smoking remains a coronary risk factor, but its usefulness is compromised because it is unable to discriminate between those who are likely to develop CHD and those who will not.

As the significance of different coronary risk factors differs from one culture to another (Booth-Kewley & Friedman, 1987), one can expect that factors able to identify those likely to incur CHD in other cultures will not necessarily be able to identify Indian men at risk for CHD. Sewdarsen et al. (1987) did not include in their study non-standard risks such as stress and TABP. Therefore, it is not evident which standard coronary risk factors are significant for Indian men, or whether nonstandard coronary risk factors are significant in this population group.

Once these issues are clarified clinicians will be able to identify areas in which intervention will be beneficial. In western cultures intervention on various levels has proved successful (Bundy, 1989). Some forms of cardiac rehabilitation are increasingly dealing with behavioral risks such as TABP, and are described below.

2.11. Cardiac Rehabilitation

Blumenthal et al. (1988) report that most patients do not die in the acute phase of illness immediately after MI. Therefore, there are large numbers of patients that require some form of rehabilitation.

From a biopsychosocial perspective, rehabilitation should address as many levels of the person's functioning as possible. Historically, the physiological level has been concentrated on to the detriment of other levels. Rehabilitation prior to 1950 usually took the form of bed rest and reduced physical and

emotional activity. Levine and Lown (in Bundy, 1989) described the disadvantages of this approach which included muscle wastage and low morale. When subsequent studies (e.g. Hellerstein, 1968) demonstrated how an increase in physical exercise was of psychological and physiological benefit to cardiac patients without increasing morbidity or mortality rates, physical exercise rehabilitation programmes became well established.

Cardiac rehabilitation has been applied to primary (prevention) and secondary (immediately after MI and recovery) phases of intervention. These will be dealt with separately, although it is acknowledged that the phases are continuous.

2.11.1 Primary Intervention and Coronary Heart Disease

Strategies for CHD prevention have relied heavily on addressing risk factors such as TABP, smoking, diet and lack of exercise. These efforts have mostly taken the form of health education (Bundy, 1989) and skills training (Jeffery, 1988) and range from self help books (e.g. Friedman & Ulmer, 1985) to community awareness programmes (e.g. Perry, Klepp & Shultz, 1988). While CHD morbidity and mortality is not a primary concern for adolescents, the behaviours that contribute to CHD may become established at a young age. Primary intervention with adolescents is a relatively new area of intervention and few studies have investigated the usefulness of this approach. Ongoing research suggests that such programmes have been successful in modifying lifestyles that may facilitate the development of CHD (Perry et al., 1988). Among elder population groups, occupational health

promotion programmes, like programmes with the youth, need more systematic study, but have revealed promising results (e.g. Glasgow & Terborg, 1988).

2.11.2. Secondary Intervention with Cardiac Patients

This phase is most likely when the CHD patient is hospitalised and is still in the acute stage of the illness. Cassem, Hacket and Wishnie (in Bundy, 1989), in one of the few systematic investigations into coronary care of hospitalised patients, report that of 441 patients that were consecutively admitted, 32.7% were referred to a psychiatrist for anxiety, depression or behavioral modification. There is evidence that this hospitalisation has different meanings for different people (Byrne, 1987b), but almost all face stress and a sense of role loss (Finlayson & McEwan, 1977). These referred patients underwent a programme which included tranquilisers/sedatives, hypnosis, cognitive stress coping methods, environmental change (e.g. introducing radios) and confrontation. Cassem et al. report a significant decline in the expected mortality rate of their sample when compared to mortality rates in the CHD population. There was, however, no control group used in this study.

A study that did include a control group was that of Gruen (1975). Seventy patients with confirmed CHD were randomised into two groups, one of which was given psychotherapy. Gruen reports that the counselling elicited feelings of hope that enabled patients to deal constructively with their anxieties and enabled them to be discharged sooner than the control patients who had

no form of counselling. The treatment sample also spent less days in intensive care, were lower on self reported anxiety and fear and returned to normal activity quicker than controls. The results presented by Oldberg (1985) support Gruen's findings and reinforce the importance of educational and counselling intervention.

Blumenthal et al. (1988) report that betablockers and anticoagulants, coronary artery bypass grafting and new surgical treatments (such as the widening of compromised coronary arteries by inflating a balloon placed in the artery and then removing the balloon) have been used successfully to assist MI patients rehabilitate. Behavioral treatments centre on aerobic exercising and modification of behaviours such as TABP.

2.11.3. TABP and Intervention

Abbott et al. (1988) studied TABP in men and women after an MI. After an MI, men's scores (assessed by JAS and videotaped SI) dropped spontaneously after 1 to 3 months on the 3 core TABP indices. For women the hard driving and competitive components of TABP dropped. However, after a year the patterns returned to the same level exhibited before the MI. This period before the resumption of premorbid TABP may be extended by counselling. Powell, Friedman, Thoresen, Gill and Ulmer (1984) found that counselling reduced TABP in postinfarction patients after a period of 24 months. Not only did the pattern change but this experimental group had a lower rate of cardiovascular recurrence than did the comparison group. There was no difference, however,

between the groups in total cholesterol or resting blood pressure.

Friedman and Ulmer (1985) are optimistic and offer a self monitored approach that looks at altering the pattern by alleviating self destructive behaviours such as time urgency and hostility. In a similar approach, Roskies (1987) developed a programme that uses relaxation principles (e.g. awareness of variation in bodily tension) to assist Type A's manage their stress levels. Nunes, Frank and Kornfeld (1987) claim that a combination of treatments may be the most effective intervention. After a meta-analysis of 10 studies that investigated CHD-TABP and intervention, they report that all of the following have had documented success: education programmes, behaviour modification, relaxation training, imagery, cognitive therapy, emotional support, and psychodynamic interpretation.

They isolate education about TABP and cognitive therapy as two of the more powerful interventions for reducing TABP. However, they also note that these methods may prepare subjects to lower their own self-report measures of TABP. They claim that intervention aimed at altering the TABP in turn improves CHD prognosis.

Blumenthal, Williams, Williams and Wallace (1980) studied the effect of exercise on 46 healthy, middle-aged men. After subjects had been divided into either A or B types according to the JAS, subjects participated in a 10-week exercise programme. At the end

of the programme, TABP pre and post intervention comparison revealed that JAS scores were reduced and other risk factors such as blood pressure were also reduced. In contrast, Type B's showed no change. However, this study made no use of a control group, so results need to be viewed with caution. Schmieder, Friedrich, Neus, Rudel and von Eiff (1984) claim that TABP can be influenced by beta blockers. They studied 19 male subjects that were divided into two groups that were matched for TABP score (SI) and cardiovascular reactivity. One group were administered beta blockers while the other group was given diuretics. It was found that speech characteristics of the TABP (eg loudness, speed) were reduced, heart rate reduced and blood pressure became less labile. They conclude that beta blockers may be a prophylactic treatment for those exhibiting the pattern.

Throughout this study, research findings have been subject to methodological problems. Before dealing with the investigation section of this study, some of these research constraints will be mentioned.

2.12. Methodological Problems with Research

2.12.1. The definition of TABP

As mentioned in the initial part of this study, there is ongoing debate as to what TABP is. Construct validity is low due to the conceptual confusion surrounding the term (Booth-Kewley & Friedman, 1987) and the psychological dimensions that give rise to the set of behaviours known as Type A have not been identified

(Matthews, 1982). In addition, the relationship between stress, TABP and CHD is confusing (Burish, 1980).

2.12.2. The Definition of Heart Disease

The definition of heart disease may also be misleading. Booth-Kewley and Friedman (1987) argue that the term "heart disease" refers to a collection of related diseases that vary in their endpoints. They claim that different studies have used different endpoints for CHD (e.g. atherosclerosis, angina, MI). The forming of fatty plaques in arteries that leads to angina or MI can occur in arteries throughout the body. Arteries that lead to other bodily organs can become blocked in a similar manner with corresponding implications. Why certain psychological factors would affect specifically the arteries of the heart is uncertain.

2.12.3. Problems with the JAS

Many of the difficulties with self-report measurements can be generalised to the measurement of TABP with the JAS. For example, respondents may be influenced by social desirability. They may distort their perceptions in a way that supports the prevailing values of a particular society (Graham & Lilly, 1984). Consequently, the person living in a western culture may score items such as time consciousness and competitiveness inaccurately high. Byrne, Rosenman, Schiller and Chesney (1985) claim that self-report measures of TABP only measure elements of the pattern. Burish (1980) notes that the JAS has been standardised on middle-aged, middle-class American, Caucasian men. Validity of the JAS is compromised when the instrument is used on samples that do not come from the standardisation population.

2.12.4. TABP and the Coronary-Prone Behaviour Pattern.

Harvey (1988) distinguishes between the use of TABP and the term "coronary-prone behaviour pattern". Although some authors consider the two to be synonymous (e.g. Blumenthal et al., 1980; Burnham et al., 1975; Carver et al., 1976; Glass et al., 1974; Hecker et al., 1988), Harvey claims that before TABP can be considered to be a coronary-prone behaviour pattern it needs to be refined so that it can account for the Type B's that suffer CHD and the Type A's that remain healthy. He further comments that the active ingredients of the pattern have not been isolated and that interventions that attempt to change the entire pattern may be misfocused.

2.12.5. Problems with Research Design

Throughout this study it has been noted that many studies are flawed because they do not make use of control groups. The retrospective nature of many studies may also lead to spurious relationships as behaviours and stresses may be reinterpreted in the face of present illness. Nunes et al. (1987) reviewed 10 studies of CHD treatment intervention and concluded that one of the major flaws facing such studies was that subjects self-selected themselves into treatment modalities/control groups. They argue that the validity of such methods is questionable as subjects may anticipate certain behaviours.

Despite these restrictions, further investigation of coronary risk factors is warranted. Findings may serve to screen risk factors that are of particular importance in identifying South

African Indians at risk for coronary heart disease. The present study attempts to address some of these methodological problems as discussed below.

2.12.6. Justification of Present Study

While, CHD has been on the decrease in South Africa, CHD among Indian males has not decreased at the same rate as CHD in white males (Taylor, Epstein, Disler, Wittaker, Rip, Derry, Sayed, Bourne, & Klopper, 1987). Taylor et al. (1987) state that

> Preventable major risk factors may be coming under control in whites to a greater extent than coloureds and Asians (p.698).

While this clearly points to the need for intervention programmes that attempt to reduce CHD (Balfe, Steinberg & Kustner, 1988), the level at which intervention takes place is critical (Janoski, 1984). Therefore, intervention needs to address those factors that appear to significantly identify those at risk for CHD. Walker and Vorster (1988) claim that lack of knowledge about the weightings of different risk factors is one of the major issues impeding effective intervention in South Africa. To investigate these different weightings, the significance of nonstandard factors such as stress and TABP needs to be compared to the significance of standard risk factors such as diabetes and hypertension.

To ensure that this comparison takes into account the limitations discussed above, the endpoint for CHD in this study was taken to be MI. Those with other endpoints (e.g. angina pectoris) for the

disease were excluded. Despite the reservations expressed about the JAS, it was used as it remains a widely accepted measure of TABP and allows comparison with other studies (Byrne, Reinhart & Heaven, 1989). The research design allowed comparison with a non-coronary hospitalised control group as well as a control group of normals. Hence the specificity of risk factors could be investigated.

CHAPTER 3: THE INVESTIGATION

3.1. Introduction

This chapter outlines the hypotheses that directed this study. It also covers the choice and description of the samples used, method of research and tests employed in the investigation.

As mentioned at the beginning of this study, the central aim of this study is to investigate whether the CHD-TABP relationship is evident among the Indian population. In addition, the possible relationship between CHD and the non-standard risk factors of stress and anger are also investigated. Lastly, the study aims to suggest which risk factors (standard and non-standard) can significantly discriminate between those at risk for CHD and those relatively free of such a risk.

3.2. Hypotheses

The specific hypotheses that were investigated in this study are set out below.

Based on bedside non-standardised interviews at ward rounds and informal discussion among team members, it was believed that CHD patients at Northdale Hospital did not typify the Type A pattern. They appeared to be relaxed people who tended to be polite and unaggressive, rather than time-conscious, aggressive Type A candidates. Therefore, the first hypothesis reads as follows:

Hypothesis 1: Based on informal observations of CHD patients, TABP does not have a strong relationship with CHD among the Indian population in South Africa.

From the literature review, the following hypotheses are generated with regard to standard and nonstandard coronary risk factors.:

Hypothesis 2: CHD patients differ from other patients and from "normals" with respect to the number of associated standard coronary risk factors they exhibit. It is assumed that each of the coronary risk factors established in western studies will be able to discriminate the experimental group from the two control groups.

Hypothesis 3: Certain stresses predispose vulnerable individuals to CHD and it is assumed that the there is an association between level of reported stress and CHD.

Hypothesis 4: There may be a relationship between dimensions of anger and CHD. As with studies conducted in the West, it is assumed that the greater the level of Suppressed Anger, the greater the likelihood for CHD.

Hypothesis 5: Certain risk factors will be able to discriminate coronary patients from control groups better than others. It is assumed than the standard risk factors will represent the more powerful coronary indicators.

Hypothesis 6: There may be certain clusters of risk factors that are able to powerfully differentiate between those with CHD (at risk for CHD) and those not at risk for CHD.

3.3. Sample

Shekelle (1978) points out that studies in this area should have hospitalised controls as well as community/neighbourhood controls. This is important in this study as the investigation

on stress and illness could be contaminated if stress scores are high in ill people as a function of illness. Three groups of subjects therefore participated in this study in accordance with Shekelle's recommendation.

a) The Experimental Group

The Experimental Group consisted of 25 Indian male patients from Northdale Hospital in Pietermartizburg with confirmed MI. All those approached in the Experimental Group agreed to participate in the study. The results of 3 subjects (10.7% of those approached) were excluded from the study. One subject had brain damage and was unable to complete the interview due to fatigue, and 2 subjects were excluded as their chest pain was not later confirmed as MI. Subjects were selected on a random basis over an 18-month period.

b) The Medical Control Group

The first of the Control Groups comprised 25 non-coronary patients from the same medical ward at Northdale Hospital. This group had the following distribution of illness:

Table 1: **Distribution of Illness among Medical Control**

Disorder	N	%
Asthma	7	28
Psychosomatic pain	3	12
Depression	4	16
Diabetes	2	8
Chest pain	2	8
Pneumonia	1	4
Gout	1	4
Stroke	1	4
Physical Injury	1	4
Vomiting	1	4
Thyroid disorder	1	4
Blood disorder	1	4
TOTAL	25	100

All of the non-coronary hospitalised patients in the Medical Control Group agreed to participate in the study, and all data was complete. Subjects were selected on a random basis over a 6 month period.

c) The Normal Control Group

The second Control Group consisted of 25 non-hospitalised, apparently healthy subjects that were chosen on a random basis. Three subjects (10.7% of those approached) from the non-hospitalised (normal) control (Group 3) declined to participate in the study. All data from this group was complete.

Groups were matched on sex (male), ethnic group, age, marital status and employment. As the study's primary focus was CHD in Indian Males, 100 % of the 75 subjects were both Indian and male. The other controlled factors were distributed among the sample as follows (see table 2):

Table 2: **Distribution of Groups according to Age**

AGE	MI		Med		Norm	
(Years)	n	%	n	%	n	%
20-25	2	8	2	8	0	0
26-30	0	0	2	8	2	8
31-35	0	0	3	12	6	24
36-40	6	24	4	16	7	28
41-45	4	16	0	0	2	8
46-50	4	16	1	4	2	8
51-55	6	24	7	28	1	4
56-60	3	12	1	4	4	16
61-65	0	0	4	16	0	0
66-70	0	0	1	4	1	4
TOTALS	25	100	25	100	25	100
Mean Age	45.4		45.8		42.5	

Where MI = Experimental group, Med = Medical control and Norm = Normal control.

Most research in the area of TABP is with middle-aged men. This sample of coronary patients at Northdale hospital yields results that can be compared to research conducted in America and Britain. Research in South Africa has tended to investigate CHD in younger men e.g. Sewdarsen et al. (1987) investigated coronary risks in a sample of men with a mean age of 36 years and a range from 21 to 40 years of age. According to Mitha (1980), the incidence of CHD peaks in men between 40 and 55 years of age.

Table 3: **Distribution of Experimental and Control Groups by Marital Status.**

Marital	MI		Med		Norm	
Status	n	%	n	%	n	%
Married	22	88	21	84	22	88
Single	1	4	3	12	2	8
Divorced	1	4	0	0	0	0
Widowed	1	4	1	4	1	4
TOTALS	25	100	25	100	25	100

There is evidence to suggest that marital status may play a salutogenic role in the long term outcome of CHD. Berkson's (in Fisher, 1986) analysis of mortality rates for different causes of death suggests that divorced and single individuals exhibit increased vulnerability for CHD and other diseases (e.g. ulcer, cancer, pneumonia). The study conducted by Sewdarsen et al. did not control for marital status. The present study attempted to match subjects for marital status (see Table 3).

Subjects were matched for employment status (see Table 4) as unemployment is experienced as stressful (Strumpfer, 1986), and could influence results if not controlled.

Table 4: **Distribution of Employment Status**

	MI		Med		Norm	
Status	n	%	n	%	n	%
Employed	17	68	14	56	17	68
Unemployed	6	24	6	24	6	24
Retired	1	4	4	16	1	4
Grant	1	4	1	4	1	4
TOTALS	25	100	25	100	25	100

3.4. Procedure

Individuals from the three groups were approached and told that the researcher was studying health issues in the Indian community. Following a short explanation that this involved an interview that lasted approximately 20 minutes and a guarantee of confidentiality, subjects were asked to participate in the study. All subjects gave consent to their inclusion in the study. Interviews were carried out in relative privacy. Hospitalised subjects were interviewed behind drawn curtains and non-hospitalised subjects were interviewed away from earshot of others (e.g. back of shops).

This study made use of various self-report questionnaires covering a range of information. However, as some subjects were illiterate, it was decided that for the sake of consistency, all subjects would be interviewed. Subjects were interviewed by the same interviewer according to a questionnaire sheet. Each interview ranged from 20 to 40 minutes and covered the following information (see Appendix A):

Biographical data: Age
Marital status
Educational level
Occupation

CHD risk factors:
- Family history of CHD
- Family history of diabetes
- Family history of hypertension
- Personal history of diabetes
- Personal history of hypertension
- Smoking habits
- Level of physical activity
- Obesity
- Recent stressful events scale
- Type A scale
- Anger scale

3.5. Instruments Used

The questionnaire made use of a data sheet that covered simple questions (e.g. Do you have problems with blood pressure? [have you ever taken pills for your blood pressure?]). The purpose of this sheet was to gather biographical data and information on coronary risks and was based on the questionnaire used by the Kaunus Rotterdam Intervention Study (Glasunov, Dowd, Baubniene, Grabauskas, Sturmans & Schuurman, 1981). Questions covered aspects that respondents were likely to know about themselves and did not cover aspects that needed measurement from a medical person (e.g. cholesterol levels). The interview also made use of three scales:

1. The Jenkins Activity Survey (Jenkins et al., 1979) was used to measure TABP.

2. The Life Experiences Survey (Sarason, Johnson & Siegal, 1978) was used to measure reported stress.

3. The Survey of Affective Stress (Goldstein, Edelberg, Meyer, & Davis, 1988) was used to measure experienced anger, expressed anger and family patterns of expressing anger.

These instruments are discussed more fully overleaf.

3.5.1. The Jenkins Activity Survey

Form N of the JAS was used in this study (see Appendix B) as it is a less time-consuming device than other forms of the JAS. Form N represents an attempt to construct a scale that was shorter (yet psychometrically acceptable) than the original 21 item B and C version of the JAS (Jenkins, in Jenkins et al., 1979). The scale can be used on people that are unemployed as all the items that pertain only to working people were eliminated from the longer forms of the scale.

Form N of the JAS consists of 13 statements that each have a range of answers e.g.

8. How was your temper when you were younger?

1. Fiery and hard to control
2. Strong, but controllable
3. No problem
4. I almost never got angry.

Respondents choose the answer that they believe is most true of themselves. The areas measured cover issues like competitiveness, time consciousness, general level of activity and approach to life.

3.5.1.1. Administration and Scoring

The scale is usually administered as a self-report scale. As some of the subjects in this study were illiterate, and others needed clarification on some of the terms used (e.g. "precise"), subjects chose answers of items that were read out to them by the researcher. Each option chosen in response to a statement is converted into a scaled score by adding specific item weights and

then entered into a formula. This formula ensures that the scaled score is comparable to published data on the JAS. The final score may be positive or negative. Scores above +5 are considered to represent Type A behaviour, while scores less than -5 are considered to be Type B. Scores between +5 and -5 represent mixed types that have both Type A and Type B characteristics.

3.5.1.2. Validity of the JAS

The validity of the JAS has been assessed in a number of ways. On being cross-validated with the SI, Jenkins et al. (1979) reports a 73% concordance rate. There was a 90% correspondence on the SI for those one standard deviation from the mean on the JAS. This may be due the difficulty both instruments have in distinguishing those close to the mean. The JAS has also been translated into other languages and has managed to retain replications of earlier studies, thus giving further support to its "robustness". These studies have on the whole been retrospective, and as such are prone to the complications that accompany such designs e.g. bias. To eliminate this there must be some sort of predictive validity.

Prospective findings are similar to those of retrospective findings. The definitive study in this area comes from the Western Collaborative Group Study. Out of the 2 750 men in the study, 120 future clinical CHD cases were distinguished from those that remained healthy (Jenkins et al., 1974). Those in the top third Type A scores were 1.7 times as likely to get CHD when compared to those in the lower third of the sample.

An increased risk of reinfarction has been associated with JAS score (Jenkins et al., 1979). Scores on the JAS were better able to distinguish recurrent MI's from single-event CHD than it was between single-event CHD and CHD-free groups.

3.5.1.3. Reliability of the JAS

The manual for the JAS (Jenkins et al., 1979) claims that the JAS compares "favorably with reliabilities of other standardized psychological tests" (p.13). The manual reports a four year test-retest coefficient of .64. This becomes .65-.82 when tested over a four-to-six month time period. They conclude:

> The consistently high correlations between each of the scores derived from the 1965 edition and its corresponding but independently derived scores from the 1966 and 1969 editions not only indicate the mathematical stability of the derived scores and the relative equivalence of the several editions of the JAS, but also imply that the traits measured by the JAS are stable in individuals at least over a four year interval. This agrees with expectations arising both from theory and from clinical observations made by physicians (p.13).

3.5.2. Life Experiences Survey

The Life Experiences Survey (L.E.S.) was developed by Sarason et al. in 1978 (see Appendix 1). The L.E.S. was developed in response to an earlier flawed instrument. Prior to this, the Schedule of Recent Experiences (S.R.E.) of Holmes and Rahe (1967) was most popular in assessing life changes. This was based upon assumptions that, according to Sarason et al.(1978), were inaccurate both from a methodological and theoretical point of view. The S.R.E. sees change of events per se as being stressful, no matter how desirable the change experienced. This schedule

then sums all the scores attributed to the recent experiences and uses this as a gauge of stress. Sarason et al. (1978) query the logic of adding negative experiences and positive experiences together. Undesirable events such as the death of a close family member may have very different effects to events experienced as positive. Room also needs to be made for the subjective experience of similar events. For example, pregnancy may be stressful for one woman and enjoyable for another.

Sarason et al. take care to individualise their rating scale so that there is room for differing perceptions of the same event, whereas the S.R.E. assumes single common stress levels for certain events. An attempt to eradicate ambiguities in the S.R.E. is made by adjusting some of the S.R.E. items to be more specific. For example, "Major change in financial status" has the options of "a lot better off or a lot worse off". The following three factors are seen to be of major importance:-

i) the events listed should be experienced relatively frequently by the population.

ii) respondents themselves should decide on the (un)desirability of events.

iii) ratings should allow for individualized impacts of events.

3.5.2.1. Administration and Scoring of the L.E.S.

The LES is a self-administered scale in which respondents indicate from a list events that have occurred in the past year of their life. The impact of these events is also indicated by giving the event a corresponding score which ranges from -3 (very

negative) to +3 (very positive). The different scores can be seen separately as either the sum of positive scores (positive change score) or the sum of negative scores (negative change score) or combined to represent a total change score.

This is helpful in determining whether it is change per se that influences experienced stress or whether it is the negative change score that has the greater influence as suggested by Mueller, Edwards and Yarvis (1977).

3.5.2.1. Reliability of the L.E.S.

Sarason et al. (1978) quote the following test-retest reliability coefficients taken from two studies that retested students after a 5-6 week interval:-

Type of Change	Study 1	Study 2
positive change	.19	.53 (p<.001)
negative change	.56 (p<.001)	.88 (p<.001)
total change	.63 (p<.001)	.64 (p<.001)

They point out the difficulties of trying to test reliability with such measures, such as intervening stressful events between the two tests. They nonetheless claim that the LES is a

> moderately reliable instrument especially when the negative and total change scores are considered (p936).

3.5.2.2. Validity of the L.E.S.

To test the extent to which the LES actually measured life stress, Sarason et al. compared it to other related and relevant

personality indices e.g. State-Trait Anxiety Inventory (Spielberger, Gorush & Lushene, in Sarason et al, 1978), Psychological Screening Inventory (Lanyon, in Sarason et al., 1978), Beck Depression Inventory (Beck, 1967), Internal-External Locus of Control (Rotter, 1966). They also compared the instrument to the Schedule of Recent Experiences (Holmes and Rahe, 1976). They report a positive and significant relationship between negative life events and a number of related independent measures of stress.

3.5.3. Survey of Affective Stress

The present study made use of the Survey of Affective Stress used by Goldstein et al. (1988) (see Appendix D). The specific areas of interest in their study, as in this study, were the variables dealing with Anger. These were Experienced Anger, Expressed Anger and Family Expressed Anger. The survey is a simple, 7-item questionnaire that evaluates the frequency and intensity of feelings of anger at home and at work. The scale was chosen as it is a short questionnaire that can be administered in a short time.

3.5.3.1. Administration and Scoring

Individuals are asked to indicate on an analog scale how often they feel angry (1 = "Never"; 10 = "Always"), how intense this feeling of anger is (1 = "No feeling like that"; 10 = "Extremely strong"), whether others are aware that they are angry, and whether their families tended to express anger when they were growing up. Scoring is determined as follows:

Experienced Anger = (work frequency x intensity + home frequency x itensity)

Expressed Anger = frequency people at work know subject is angry + frequency family know subject is angry.

Family Expressed Anger = single item score.

No figures of reliability or validity are given.

CHAPTER 4: COMPARISON OF CORONARY GROUP WITH CONTROL GROUPS WITH RESPECT TO CORONARY RISK FACTORS

4.1. Results

This chapter forms a response to the hypotheses set out in section 3.2.

4.1.1. TABP

Hypothesis 1 states that, consistent with informal observations of CHD patients, TABP does not have a strong relationship with CHD among the Indian population in South Africa. The results from the study are tabulated as follows:

Table 5: **Distribution of TABP Among Samples**

Distribution of JAS Scores	MI n	MI %	Med n	Med %	Norm n	Norm %
Type A	16	64	4	16	6	24
Mixed	3	12	10	40	5	20
Type B	6	24	11	44	14	56
TOTALS	25	100	25	100	25	100
MEANS	6.33		3.57		-4.62	
S.D.	11.28		10.44		12.89	

* see graph 1.

The hypothesis was tested by using the Chi-Square test (for the different categories) and the Kruskal-Wallis 1-way ANOVA for actual JAS scores. Both these tests were done by Sperry mainframe computer on the SPSS statistical package and details are found in Appendix E.

The Chi-Square Test (3x3 tabulation) found that $p = 0.0019$ for all 3 categories (TABP, Mixed and TBBP) while exclusion of the

Mixed category (2x3 cross tabulation) revealed $p = 0.0049$. This indicates that the 3 groups are significantly ($p < 0.005$) different with respect to Type A categories. Kruskal-Wallis 1 way ANOVA further revealed $p = 0.003$, and indicates that those scoring high on the JAS come from a different population, when compared to those scoring low on the JAS. When both control groups were collapsed into a non-MI group, 2x2 Chi-Square revealed an even more significant result ($p < 0.0005$).

This suggests that TABP is a significant discriminator between those at risk for CHD and can identify these individuals from a sample which includes other medical patients as well as normals.

These results run counter to informal observations and suggest that Indian males exhibit similar TABP-CHD associations as do men in industrialised countries. There are a number of reasons that could account for this surprise finding and these will be dealt with in section 4.2.

4.1.2. Standard Coronary Risks

Hypothesis 2 assumes that CHD patients exhibit significantly more of the standard coronary risk factors than both normals and other hospitalised patients. It is taken that each of the coronary risk factors established in western studies can be generalised to Indian males living in South Africa. Observations would suggest that a large proportion of those with MI exhibit these risk factors (see Table 6 and Graph 1).

Table 6: **Distribution of Standard CHD Risk Factors by Group**

Risk Factor	MI n	MI %	Med n	Med %	Norm n	Norm %
Diabetes	7	28	4	16	2	8
Previous MI	5	20	4	16	0	0
Hypertension	11	44	8	32	1	4
Smoking	20	80	11	44	16	64
Alcohol	19	76	16	64	16	64
Physical Inactivity	21	84	21	84	12	48
Obesity	8	32	5	20	2	8
Family History of CHD	11	44	9	36	13	52

*see graph 1.

a) Diabetes: Chi-Square test of all three groups (2x3 cross tabulation) has $p = 0.1707$. This would suggest that the three groups are not significantly different with respect to diabetes.

b) Previous MI: Significance levels when all three groups are considered, approaches that of significance at the $p < 0.05$ level ($p = 0.0705$; Chi-Square Test). This level becomes significant when the MI group is compared with the Normal Control group ($p < 0.025$). However, a history of MI is not able to identify those at risk for MI specifically, and should be seen rather as an indicator for those who are ill (MI and Medical groups combined: $p < 0.025$).

c) Hypertension: The Chi-Square test has $p < 0.005$ ($p = 0.0046$) for all three groups together suggesting that the Normal group is significantly different from the other two groups. When the MI and Medical groups are collapsed into one hospitalised group, this significance level remains at $p < 0.005$. This suggests that hypertension is a factor representative of illness as well as an indicator for those at risk for CHD. However, there is no

difference between Medical and MI groups, casting doubt on the specificity of the risk factor.

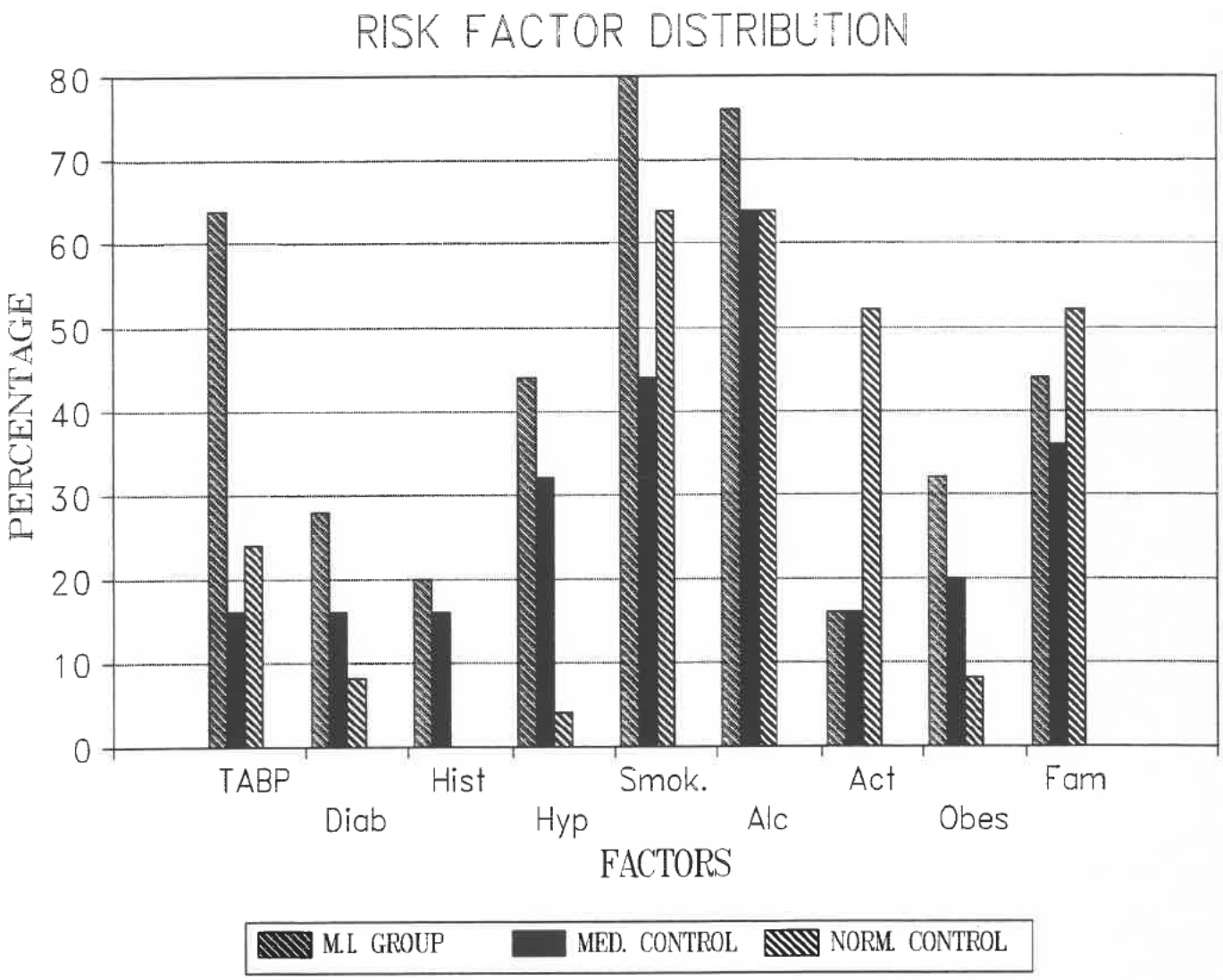
d) Smoking: There appears to be no significant difference between MI and Normal groups ($p < 0.35$). However, both these groups differ from the Medical group ($p < 0.05$) in that the Medical group has a lower incidence of smoking. This is a surprise result that may be attributable to the proportion of asthmatics in the non-coronary hospitalised sample. Many of these people had received specific recommendation from their doctors to stop smoking. The results indicate that smoking is not a factor that is able to distinguish between those with CHD and those that are apparently free of CHD.

e) Alcohol: There were no differences in the amounts of alcohol consumption in the three groups ($p = 0.7944$).

f) Physical Inactivity: The Normal group was significantly different from both of the other two groups ($p < 0.005$). There was no difference in levels of physical inactivity between the MI and Medical groups. Again this suggests that physical inactivity is an indicator for illness in general and not specifically CHD.

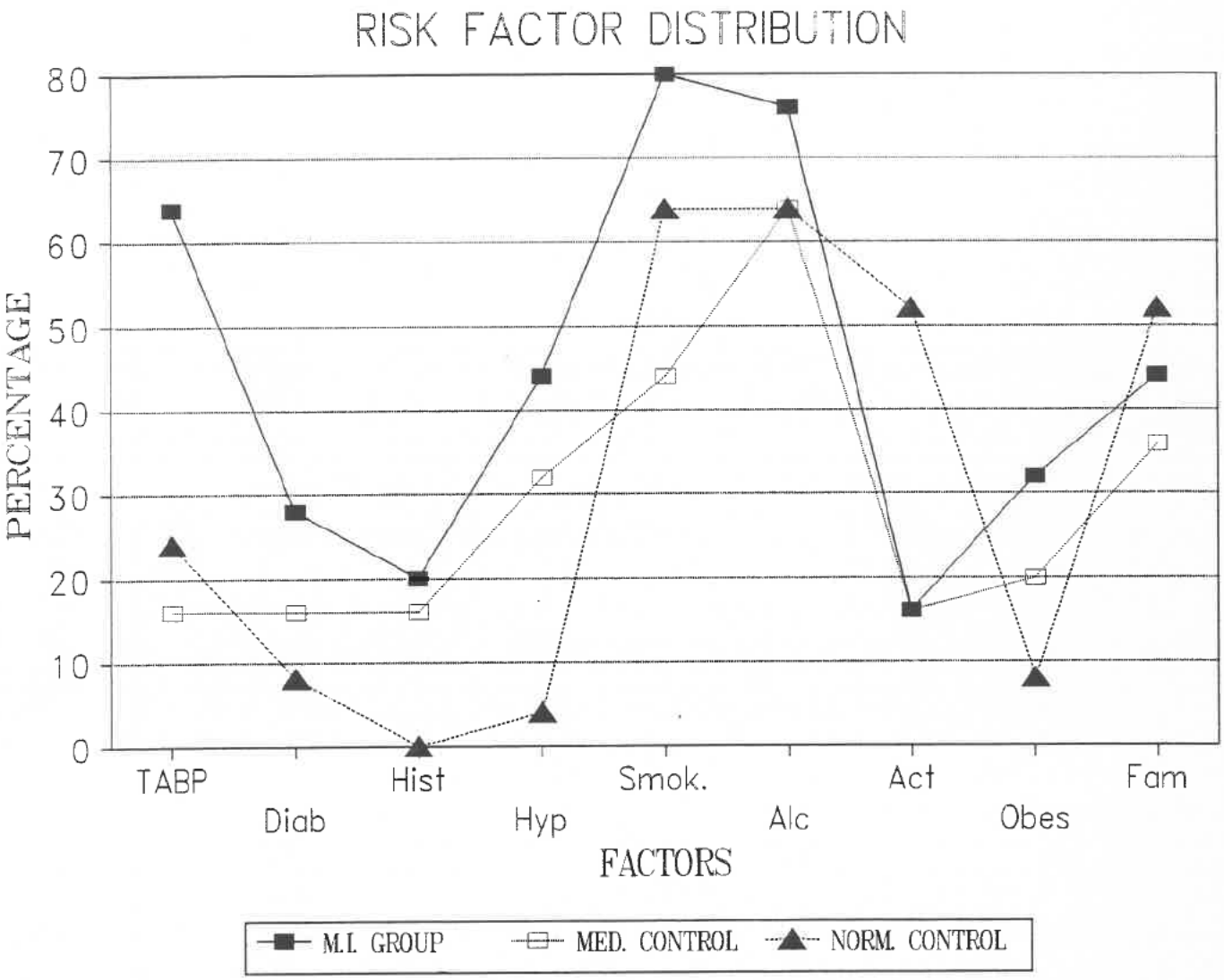
g) Obesity: Obesity was able to demarcate those specifically at risk for CHD ($p < 0.05$) when compared to the pooled sample of Normals and the Medical group. However there is no significant difference between Medical and MI patients ($p < 0.4$). This is consistent with the use of obesity as an indicator for illness in general (Jenkins, 1988).

Graph 1 (a): **Bar Graph of Distribution of Risk Factors among Samples.**



Where "Diab" = Diabetes, "Hist" = History of MI, "Hyp" = Hypertension, "Smok" = Smoker, "Alc" = Consumes alcohol, "Act" = Physically Active, "Obes" = Obese and "Fam" = Family history of MI.

Graph 1 (b): **Line Graph of Distribution of Risk Factors among Samples.**



Where "Diab" = Diabetes, "Hist" = History of MI, "Hyp" = Hypertension, "Smok" = Smoker, "Alc" = Consumes alcohol, "Act" = Physically Active, "Obes" = Obese and "Fam" = Family history of MI.

h) Family history of CHD: There was no significant difference between the three groups (p = 0.5224).

4.1.3. Stress

Hypothesis 3 states that certain stresses predispose vulnerable individuals to CHD. It is assumed that the higher the reported level of life stress, the greater the likelihood for illness, particularly CHD.

This hypothesis would be verified if coronary patients reported greater levels of experienced stress. The breakdown of stress scores are tabulated as follows:

Table 7: **Total Change Score Distribution**

SCORE	MI		Med		Norm	
	n	%	n	%	n	%
-20 - -16	1	4	4	16	0	0
-15 - -11	4	16	2	8	3	12
-10 - -6	9	54	8	32	5	20
-5 - 0	11	44	11	44	15	60
1 - 5	0	0	0	0	2	8
TOTALS	25	100	25	100	25	100
MEANS	-6.72		-6.64		-4.76	
S.D.	4.83		6.42		4.06	

Kruskal-Wallis 1 way ANOVA with all three groups revealed a non-significant result (Chi-Square = 1.953, p = 0.3739). This indicates that the three groups could have all come from the same sample. In other words, levels of reported stress do not constitute a marker for those at risk for CHD. When medical and MI groups were collapsed into one hospitalised group, the result was still not significant at the p = 0.05 level (Mann-Whitney U

Test has 2-tailed p = 0.1985). The Experimental Group has only negative total change scores which would be consistent with literature. However, this result is not significant when compared to control groups. One can conclude that stress levels do not constitute an indicator for those at risk for MI or illness in general.

4.1.4. Anger

Hypothesis 4 holds that there may be a relationship between dimensions of anger and CHD. As with studies conducted in the West, it is assumed that the greater the level of Suppressed Anger, the greater the likelihood for CHD.

Table 8: **Summary of Anger Score Distribution**

Anger	MI		Med		Norm	
Dimen	Mean	S.D.	Mean	S.D.	Mean	S.D.
XA	67.76	45.03	55.12	33.13	39.44	40.40
EP	13.56	6.09	13.76	6.58	12.68	5.74
FS	5.28	3.24	5.91	2.95	6.16	2.32

where XA = Experienced Anger Score
EP = Expressed Anger Score
FS = Family Style (i.e. expressed or withheld)

Table 9: **Breakdown of Mean Ranks by Group**

Anger	MI	Med	Norm	Signif-
Dimen	Mean Rank	Mean Rank	Mean Rank	icance.
XA	44.84	40.76	28.40	0.021
EP	38.58	40.66	34.76	0.624
FS	34.04	38.00	39.04	0.680

Results indicate that the three groups are not significantly different with respect to Expressed Anger (0.6242), nor are they significantly different on the dimension of Family Expressed

Anger ($p = 0.6809$).

However, the groups were different on the dimension of Experienced Anger ($p = 0.0211$) with the Experimental group having a higher level of Experienced Anger than controls. This level of significance increased according to the Mann-Whitney U Test ($p = 0.0070$; $p < 0.01$) when MI and Medical groups were combined. This suggests that levels of Experienced Anger are better able to identify those who are ill than those with MI specifically.

4.1.5. Specificity of Risk Factors

Hypothesis 5 held that risk factors would differ in their ability to identify those with CHD. It was assumed that the standard risk factors would be more powerful indicators.

It appears that there are no traditional coronary risk factors that are able to distinguish MI subjects from both Medical and Normal controls (see graph 1). This suggests that the specificity of standard coronary risk factors is not evident in this sample. The standard risk factors covered in this study appear to be better at identifying those that are ill (MI and Medical groups combined) compared to those that are apparently healthy. All of the risks appeared to be able to do this, except for smoking, alcohol consumption and a family history of CHD, which were not able to discriminate between the groups at all.

When the nonstandard factors of stress and anger were considered, only Experienced Anger was able to identify hospitalised (MI and Medical groups combined) subjects from normals. Stress, Expressed

Anger and Family Anger were not significantly different for any of the groups.

Of major importance was the finding that TABP was both able to identify the MI group from both the Medical and Normal groups (see graph 1). This was the only factor included in this study that was able to do so.

4.1.6. Risk Clusters

Hypothesis 6 states that there may be certain combinations of factors that are able to differentiate the three groups. Preliminary discriminant analysis with TABP, Experienced Anger and Hypertension was only able to correctly classify 50.67% of cases. This increased to 57.33% when combinations of TABP, Physical Inactivity and Hypertension were used.

4.2. Discussion of Results

The most important result of this study was the surprise finding that TABP was significantly associated with CHD in this population. Also of importance was the finding that, while risk factors are evident in the coronary group, comparison with controls reveals that the majority of factors are not able to differentiate between coronary and control groups. This chapter deals with these two core issues in more detail.

4.2.1. TABP-CHD Association

The initial hypothesis that the TABP-CHD association would not be evident in this population was based on observation. These observations were that coronary patients at Northdale Hospital were, on the whole, relaxed and accommodating people. This was in contrast to the Type A traits of aggressiveness, competitiveness and time-consciousness. Hence it was assumed that the TABP-CHD association was not evident in this population. The results obtained in this study suggest that (a) observations were inaccurate or (b) observations were accurate and TABP was not manifested by Type A patients for some reason. While the possibility exists that observations were inaccurate, there may be factors that reduced the overt expressions of the A pattern. As these observations were made during ward rounds, the possibility exists that patients who normally exhibited the A pattern did not do so during the ward round. Politeness and feelings of intimidation are two variables that may have influenced patient behaviour.

Another reason suggested by the literature could be that the manifest aspects of the pattern (measured by SI) temporarily decline immediately after MI (see the findings of Abbott et al., [1988] in section 2.11.3.). As patients were seen soon after admission, this phenomenon may have been evident. The JAS tends to measure content as opposed to the more process measurements of the SI (Rosenman, 1978). One would therefore expect JAS scores to be unaffected, while the overt behaviours measured by the SI would change.

Other reasons for this error could be that a ward round does not constitute an environmental challenge for the Type A person. Without a challenge the pattern would not be elicited (Friedman & Rosenman, 1977). Lastly, there may have been inaccuracies in the observers' understandings of what constituted TABP.

4.2.2. Standard Risk Factors

This study suggests a failure of the majority of standard risk factors to identify those at risk for CHD. This failure is only evident when control groups are used. These results may be due to inaccuracies in the data sheet which relied on respondents' self knowledge in health areas. If the results are accurate, they should be comparable to other studies with Indian men that were more rigorous. One such study was conducted by Sewdarsen et al. (1987) at the R.K. Kahn Hospital, where patients were medically examined. Sewdarsen at al. found the following percentages of standard risk factors in their sample of 108 men with an average age of 36 years (the results of this study are shown in adjacent brackets):

Table 9 : **Comparison of Results with Sewdarsen et al. (1987)**

Risk Factor	%	%
Hypercholesterolaemia	50	
Hypertriglceridaemia	53	
HDL cholesterol<0,38 mmol/l	52	
Family history of CAD	47	(44)*
Hypertension	32	(44)
Diabetes mellitus	30	(28)
Impaired glucose tolerance	7	
Previous history of MI/angina	31	(20)**
Obesity	27	(32)
Smokes cigarettes	79	(80)

* = Family history of CHD
** = Previous history of MI only

Results in all areas are similar suggesting a close match between the results of this study and the results gained by Sewdarsen at al. (1987).

Caution needs to be exercised when considering these percentages. In the absence of a control group, there is no basis on which to judge the relative strengths of these risks. Results have indicated that the experimental group and control groups used in this study are significantly different with respect to only TABP. The risk factors investigated in this study are distributed as follows:

Table 10: **Risk Factor Distribution Including Results of Sewdarsen et al. (1987)**

Factor	* Sew	MI	Med	Norm
Family history	%	%	%	%
of CAD or CHD	47	44	36	52
Hypertension	32	44	32	4
Diabetes	30	28	16	8
Previous MI	31	20	16	0
Obesity	27	32	16	8
Cigarettes	79	80	44	64
Phys Active		16	12	52
Alcohol		80	56	64
TABP		64	12	24

* Sew = Results of Sewdarsen et al. (1987).

Significantly specific CHD indicators would have to be significantly different to **both** Medical and Normal controls. Any similarity between the MI group and either of the control groups (i.e. MI-Med, MI-Norm) would suggest that the risk factor is unable to mark those with specifically CHD (see graph 1.).

4.2.3. Implications for Intervention

The central implication arising from this study is that confidence in the ability of standard coronary risks to identify Indian men at risk for CHD may be unfounded. Furthermore, inclusion of TABP as a risk factor for CHD may assist clinicians in better predicting those at risk for CHD. Evidence suggesting that TABP is able to be modified and that this modification in turn reduces CHD risk (Powell et al., 1984), may also assist clinicians in CHD treatment.

More generally, this study suggests that clinical and behavioural observations of TABP in those recovering from MI, may be unreliable. This may be because overt elements of the pattern decrease immediately after MI. This casts doubt on the SI as a method of TABP assessment in recent MI victims as it relies on overt behaviour. This may mean that JAS scores measure the enduring elements of TABP better than the SI in such patients, and is thus a more reliable instrument.

4.2.4. Limitations of This Study

This study made extensive use of the JAS. As mentioned earlier (section 2.12.4.), the JAS was standardised on American men and has not been standardised for South African males. The results in the present study could be due to unreliability of the JAS with this sample. This is seen to be a major limitation in this study. Furthermore, Form N of the JAS was used, which provides a global TABP score. Other forms of the JAS (e.g. Form C), make use of subscales that assess the core behaviours of the pattern (time consciousness, hostility and competitiveness) as well.

These scales often have stronger relationships with CHD independent from the global score (Booth-Kewley & Friedman, 1987).

Some of the terms in the JAS were not fully understood e.g. "hard-driving" and one item was phrased in the negative ("Would people who know you well agree that you have less energy than most people?"), which was sometimes experienced as confusing.

As all the data was collected by the researcher, there is the possibility of experimenter bias.

No medical corroboration was given for self-reported health status in the respondents. However, the similarity of the coronary group's results in this study compare favourably with the results presented by Sewdarsen et al. (1987) in which subjects were medically examined.

This study was restricted to those with MI. While this does provide consistency in terms of CHD endpoint, other forms of CHD (e.g. angina pectoris, arteriosclerosis) were excluded. Conclusions from this research should strictly be generalised to MI only.

Not all forms of coronary heart disease are experienced as severe. People experiencing angina pectoris may not seek medical help as chest pain may not be extreme. Three subjects from the experimental group discovered after medical examination that they

had suffered previous undetected CHD. Members of the control groups may also have had undetected CHD, in which case a history of no CHD would be misrepresented.

Attempts were made in this study to ensure a sample (n = 75) that included both hospitalised and non-hospitalised controls. However, the number of subjects per sample group (n = 25) is relatively small.

This study used a cross-sectional design that was retrospective in nature. Longitudinal research that takes a prospective approach would give a more rigorous design.

4.4. Conclusion

Given the limitations discussed above, the present study indicates that the standard coronary risk factors investigated do not significantly distinguish coronary patients from other hospitalised patients and normal controls. Results also indicate that the Type A Behaviour Pattern is able to significantly distinguish individuals with coronary heart disease from both apparently healthy individuals and those hospitalised with non-coronary complaints. Despite the exploratory nature of the study, findings suggest that the Type A Behaviour Pattern does constitute a valid coronary risk factor among Indian males. Given the significance of the factor in this study, it appears to be an issue that needs to be included in intervention programmes at both a primary and secondary level. As the pattern is behavioural and lends itself to modification, it may constitute an area where intervention may be especially productive.

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Appendix A

U N I V E R S I T Y O F N A T A L

P I E T E R M A R I T Z B U R G

PSYCHOLOGY DEPARTMENT

I am presently studying coronary heart disease in the Indian community in Pietermaritzburg. To do this effectively, there are a number of areas that must be investigated. I would greatly appreciate it if you would be prepared to participate in such a study as it is an important area of investigation. If you are prepared to take part, I must ask for two things:-

a) your permission
b) your time in completing a questionnaire.

Please realise that you are under no obligation to take part at all.

YOU ARE ASSURED OF COMPLETE CONFIDENTIALITY

Thank you for considering this request and your participation should you decide to carry on.

Tim Barry
Clinical Psychology Intern

I hereby voluntarily agree to take part in this research on the condition that my name does not appear in the written study nor is there any reference to my identity.

Signed_____

Name Please print_____

Witness_____ Date _____

Questionnaire Section

Thank you for your co-operation.

This questionnaire is quite long. It does not have to be done in one sitting and feel free to take a break at any time.

Date of birth_____ Age_____

		tick
Present marital status	single	
	married	
	separated	
	divorced	
	widowed	

Highest school standard passed_____

University or technical qualification_____

Father's educational standard_____

Mother's educational standard_____

Present occupation_____

Have you or any of your parents suffered from any of the following:-
(✓ for YES X for NO)

Mother	diabetes	
	heart attack	
	hypertension	
Father	diabetes	
	heart attack	
	hypertension	
Yourself	diabetes	
	heart attack	
	hypertension	

Do you suffer from any other major physical illness?
If NO please ignore.
If YES please state_____

Smoking habits	never smoked	
	used to smoke	
	smoke less than 10 a day	
	smoke 10 to 19 a day	
	smoke 20 to 29 a day	
	smoke more than 30 a day	

Have you been medically advised to stop smoking (/ or X)_____

Alcohol consumption	never drink alcohol	
	used to drink alcohol	
	drink once a month	
	drink once a week	
	drink every day	*

* more than 1 litre of beer/3 glasses of spirits	
less than 1 litre of beer/3 glasses of spirits	

Physical Activity: how many hours at work do you spend on the following (an estimate will do):-

__sitting_____
__standing_____
__moving_____
__carrying a load_____

How far away is work?_____kms

Do you go to work by_____motor vehicle_____|__|
_____bicycle_____|__|
_____walking_____|__|
_____bus_____|__|
_____taxi_____|__|

Do you do any physical activity in your leisure?
_____NO___|__|
_____YES___|__|*

*What kind of activity?_____
*How often?_____

When last did you do any exercise?_____

Have you been medically advised to start exercising?_____
Have you been medically advised to stop exercise?_____

Are you overweight?_____
If YES, please state amount in kgs_____
Have you ever been medically advised to lose weight?_____

Appendix C

Listed below are a number of events which sometimes bring about change in the lives of those who experience them and which necessitate social readjustment. Please check those events that you have experienced in the recent past and indicate the time period during which you have experienced each event. Be sure that all check marks are directly across from the items they correspond to.

Also for each item checked below, please indicate the extent to which you viewed the event as having either a positive or negative impact on your life at the time the event occurred. That is, indicate the type and extent of impact that the event had. A rating of -3 would indicate an extremely negative impact. A rating of 0 suggests no impact either positive or negative. A rating of +3 would indicate an extremely positive impact.

Please ring the score that applies to you:

	0 to 6 mo	7 mo to 1 yr	negative <--> positive
1 Marriage_____			-3 -2 -1 0 +1 +2 +3

2 Detention in jail or similar institution _____ -3 -2 -1 0 +1 +2 +3

3 Death of wife _____ -3 -2 -1 0 +1 +2 +3

4 Major change in sleeping habits (much more or much less) _____ -3 -2 -1 0 +1 +2 +3

5 Death of a close family member

____a) mother _____ -3 -2 -1 0 +1 +2 +3

____b) father _____ -3 -2 -1 0 +1 +2 +3

____c) sister _____ -3 -2 -1 0 +1 +2 +3

____d) brother _____ -3 -2 -1 0 +1 +2 +3

____e) grandmother _____ -3 -2 -1 0 +1 +2 +3

____f) grandfather _____ -3 -2 -1 0 +1 +2 +3

____g) other (specify) _____ -3 -2 -1 0 +1 +2 +3

6 Major change in eating habits (much more or much less) _____ -3 -2 -1 0 +1 +2 +3

7 Foreclosure on mortgage or loan _____ -3 -2 -1 0 +1 +2 +3

8 Death of a close friend _____ -3 -2 -1 0 +1 +2 +3

9 Outstanding personal achievement _____ -3 -2 -1 0 +1 +2 +3

10 Minor law violations _____ -3 -2 -1 0 +1 +2 +3

11 Wife/girlfriend's pregnancy _____ -3 -2 -1 0 +1 +2 +3

12 Changed work situation _____ -3 -2 -1 0 +1 +2 +3

13 New job _____ -3 -2 -1 0 +1 +2 +3

14 Serious injury or illness to close family member:

_a) father _____ -3 -2 -1 0 +1 +2 +3

_b) mother _____ -3 -2 -1 0 +1 +2 +3

_c) sister _____ -3 -2 -1 0 +1 +2 +3

_d) brother _____ -3 -2 -1 0 +1 +2 +3

_e) grandfather _____ -3 -2 -1 0 +1 +2 +3

_f) grandmother _____ -3 -2 -1 0 +1 +2 +3

_g) other (specify) _____ -3 -2 -1 0 +1 +2 +3

15 Sexual difficulties _____ -3 -2 -1 0 +1 +2 +3

16 Trouble with employer _____ -3 -2 -1 0 +1 +2 +3

17 Trouble with in-laws _____ -3 -2 -1 0 +1 +2 +3

18 Major change in financial status _____ -3 -2 -1 0 +1 +2 +3

19 Major change in family closeness _____ -3 -2 -1 0 +1 +2 +3

20 Gaining a new family member _____ -3 -2 -1 0 +1 +2 +3

21 Change in residence _____ -3 -2 -1 0 +1 +2 +3

22 Marital separation due to conflict _____ -3 -2 -1 0 +1 +2 +3

23 Major change in church activity (more or less) _____ -3 -2 -1 0 +1 +2 +3

24 Marital reconciliation with wife _____ -3 -2 -1 0 +1 +2 +3

25 Major change in number of arguments with wife (a lot more or a lot less) _____ -3 -2 -1 0 +1 +2 +3

26 Change in wife's work outside the home (new job...) _____ -3 -2 -1 0 +1 +2 +3

27 Major change in amount and/or usual type of recreation _____ -3 -2 -1 0 +1 +2 +3

28 Borrowing more than R10 000 _____ -3 -2 -1 0 +1 +2 +3

29 Borrowing less than R10 000 _____ -3 -2 -1 0 +1 +2 +3

30 Being fired from job _____ -3 -2 -1 0 +1 +2 +3

31 Wife/girlfriend having abortion _____ -3 -2 -1 0 +1 +2 +3

32 Major personal illness or injury _____ -3 -2 -1 0 +1 +2 +3

33 Major change in social activities eg parties _____ -3 -2 -1 0 +1 +2 +3

34 Major change in living conditions of family _____ -3 -2 -1 0 +1 +2 +3

35 Divorce _____ -3 -2 -1 0 +1 +2 +3

36 Serious illness or injury of close friend _____ -3 -2 -1 0 +1 +2 +3

37 Retirement from work _____ -3 -2 -1 0 +1 +2 +3

38 Son or daughter leaving home (eg marriage) _____ -3 -2 -1 0 +1 +2 +3

39 Separation from wife (due to work etc) _____ -3 -2 -1 0 +1 +2 +3

40 Engagement _____ -3 -2 -1 0 +1 +2 +3

41 Breaking up with girlfriend _____ -3 -2 -1 0 +1 +2 +3

42 Leaving home for the first time _____ -3 -2 -1 0 +1 +2 +3

43 Reconciliation with girlfriend _____ -3 -2 -1 0 +1 +2 +3

Other recent experiences which have had an impact on your life. List and rate.

	0 to 6 mo	7 mo to 1 yr	positive <---> negative
44 _____			-3 -2 -1 0 +1 +2 +3
45 _____			-3 -2 -1 0 +1 +2 +3
46 _____			-3 -2 -1 0 +1 +2 +3
47 _____			-3 -2 -1 0 +1 +2 +3

Appendix B

Please circle the option that is most true for you:-

1. When you are under pressure or stress, do you usually:
   a) Do something about it immediately?
   b) Plan carefully before taking any action?

2. When you listen to someone talking, and this person takes too long to come to the point, do you feel like hurrying him along?
a) Frequently
b) Occasionally
c) Almost never

3. How often do you "put words in his mouth" in order to speed things up?
a) Frequently
b) Occasionally
c) Almost never

4. If you tell your wife or a friend that you will meet them somewhere at a definite time, how often do you arrive late?
a) Once in a while
b) Rarely
c) I am never late

5. When you were young, did most people consider you to be
a) Definitely hard-driving and competitive?
b) Probably hard driving and competitive?
c) Probably more relaxed and easy-going?
d) Definitely relaxed and easy-going?

6. How would your wife (or closest friend) rate you currently?
a) Definitely hard-driving and competitive?
b) Probably hard driving and competitive?
c) Probably more relaxed and easy-going?
d) Definitely relaxed and easy-going?

7. How would your wife (or closest friend) rate your general level of activity?
a) Too slow. Should be more active.
b) About average. Is busy much of the time.
c) Too active. Needs to slow down.

8. How was your "temper" when you were younger?
a) Fiery and hard to control.
b) Strong, but controllable.
c) No problem.
d) I almost never got angry.

9. Would people who know you well agree that you have less energy than most people?
a) Definitely Yes.
b) Probably Yes.
c) Probably No.
d) Definitely No.

10. When you are in a group, do other people tend to look to you to provide leadership?
a) Rarely.
b) About as often as they look to others.
c) More often than they look to others.

FOR EACH OF THE NEXT QUESTIONS, COMPARE YOURSELF WITH THE AVERAGE PERSON HAVING THE SAME KIND OF DAILY ACTIVITIES AND LIFE SITUATION AS YOU.

11. In being precise (careful about detail), I am
    a) Much more precise.
    b) A little more precise.
    c) A little less precise.
    d) Much less precise.

12. I approach life in general
    a) Much more seriously.
    b) A little more seriously.
    c) A little less seriously.
    d) Much less seriously.

13. When you were at high school, college or university, did you play on any athletic teams?
    a) No
    b) Yes, one team
    c) Yes, two or more teams

Appendix D

This is the last section: Please circle the option most true of you.

1. At WORK how often do you feel angry?

Never									Always
0	1	2	3	4	5	6	7	8	9	10

2. How STRONG are these feelings of anger at work?

No feelings like that		Slightly strong		Moderately strong		Markedly strong		Extremely strong
0	1	2	3	4	5	6	7	8	9	10

3. At HOME, ho often do you feel angry?

Never									Always
0	1	2	3	4	5	6	7	8	9	10

4. How STRONG are these feelings of anger at home?

No feelings like that		Slightly strong		Moderately strong		Markedly strong		Extremely strong
0	1	2	3	4	5	6	7	8	9	10

5. When you are feeling angry does your spouse (if not living with a spouse, does your best friend) know it?

Never									Always
0	1	2	3	4	5	6	7	8	9	10

6. When you are feeling angry at work are the people you work with aware of your feelings?

Never Rarely On Occasion Often Very Often Always
0_____1_____2_____3_____4_____5_____6_____7_____8_____9_____10

7. Was it usual in your family when you were growing up, for people to show their anger if they were feeling it?

Never Rarely On Occasion Often Very Often Always
0_____1_____2_____3_____4_____5_____6_____7_____8_____9_____10

THANK YOU VERY MUCH FOR YOUR CO-OPERATION.

Chi Square Test for Two Independent Samples
(Taken from Siegel, 1956, p.107)

The following Chi-Square tests with 2x2 contingency tables were calculated according to the formula:

$$\chi^2 = \frac{N\,([AD-BC]-N/2)}{(A+B)(C+D)(A+C)(B+D)}$$

df = 1

Diabetes:

	MI	Norm	
Non-diabetic	18	23	41
Diabetic	7	2	9
	25	25	50

$$\chi^2 = \frac{500000}{230625}$$

$$= 2.16$$

With df = 1, p < 0.5(0.2) = p < 0.1.

Previous MI:

	MI	Norm	
No prev. MI	20	25	45
Previous MI	5	0	5
	25	25	50

$$\chi^2 = \frac{500000}{140625}$$

$$= 3.56$$

With df = 1, p < 0.5(0.05) = p < 0.025.

	MI+Med	Norm	
No prev. MI	41	25	66
Previous MI	9	0	9
	50	25	75

$$\chi^2 = \frac{2636718.8}{742500}$$

$$= 3.55$$

With df = 1, p < 0.5(0.05) = p < 0.025.

Hypertension:

	MI+Med	Norm	
Hypertension	31	24	55
No Hyperten.	19	1	20
	50	25	75

$$\chi^2 = \frac{11261719}{1375000}$$

$$= 8.19$$

With df = 1, p < 0.5(0.01) = p < 0.005.

Smoking:

	MI	Norm	
Smoker	20	16	36
Non-smoker	5	9	14
	25	25	75

$$\chi^2 = \frac{281250}{315000}$$

$$= 0.893$$

With df = 1, p < 0.5(0.7) = p < 0.35.

Obesity:

	MI	Norm+Med	
Obese	8	7	15
Non-obese	17	43	60
	25	50	75

$$\chi^2 = \frac{2636718.8}{1125000}$$

$$= 2.34$$

With df = 1, p < 0.5(0.1) = p < 0.05.

	MI	Med	
Obese	8	5	13
Non-obese	17	20	37
	25	25	50

$$\chi^2 = \frac{125000}{300625}$$

$$= 0.416$$

With df = 1, p < 0.5(0.8) = p < 0.4.

SMOKING	COUNT	GROUP 1 M.I.	2 MEDICAL CONTROL	3 NORMAL CONTROL	ROW TOTAL
DO NOT SMOKE	6	5	14	9	28 37.3
SMOKERS	7	20	11	16	47 62.7
	COLUMN TOTAL	25 33.3	25 33.3	25 33.3	75 100.0

CHI-SQUARE	D.F.	SIGNIFICANCE	MIN E.F.	CELLS WITH E.F.< 5
6.95289	2	.0309	9.333	NONE

NUMBER OF MISSING OBSERVATIONS = 0
:0 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
)8:30:37 University of Natal SPERRY 1100/70 39R3E

- C R O S S T A B U L A T I O N O F ALCOHOL BY GROUP - PAGE 1 OF

ALCOHOL	COUNT	GROUP 1 M.I.	2 MEDICAL CONTROL	3 NORMAL CONTROL	ROW TOTAL
	0	6	8	9	23 31.1
	1	4	4	3	11 14.9
	2	1	3	2	6 8.1
	3	11	6	6	23 31.1
	4	3	3	5	11 14.9
	COLUMN TOTAL	25 33.8	24 32.4	25 33.8	74 100.0

CHI-SQUARE	D.F.	SIGNIFICANCE	MIN E.F.	CELLS WITH E.F.< 5
4.64401	8	.7949	1.946	9 OF 15 (60.0%)

NUMBER OF MISSING OBSERVATIONS = 1
:0 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
)8:30:37 University of Natal SPERRY 1100/70 39R3E

THIS PRINTOUT CREATED "PARENT#MI# AND EXCLUDED MIXED CASES ETC DATE 072491

- C R O S S T A B U L A T I O N O F PREV#MI BY GROUP - PAGE 1 OF

PREV#MI	COUNT	GROUP 1 M.I.	2 MEDICAL CONTROL	3 NORMAL CONTROL	ROW TOTAL
	0	20	21	25	66 88.0
	1	5	4		9 12.0
	COLUMN TOTAL	25 33.3	25 33.3	25 33.3	75 100.0

CHI-SQUARE	D.F.	SIGNIFICANCE	MIN E.F.	CELLS WITH E.F.< 5
5.30303	2	.0705	3.000	3 OF 6 (50.0%)

NUMBER OF MISSING OBSERVATIONS = 0
24 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
12:37:36 University of Natal SPERRY 1100/70 39R3E

- C R O S S T A B U L A T I O N O F HYPERTEN BY GROUP - PAGE 1 OF

HYPERTEN	COUNT	GROUP 1 M.I.	2 MEDICAL CONTROL	3 NORMAL CONTROL	ROW TOTAL
	0	14	17	24	55 73.3
	1	11	8	1	20 26.7
	COLUMN TOTAL	25 33.3	25 33.3	25 33.3	75 100.0

CHI-SQUARE	D.F.	SIGNIFICANCE	MIN E.F.	CELLS WITH E.F.< 5
10.77273	2	.0046	6.667	NONE

NUMBER OF MISSING OBSERVATIONS = 0
24 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
12:37:36 University of Natal SPERRY 1100/70 39R3E

- C R O S S T A B U L A T I O N O F SMOKING BY GROUP - PAGE 1 OF

GROUP

TOTAL	33.8	32.4	33.8	100.0

CHI-SQUARE	D.F.	SIGNIFICANCE	MIN E.F.	CELLS WITH E.F.< 5
4.64401	8	.7949	1.946	9 OF 15 (60.0%)

NUMBER OF MISSING OBSERVATIONS = 1

24 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
12:37:38 University of Natal SPERRY 1100/70 39R3E

- C R O S S T A B U L A T I O N O F PHYS$ACT BY GROUP - PAGE 1 0

PHYS$ACT \ GROUP	M.I. (1)	MEDICAL CONTROL (2)	NORMAL CONTROL (3)	ROW TOTAL
0	21	21	12	54 / 72.0
1	4	4	13	21 / 28.0
COLUMN TOTAL	25 / 33.3	25 / 33.3	25 / 33.3	75 / 100.0

CHI-SQUARE	D.F.	SIGNIFICANCE	MIN E.F.	CELLS WITH E.F.< 5
10.71429	2	.0047	7.000	NONE

NUMBER OF MISSING OBSERVATIONS = 0

24 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
12:37:38 University of Natal SPERRY 1100/70 39R3E

- C R O S S T A B U L A T I O N O F START$EX BY GROUP - PAGE 1 0

START$EX \ GROUP	M.I. (1)	MEDICAL CONTROL (2)	NORMAL CONTROL (3)	ROW TOTAL
0	18	24	25	67 / 89.3
1	7	1		8 / 10.7
COLUMN TOTAL	25 / 33.3	25 / 33.3	25 / 33.3	75 / 100.0

CHI-SQUARE	D.F.	SIGNIFICANCE	MIN E.F.	CELLS WITH E.F.< 5
12.03358	2	.0024	2.667	3 OF 6 (50.0%)

THIS PRINTOUT CREATED "PARENTMI AND EXCLUDED MIXED CASES ETC DATE 072491

SMOKING \ GROUP	M.I. (1)	MEDICAL CONTROL (2)	NORMAL CONTROL (3)	ROW TOTAL
0	3	9	5	17 / 22.7
1	2	5	4	11 / 14.7
2	8	6	4	18 / 24.0
3	4	2	2	8 / 10.7
4	3	2	9	14 / 18.7
5	5	1	1	7 / 9.3
COLUMN TOTAL	25 / 33.3	25 / 33.3	25 / 33.3	75 / 100.0

CHI-SQUARE	D.F.	SIGNIFICANCE	MIN E.F.	CELLS WITH E.F.< 5
17.61446	10	.0618	2.333	12 OF 18 (66.7%)

NUMBER OF MISSING OBSERVATIONS = 0

24 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
12:37:37 University of Natal SPERRY 1100/70 39R3E

- C R O S S T A B U L A T I O N O F ALCOHOL BY GROUP - PAGE 1 0

ALCOHOL \ GROUP	M.I. (1)	MEDICAL CONTROL (2)	NORMAL CONTROL (3)	ROW TOTAL
0	6	8	9	23 / 31.1
1	4	4	3	11 / 14.9
2	1	3	2	6 / 8.1
3	11	6	6	23 / 31.1
4	3	3	5	11 / 14.9
COLUMN	25	24	25	74

- C R O S S T A B U L A T I O N O F - - - - - - - - - - - - - - - - - - -
BY GROUP
OVERWGHT PAGE 1 OF

COUNT	GROUP M.I. (1)	MEDICAL CONTROL (2)	NORMAL CONTROL (3)	ROW TOTAL
OVERWGHT 0	17	20	23	60 80.0
1	8	5	2	15 20.0
COLUMN TOTAL	25 33.3	25 33.3	25 33.3	75 100.0

CHI-SQUARE	D.F.	SIGNIFICANCE	MIN E.F.	CELLS WITH E.F.< 5
4.50000	2	.1054	5.000	NONE

NUMBER OF MISSING OBSERVATIONS = 0
24 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
12:37:40 University of Natal SPERRY 1100/70 39R3E

- C R O S S T A B U L A T I O N O F - - - - - - - - - - - - - - - - - - -
BY GROUP
ADD#ILLN PAGE 1 OF

COUNT	GROUP M.I. (1)	MEDICAL CONTROL (2)	NORMAL CONTROL (3)	ROW TOTAL
ADD#ILLN 0	15	23	15	53 70.7
.1	10	2	10	22 29.3
COLUMN TOTAL	25 33.3	25 33.3	25 33.3	75 100.0

CHI-SQUARE	D.F.	SIGNIFICANCE	MIN E.F.	CELLS WITH E.F.< 5
8.23328	2	.0163	7.333	NONE

NUMBER OF MISSING OBSERVATIONS = 0
[illegible] SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
12:37:40 University of Natal

- C R O S S T A B U L A T I O N O F - - - - - - - - - - - - - - - - - - -
BY GROUP
PARNT#MI

THIS PRINTOUT CREATED PARENT#MI# AND EXCLUDED MIXED CASES ETC. DATE 07/24/91

- PAGE 1 O

COUNT	GROUP M.I. (1)	MEDICAL CONTROL (2)	NORMAL CONTROL (3)	ROW TOTAL
PARNT#MI 0 NEITHER PARENT	14	16	12	42 56.0
1 EITHER PARENT	11	9	13	33 44.0
COLUMN TOTAL	25 33.3	25 33.3	25 33.3	75 100.0

CHI-SQUARE	D.F.	SIGNIFICANCE	MIN E.F.	CELLS WITH E.F.< 5
1.29870	2	.5224	11.000	NONE

NUMBER OF MISSING OBSERVATIONS = 0
24 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
12:37:41 University of Natal SPERRY 1100/70 39R3E

PRECEDING TASK REQUIRED 3.02 SECONDS CPU TIME; 10.23 SECONDS ELAPSED.

```
  22 .0    BREAKDOWN VARIABLES=T#SCORE STRESS EXP$ANGR EXPRESSD FAML#ANG (LO,HI)
  23 .0      GROUP(1,3)/TABLES=T#SCORE TO FAML#ANG BY GROUP
INTEGER BREAKDOWN NEEDS      108 WORDS OF MEMORY.
THERE ARE   12028 WORDS OF MEMORY AVAILABLE.
THE LARGEST CONTIGUOUS AREA HAS   12028 WORDS.
```

24 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
12:37:42 University of Natal SPERRY 1100/70 39R3E

- D E S C R I P T I O N O F S U B P O P U L A T I O N S - - - - - - - - - -

CRITERION VARIABLE T#SCORE
BROKEN DOWN BY GROUP

VARIABLE	VALUE	LABEL	MEAN	STD DEV	CASES
FOR ENTIRE POPULATION			-.6173	12.4577	75
GROUP	1		6.3344	11.2814	25
GROUP	2		-3.5696	10.4421	25
GROUP	3		-4.6168	12.8890	25

TOTAL CASES = 75
24 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
12:37:42 University of Natal SPERRY 1100/70 39R3E

- D E S C R I P T I O N O F S U B P O P U L A T I O N S - - - - - - - - - -

CRITERION VARIABLE STRESS
BROKEN DOWN BY GROUP

VARIABLE	VALUE	LABEL	MEAN	STD DEV	CASES
FOR ENTIRE POPULATION			-6.0400	5.2025	75

```
  BY GROUP

     MEAN RANK     CASES

          44.84        25    GROUP = 1    M.I.
          40.76        25    GROUP = 2    MEDICAL CONTROL
          28.40        25    GROUP = 3    NORMAL CONTROL
                       --
                       75    TOTAL

                                                CORRECTED FOR TIES
          CASES     CHI-SQUARE  SIGNIFICANCE     CHI-SQUARE  SIGNIFICANCE
             75         7.7139         .0211         7.7196         .0211

- - - - - KRUSKAL-WALLIS 1-WAY ANOVA
     EXPRESSD
  BY GROUP

     MEAN RANK     CASES

          38.58        25    GROUP = 1    M.I.
          40.66        25    GROUP = 2    MEDICAL CONTROL
          34.76        25    GROUP = 3    NORMAL CONTROL
                       --
                       75    TOTAL

                                                CORRECTED FOR TIES
          CASES     CHI-SQUARE  SIGNIFICANCE     CHI-SQUARE  SIGNIFICANCE
             75          .9426         .6242          .9585         .6193

30 JUL 91    SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:30:41     University of Natal              SPERRY 1100/70    39R3E

- - - - - KRUSKAL-WALLIS 1-WAY ANOVA
     FAML#ANG
  BY GROUP

     MEAN RANK     CASES

          34.04        25    GROUP = 1    M.I.
          38.00        23    GROUP = 2    MEDICAL CONTROL
          39.04        25    GROUP = 3    NORMAL CONTROL
                       --
                       73    TOTAL

                                                CORRECTED FOR TIES
          CASES     CHI-SQUARE  SIGNIFICANCE     CHI-SQUARE  SIGNIFICANCE
             73          .7688         .6809          .7807         .6768

30 JUL 91    SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:30:41     University of Natal              SPERRY 1100/70    39R3E

PRECEDING TASK REQUIRED         .92 SECONDS CPU TIME;        2.09 SECONDS ELAPSED.

  22  0          DISCRIMINANT GROUPS=GROUP(1,3)/VARIABLES=TYPE#A EXP#ANGR HYPERTEN
  23  0          STATISTICS 1 4 5 6 7 10 11 13 15
THERE ARE    11942 WORDS OF MEMORY AVAILABLE.
THE LARGEST CONTIGUOUS AREA HAS    11942 WORDS.
SINCE ANALYSIS= WAS OMITTED FOR THE FIRST ANALYSIS ALL VARIABLES
ON THE VARIABLES= LIST WILL BE ENTERED AT LEVEL 1.

THIS DISCRIMINANT ANALYSIS REQUIRES      4448 (      4.3K) WORDS OF WORKSPACE.
30 JUL 91    SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
```



```
                  +---------+---------+---------+
              1   I     11  I      9  I     13  I     33
  EITHER PARENT   I         I         I         I  44.0
                  +---------+---------+---------+
           COLUMN       25        25        25        75
            TOTAL     33.3      33.3      33.3     100.0

 CHI-SQUARE     D.F.       SIGNIFICANCE        MIN E.F.       CELLS WITH E.F.< 5
 ----------     ----       ------------        --------       ------------------

    1.29870        2             .5224           11.000          NONE
NUMBER OF MISSING OBSERVATIONS =     0
30 JUL 91    SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:30:39     University of Natal              SPERRY 1100/70    39R3E

PRECEDING TASK REQUIRED        3.01 SECONDS CPU TIME;        6.79 SECONDS ELAPSED.

  21  0          NPAR TESTS K-W=T#SCORE STRESS EXP#ANGR EXPRESSD FAML#ANG BY GROUP(1,3).
THERE ARE    11028 WORDS OF MEMORY AVAILABLE.
THE LARGEST CONTIGUOUS AREA HAS    11028 WORDS.
 ***** WORKSPACE ALLOWS FOR     916 CASES FOR NPAR TESTS *****

30 JUL 91    SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:30:40     University of Natal              SPERRY 1100/70    39R3E

- - - - - KRUSKAL-WALLIS 1-WAY ANOVA
     T#SCORE
  BY GROUP

     MEAN RANK     CASES

          49.92        25    GROUP = 1    M.I.
          33.36        25    GROUP = 2    MEDICAL CONTROL
          30.72        25    GROUP = 3    NORMAL CONTROL
                       --
                       75    TOTAL

                                                CORRECTED FOR TIES
          CASES     CHI-SQUARE  SIGNIFICANCE     CHI-SQUARE  SIGNIFICANCE
             75         11.4008         .0033        11.4011         .0033

- - - - - KRUSKAL-WALLIS 1-WAY ANOVA
     STRESS
  BY GROUP

     MEAN RANK     CASES

          34.00        25    GROUP = 1    M.I.
          37.44        25    GROUP = 2    MEDICAL CONTROL
          42.56        25    GROUP = 3    NORMAL CONTROL
                       --
                       75    TOTAL

                                                CORRECTED FOR TIES
          CASES     CHI-SQUARE  SIGNIFICANCE     CHI-SQUARE  SIGNIFICANCE
             75          1.9530         .3766          1.9674         .3739

30 JUL 91    SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:30:40     University of Natal              SPERRY 1100/70    39R3E

- - - - - KRUSKAL-WALLIS 1-WAY ANOVA
     EXP#ANGR
```

WILKS' LAMBDA (U-STATISTIC) AND UNIVARIATE F-RATIO
WITH 2 AND 72 DEGREES OF FREEDOM

VARIABLE	WILKS' LAMBDA	F	SIGNIFICANCE
TYPE#A	.85553	6.079	.0036
PHYS#ACT	.85714	6.000	.0039
HYPERTEN	.85636	6.038	.0038

06 AUG 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:38:14 University of Natal SPERRY 1100/70 39R3E

- D I S C R I M I N A N T A N A L Y S I S - - - - - - - - - - - - - - - -

ON GROUPS DEFINED BY GROUP

ANALYSIS NUMBER 1
DIRECT METHOD: ALL VARIABLES PASSING THE TOLERANCE TEST ARE ENTERED.

MINIMUM TOLERANCE LEVEL..... .00100
CANONICAL DISCRIMINANT FUNCTIONS
MAXIMUM NUMBER OF FUNCTIONS..... 2
MINIMUM CUMULATIVE PERCENT OF VARIANCE... 100.00
MAXIMUM SIGNIFICANCE OF WILKS' LAMBDA.... 1.0000

PRIOR PROBABILITY FOR EACH GROUP IS .33333

CANONICAL DISCRIMINANT FUNCTIONS

FUNCTION	EIGENVALUE	PERCENT OF VARIANCE	CUMULATIVE PERCENT	CANONICAL CORRELATION	AFTER FUNCTION	WILKS' LAMBDA	CHI-SQUARED	D.F.	SIGNIFICAN
					0	.6753500	27.869	6	.0001
1*	.32368	82.75	82.75	.5215618	1	.9277126	5.3274	2	.0697
2*	.07792	17.25	100.00	.2688631					

* MARKS THE 2 CANONICAL DISCRIMINANT FUNCTION(S) TO BE USED IN THE REMAINING ANALYSIS.

STANDARDIZED CANONICAL DISCRIMINANT FUNCTION COEFFICIENTS

	FUNC 1	FUNC 2
TYPE#A	.62500	.72570
PHYS#ACT	.41816	-.68480
HYPERTEN	-.58727	.03267

06 AUG 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:38:15 University of Natal SPERRY 1100/70 39R3E

STRUCTURE MATRIX:
POOLED WITHIN-GROUPS CORRELATIONS BETWEEN CANONICAL DISCRIMINANT FUNCTIONS AND DISCRIMINATING VARIABLES
VARIABLES ARE ORDERED BY THE FUNCTION WITH LARGEST CORRELATION AND THE MAGNITUDE OF THAT CORRELATION.

	FUNC 1	FUNC 2
HYPERTEN	-.65784*	.27787
TYPE#A	.58651	.71940*
PHYS#ACT	.59015	-.68466*

- D I S C R I M I N A N T A N A L Y S I S - - - - - - - - - - - - - - - -

ON GROUPS DEFINED BY GROUP

75 (UNWEIGHTED) CASES WERE PROCESSED.
0 OF THESE WERE EXCLUDED FROM THE ANALYSIS.
75 (UNWEIGHTED) CASES WILL BE USED IN THE ANALYSIS.

NUMBER OF CASES BY GROUP

GROUP	NUMBER OF CASES UNWEIGHTED	WEIGHTED	LABEL
1	25	25.0	M.I.
2	25	25.0	MEDICAL CONTROL
3	25	25.0	NORMAL CONTROL
TOTAL	75	75.0	

GROUP MEANS

GROUP	TYPE#A	EXP#ANGR	HYPERTEN
1	1.60000	67.76000	.44000
2	2.28000	55.12000	.32000
3	2.32000	39.44000	.04000
TOTAL	2.06667	54.10667	.26667

POOLED WITHIN-GROUPS CORRELATION MATRIX

	TYPE#A	EXP#ANGR	HYPERTEN
TYPE#A	1.00000		
EXP#ANGR	-.09722	1.00000	
HYPERTEN	.07457	.05030	1.00000

CORRELATIONS WHICH CANNOT BE COMPUTED ARE PRINTED AS 99.0.
30 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:30:43 University of Natal SPERRY 1100/70 39R3E

WILKS' LAMBDA (U-STATISTIC) AND UNIVARIATE F-RATIO
WITH 2 AND 72 DEGREES OF FREEDOM

VARIABLE	WILKS' LAMBDA	F	SIGNIFICANCE
TYPE#A	.85553	6.079	.0036
EXP#ANGR	.91901	3.172	.0478
HYPERTEN	.85636	6.038	.0038

30 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:30:43 University of Natal SPERRY 1100/70 39R3E

- D I S C R I M I N A N T A N A L Y S I S - - - - - - - - - - - - - - - -

ON GROUPS DEFINED BY GROUP

ANALYSIS NUMBER 1
DIRECT METHOD: ALL VARIABLES PASSING THE TOLERANCE TEST ARE ENTERED.

UNSTANDARDIZED CANONICAL DISCRIMINANT FUNCTION COEFFICIENTS

	FUNC 1	FUNC 2
TYPE#A	.7616611	.8843886
PHYS#ACT	.9856175	-1.614096
HYPERTEN	-1.407268	.7823154-001
(CONSTANT)	-1.474801	-1.396651

CANONICAL DISCRIMINANT FUNCTIONS EVALUATED AT GROUP MEANS (GROUP CENTROIDS)

GROUP	FUNC 1	FUNC 2
1	-.71764	-.20546
2	-.03084	.38653
3	.74848	-.18107

TEST OF EQUALITY OF GROUP COVARIANCE MATRICES USING BOX'S M
THE RANKS AND NATURAL LOGARITHMS OF DETERMINANTS PRINTED ARE THOSE OF THE GROUP COVARIANCE MATRICES.

GROUP	LABEL	RANK	LOG DETERMINANT
1	M.I.	3	-3.876214
2	MEDICAL CONTROL	3	-4.190488
3	NORMAL CONTROL	3	-4.959863
	POOLED WITHIN-GROUPS COVARIANCE MATRIX	3	-3.944324

BOX'S M	APPROXIMATE F	DEGREES OF FREEDOM		SIGNIFICANCE
28.646	2.2424	12,	25122.5	.0080

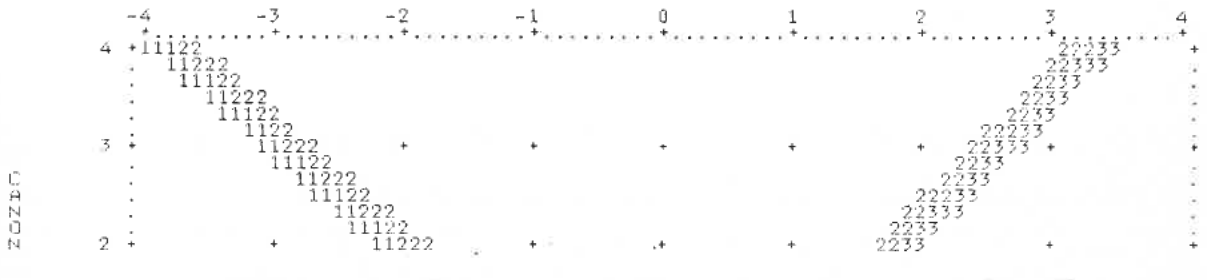
06 AUG 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:38:15 University of Natal SPERRY 1100/70 39R3E

SYMBOLS USED IN TERRITORIAL MAP

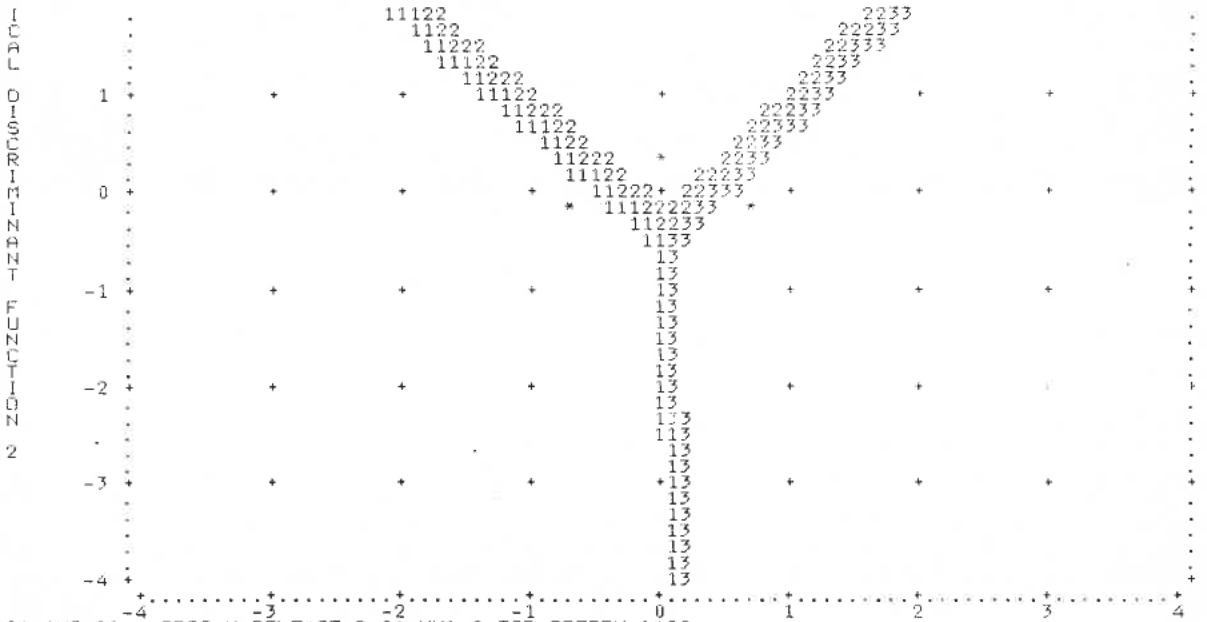
SYMBOL	GROUP	LABEL
1	1	M.I.
2	2	MEDICAL CONTROL
3	3	NORMAL CONTROL
*		GROUP CENTROIDS

06 AUG 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:38:17 University of Natal SPERRY 1100/70 39R3E

TERRITORIAL MAP * INDICATES A GROUP CENTROID
CANONICAL DISCRIMINANT FUNCTION 1



A NEW SELECTION OF PREDICTORS FOR DISCRIMINATING THE 3 GROUPS DATE 080691



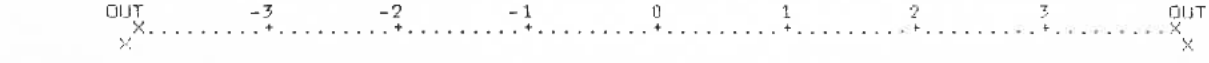
06 AUG 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:38:17 University of Natal SPERRY 1100/70 39R3E

SYMBOLS USED IN PLOTS

SYMBOL	GROUP	LABEL
1	1	M.I.
2	2	MEDICAL CONTROL
3	3	NORMAL CONTROL
*		GROUP CENTROIDS

06 AUG 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:38:18 University of Natal SPERRY 1100/70 39R3E

ALL-GROUPS SCATTERPLOT - * INDICATES A GROUP CENTROID
CANONICAL DISCRIMINANT FUNCTION 1



GROUP	2	25	5	16	4
MEDICAL CONTROL			20.0%	64.0%	16.0%
GROUP	3	25	3	9	13
NORMAL CONTROL			12.0%	36.0%	52.0%

PERCENT OF "GROUPED" CASES CORRECTLY CLASSIFIED: 57.33%

CLASSIFICATION PROCESSING SUMMARY
- 75 CASES WERE PROCESSED.
- 0 CASES WERE EXCLUDED FOR MISSING OR OUT-OF-RANGE GROUP CODES.
- 0 CASES HAD AT LEAST ONE MISSING DISCRIMINATING VARIABLE.
- 75 CASES WERE USED FOR PRINTED OUTPUT.

06 AUG 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:38:18 University of Natal SPERRY 1100/70 39R3E

PRECEDING TASK REQUIRED 3.28 SECONDS CPU TIME: 6.67 SECONDS ELAPSED.

```
  18  0          RECODE GROUP(1=2)
  19  0          NPAR TESTS M-W=STRESS EXP#ANGR EXPRESSD HYPERTEN BY GROUP(2,3)
THERE ARE    11002 WORDS OF MEMORY AVAILABLE.
THE LARGEST CONTIGUOUS AREA HAS    11002 WORDS.
 ***** WORKSPACE ALLOWS FOR     998 CASES FOR NPAR TESTS *****
```

06 AUG 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:38:20 University of Natal SPERRY 1100/70 39R3E

- - - - - MANN-WHITNEY U - WILCOXON RANK SUM W TEST

STRESS
BY GROUP

MEAN RANK	CASES	
35.72	50	GROUP = 2 MEDICAL CONTROL
42.56	25	GROUP = 3 NORMAL CONTROL
	75	TOTAL

U	W	CORRECTED FOR TIES Z	2-TAILED P
511.0	1064.0	-1.2859	.1985

- - - - - MANN-WHITNEY U - WILCOXON RANK SUM W TEST

EXP#ANGR
BY GROUP

MEAN RANK	CASES	
42.80	50	GROUP = 2 MEDICAL CONTROL
28.40	25	GROUP = 3 NORMAL CONTROL
	75	TOTAL

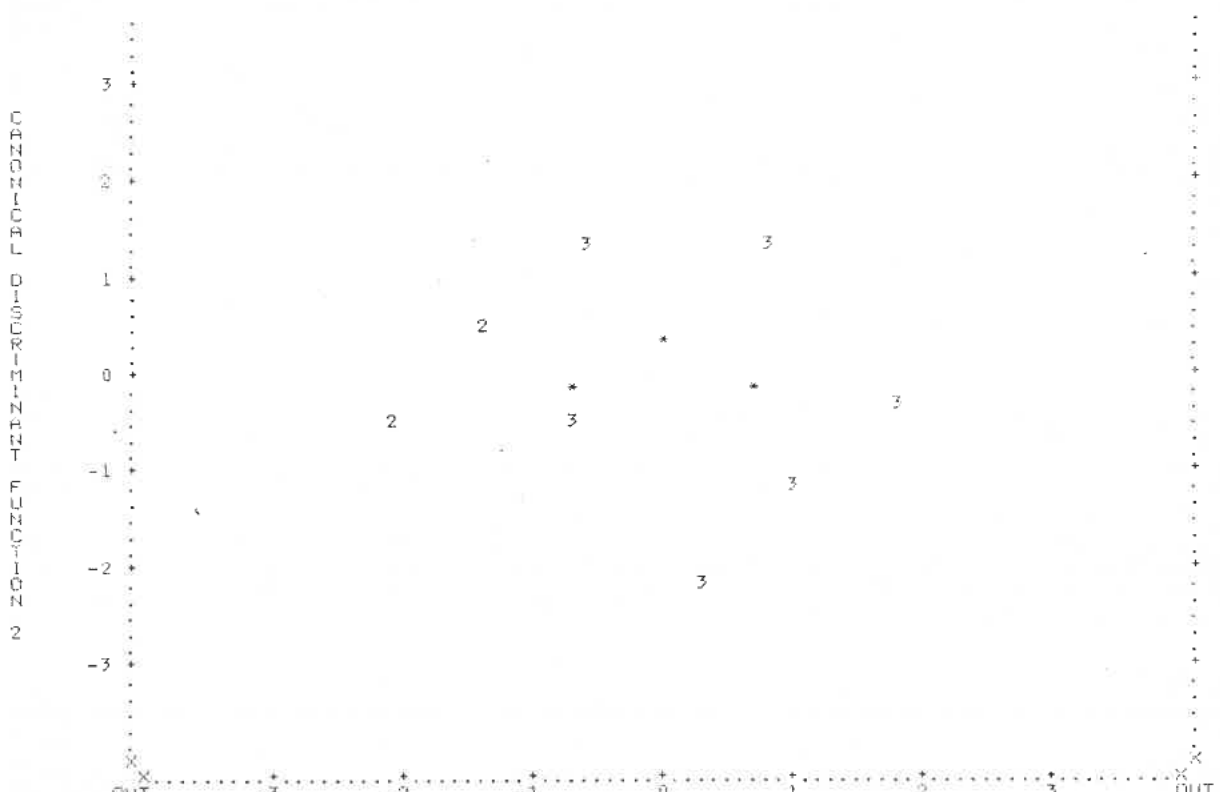
U	W	CORRECTED FOR TIES Z	2-TAILED P
385.0	710.0	-2.6984	.0070

- - - - - MANN-WHITNEY U - WILCOXON RANK SUM W TEST

EXPRESSD
BY GROUP

MEAN RANK	CASES

A NEW SELECTION OF PREDICTORS FOR DISCRIMINATING THE 3 GROUPS DATE 080691



06 AUG 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:38:18 University of Natal SPERRY 1100/70 39R3E

CLASSIFICATION RESULTS -

ACTUAL GROUP	NO. OF CASES	PREDICTED GROUP MEMBERSHIP 1	2	3
GROUP 1	25	14	7	4

3 NORMAL CONTROL 3 5.220229

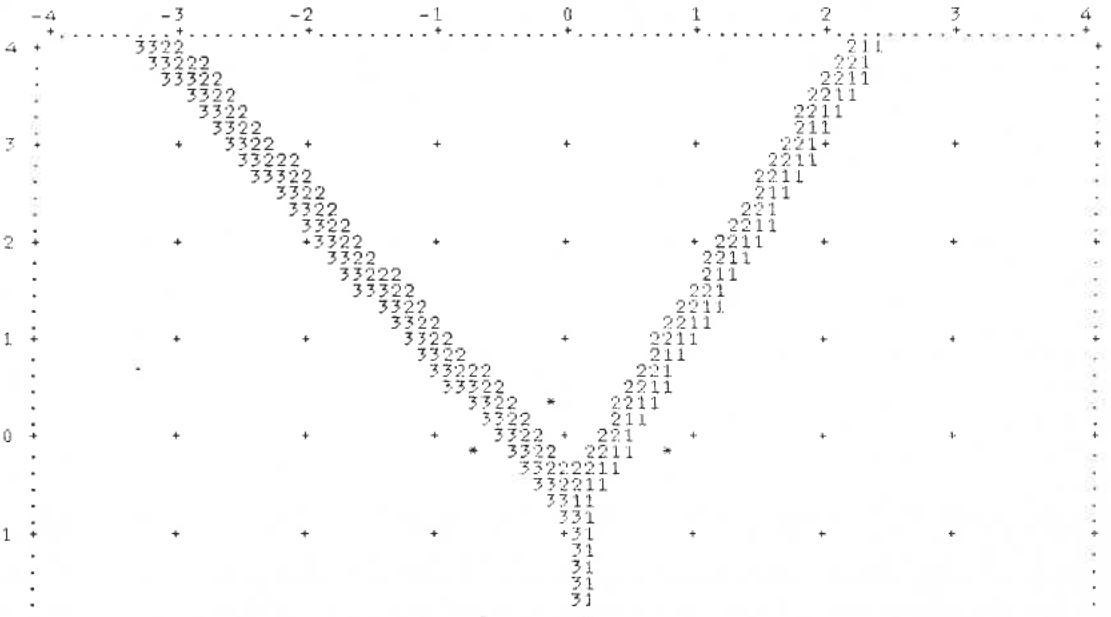
POOLED WITHIN-GROUPS COVARIANCE MATRIX 3 5.208931

BOX'S M	APPROXIMATE F	DEGREES OF FREEDOM		SIGNIFICANCE
29.942	2.0307	12.	25122.5	.0181

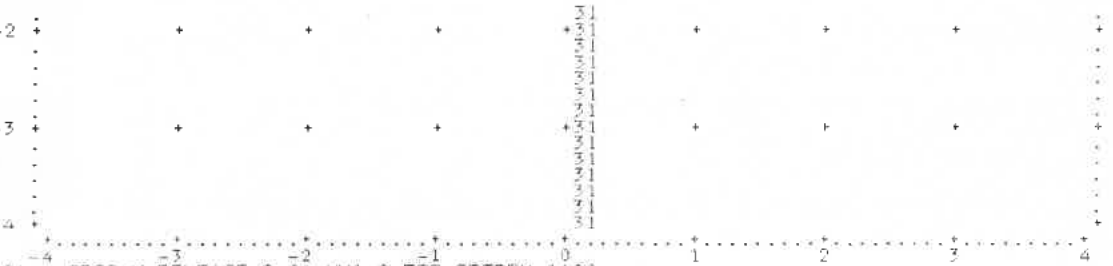
SYMBOLS USED IN TERRITORIAL MAP

SYMBOL	GROUP	LABEL
1	1	M.I.
2	2	MEDICAL CONTROL
3	3	NORMAL CONTROL
*		GROUP CENTROIDS

TERRITORIAL MAP * INDICATES A GROUP CENTROID
CANONICAL DISCRIMINANT FUNCTION 1



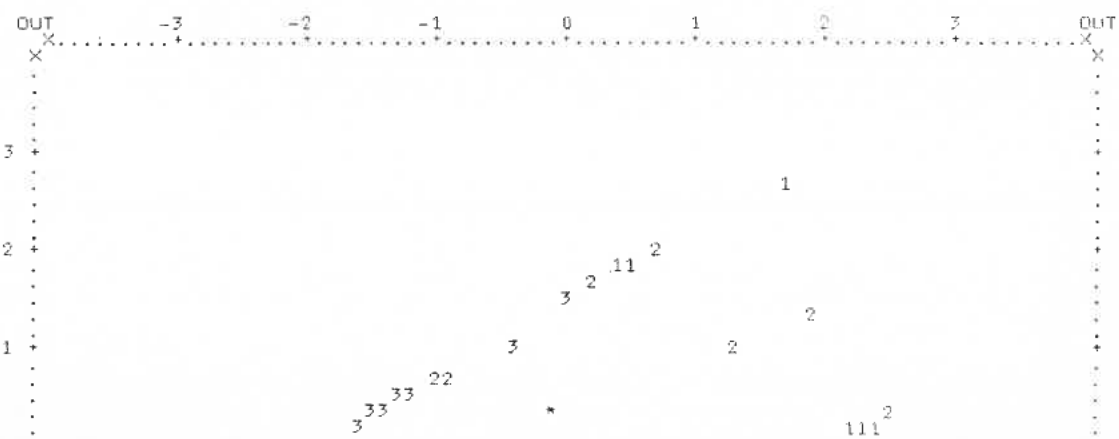
CROSSTABS INCLUING MIXED CASES, KRUSKAL-WALLISES AND DISCTRIMINAS DATE 073091 PA



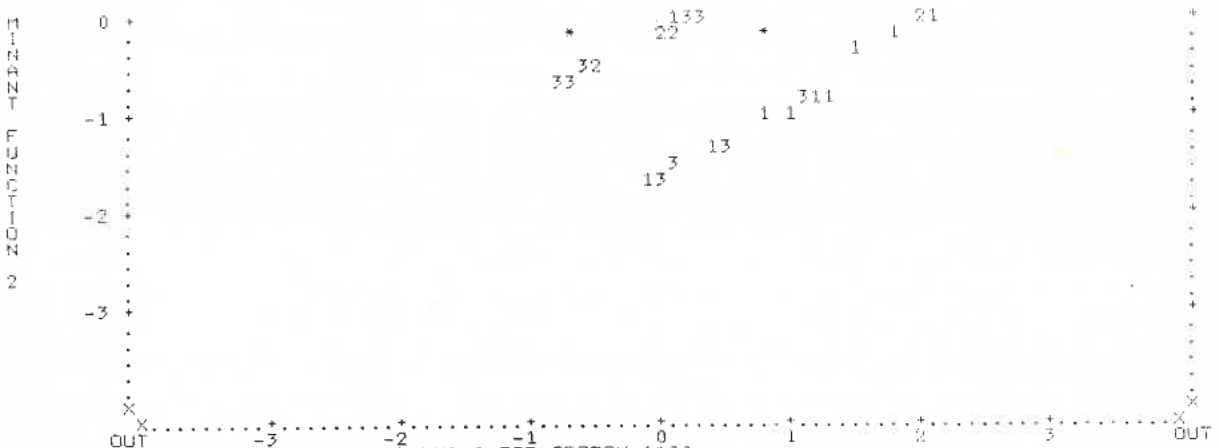
YMBOLS USED IN PLOTS

YMBOL	GROUP	LABEL
1	1	M.I.
2	2	MEDICAL CONTROL
3	3	NORMAL CONTROL
*		GROUP CENTROIDS

ALL-GROUPS SCATTERPLOT - * INDICATES A GROUP CENTROID
CANONICAL DISCRIMINANT FUNCTION 1



CROSSTABS INCLUING MIXED CASES, KRUSKAL-WALLISES AND DISCTRIMINAS DATE 073091



30 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:30:42 University of Natal SPERRY 1100/70 39R3E

CLASSIFICATION RESULTS -

ACTUAL GROUP	NO. OF CASES	PREDICTED GROUP MEMBERSHIP 1	2	3
GROUP 1 M.I.	25	14 56.0%	4 16.0%	7 28.0%
GROUP 2 MEDICAL CONTROL	25	6 24.0%	7 28.0%	12 48.0%
GROUP 3 NORMAL CONTROL	25	4 16.0%	4 16.0%	17 68.0%

PERCENT OF "GROUPED" CASES CORRECTLY CLASSIFIED: 50.67%

CLASSIFICATION PROCESSING SUMMARY
75 CASES WERE PROCESSED.
0 CASES WERE EXCLUDED FOR MISSING OR OUT-OF-RANGE GROUP CODES.
0 CASES HAD AT LEAST ONE MISSING DISCRIMINATING VARIABLE.
75 CASES WERE USED FOR PRINTED OUTPUT.

30 JUL 91 SPSS-X RELEASE 2.0A-UW1.0 FOR SPERRY 1100
08:30:47 University of Natal SPERRY 1100/70 39R3E

PRECEDING TASK REQUIRED 3.27 SECONDS CPU TIME; 6.37 SECONDS ELAPSED.

```
  24  0          FINISH
    24 COMMAND LINES READ.
     0 ERRORS DETECTED.
     2 WARNINGS ISSUED.
    11 SECONDS CPU TIME.
```

Chi Square Test for Two Independent Samples
(Taken from Siegel, 1956, p.107)

The following Chi-Square tests with 2x2 contingency tables were calculated according to the formula:

$$\chi^2 = \frac{N\,([AD-BC] - N/2)}{(A+B)(C+D)(A+C)(B+D)}$$

df = 1

Diabetes:

	MI	Norm	
Non-diabetic	18	23	41
Diabetic	7	2	9
	25	25	50

$$\chi^2 = \frac{500000}{230625}$$

$$= 2.16$$

With df = 1, p < 0.5(0.2) = p < 0.1.

Previous MI:

	MI	Norm	
No prev. MI	20	25	45
Previous MI	5	0	5
	25	25	50

$$\chi^2 = \frac{500000}{140625}$$

$$= 3.56$$

With df = 1, p < 0.5(0.05) = p < 0.025.

	MI+Med	Norm	
No prev. MI	41	25	66
Previous MI	9	0	9
	50	25	75

$$\chi^2 = \frac{2636718.8}{742500}$$

$$= 3.55$$

With df = 1, p < 0.5(0.05) = p < 0.025.

Hypertension:

	MI+Med	Norm	
Hypertension	31	24	55
No Hyperten.	19	1	20
	50	25	75

$$\chi^2 = \frac{11261719}{1375000}$$

$$= 8.19$$

With df = 1, p < 0.5(0.01) = p < 0.005.

Smoking:

	MI	Norm	
Smoker	20	16	36
Non-smoker	5	9	14
	25	25	75

$$\chi^2 = \frac{281250}{315000}$$

$$= 0.893$$

With df = 1, p < 0.5(0.7) = p < 0.35.

Obesity:

	MI	Norm+Med	
Obese	8	7	15
Non-obese	17	43	60
	25	50	75

$$\chi^2 = \frac{2636718.8}{1125000}$$

$$= 2.34$$

With df = 1, p < 0.5(0.1) = p < 0.05.

	MI	Med	
Obese	8	5	13
Non-obese	17	20	37
	25	25	50

$$\chi^2 = \frac{125000}{300625}$$

$$= 0.416$$

With df = 1, p < 0.5(0.8) = p < 0.4.