PREDICTING THE PSYCHOLOGICAL AND PHYSIOLOGICAL PROGNOSIS OF CARDIAC REHABILITATION PATIENTS

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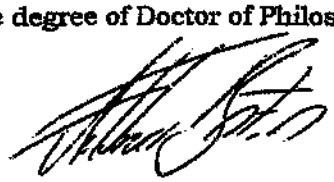
I hereby declare that this thesis is my own work and that I have not submitted it for the degree of Doctor of Philosophy to any other university.



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ABSTRACT

The large percentage of deaths attributed to recurrent Coronary Heart Disease (CHD) has generated a search for behavioural and psychological factors which mitigate the consequences of CHD. An aim of the present thesis is to extend this search by identifying factors which predict prognosis for recovery from CHD. Two key factors seen to influence prognosis are aerobic exercise and Type A behaviour. Extant research into the role of exercise has been hindered by methodological weaknesses. As a consequence, the precise means by which exercise influences CHD patients' psychological and physiological strain remains unclear. Similarly, the Type A literature has been flawed by the inaccurate conceptualisation and measurement of Type A behaviour as a global, and not a multidimensional, construct. Research which has examined the multidimensional nature of Type A behaviour has been restricted to low risk samples. Thus, a further aim of the present research is to examine the prognostic role of exercise and Type A components within the context of cardiac rehabilitation. In achieving this aim, three studies are conducted.

Utilising a controlled longitudinal design, the first study assesses whether exercise ameliorates the consequences of CHD by comparing the change in anxiety and depression and number of subsequent cardiac events of 20 cardiac patients who attended a six month exercise programme with a matched group of patients who had withdrawn from the same programme after admission. This study fails to yield any differences between groups. Use of attendance as the single measure of compliance may have been responsible for the absence of significant results. The methodological limitations of the study are discussed with suggestions as to how they may be overcome in future research focused on the prognostic role of exercise.

Before the independent and differential prognostic roles of the Type A components could be assessed, it was necessary to develop and test a questionnaire of Type A components, which reflects the behaviour of cardiac rehabilitation patients. To this end, the second study comprises the development and testing of the Type A Component Questionnaire on a sample of 217 cardiac rehabilitation patients. Confirmatory factor analysis of the 26 item Type A Component Questionnaire yields five independent factors, namely, achievement striving, impatience irritability, acting-out hostility, suppressed anger (anger-in) and expressed anger (anger-out). The Type A Component Questionnaire demonstrates satisfactory internal and temporal consistency. A separate sample of 31 CHD patients was used to determine the latter. Thus, the second study generates a valid and reliable psychometric measure of the Type A components relevant to cardiac rehabilitation patients' behaviour.

In the third study, a multiple regression model of exercise and Type A components is developed and tested. The model comprises the achievement striving, impatience irritability, acting-out hostility, anger-in and anger-out components contained in the Type A Component Questionnaire and the frequency and duration measures of exercise compliance. It is proposed that these seven dimensions of cardiac rehabilitation patients' behaviour will act as differential and independent predictors of CHD prognosis. The study is longitudinal in design and utilises a sample of 181 cardiac rehabilitation patients. The results of the study show that the Type A and exercise compliance components are differentially related to psychological and physiological outcomes as measured by change in anxiety, depression and maximal oxygen uptake (VO_2max). Specifically, anger-out and exercise duration are negatively associated with change in anxiety, while anger-in and anger-out are negatively associated with change in depression. Exercise duration is positively related to change in maximal oxygen uptake. As the sample experienced a reduction in anxiety and depression and an increase in maximal oxygen uptake

over the six months of study, these results are conceptually consistent. These results are discussed in terms of the relevant conceptual and empirical literature. Also discussed are the theoretical and practical implications of the study and the limitations thereof. Following this discussion a refined, transactional model of CHD patients' behaviour is proposed. The model integrates the findings of the present research with additional antecedents, covariates, moderator and outcome variables thought to be relevant to the process of adjustment to CHD. Finally, it is asserted that by testing the refined model, future research will achieve greater insight into the predictive role of Type A behaviour and exercise compliance within the context of cardiac rehabilitation.

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CHAPTER ONE

THEORY OF STRESS AND STRAIN

Coronary heart disease[1] (CHD) is a leading cause of premature disability and death in most developed countries (Booth-Kewley & Friedman, 1987; Palmer, Langeluddecke, Jones & Tennant, 1992). In developing countries, the disease accounts for an increasing number of deaths (World Health Organization Report, 1981). The 48 000 South Africans who contract the disease and the approximate 12 000 who die from it each year (Gordon & Gibbons, 1991), suggest that CHD has reached epidemic proportions in South Africa (Giese & Schomer, 1986).

In the past four decades, the prevalence of CHD has inspired a search among scientists and epidemiologists for physical and biological precursors of CHD (Ragland & Brand, 1988b). As a result, smoking, serum cholesterol, hypertension[2], Diabetes Mellitus[3], obesity, heredity, age and masculine gender have been established as CHD risk factors[4] (Kuller, 1976; Leon, Finn, Murray, & Bailey, 1988; Warren & Subak-Sharpe, 1984; Wright, 1992). However, these traditional risk

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1 *Coronary Heart Disease, a clinical condition which occurs as a result of degeneration of the coronary arteries (Friedberg, 1966), is symptomatically manifest as myocardial infarction and / or angina pectoris (Baker, Abbott, & Kannel, 1989). The term coronary heart disease encompasses a collection of disease endpoints (Friedman & Booth-Kewley, 1987). Underlined words are defined in subsequent footnotes.*

2 *Hypertension is the chronic elevation of blood pressure in the arteries which occurs with or without demonstrable organic origin (Diamond, 1982).*

3 *Diabetes Mellitus is a disorder of carbohydrate metabolism caused either by insufficient production or ineffective utilization of insulin (Bradley, 1988).*

4 *A risk factor is any feature of a given population or environment which enhances the probability of developing CHD (Krantz, Grunberg, & Baum, 1985).*

factors account for only half the number of patients afflicted by CHD (Wood, 1981). Therefore, attempts to identify risk factors have been extended to include psychosocial factors (Rosenman & Chesney, 1982). Research of the last three decades has implicated Type A behaviour as a risk factor (Evans, 1990; Ganster, Schaubroeck, Sime, & Mayes, 1991; Miller, Turner, Tindale, Posavac, & Dugoni, 1991; Warren & Subak-Sharpe, 1984; Wright, 1992). Type A behaviour is an action-emotion complex comprising competitive, time-urgent, impatient, hostile and aggressive behaviour (Friedman & Rosenman, 1974). Its antithesis, Type B behaviour, is characterised by the relative absence of these attributes (Jenkins, Rosenman & Friedman, 1967). It is generally accepted that the traditional risk factors and Type A behaviour predispose individuals to CHD (Leaf & Ryan, 1990; Warren & Subak-Sharpe, 1984). These factors do not, however, precipitate the actual coronary event[5] (Selye, 1976). In the final analysis, the precipitating factor is often an extreme physiological response to stress (Belshin 1990; Selye, 1976).

How stress influences the development, onset and recurrence of CHD will be examined in the following chapters. To provide a theoretical basis for this examination, the present chapter outlines the stress process. It will be argued in this chapter that traditional stress models, which assume strain to be the endpoint in the process, fail to recognise strain as a potential source of further stress. In this chapter it is concluded that CHD, traditionally regarded as a manifestation of strain, may also function as a source of stress. This conclusion renders traditional stress-strain models inappropriate in studies concerning the stressors and strains

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[5] *In the present research a coronary event or episode refers to a symptomatic manifestation of CHD, such as myocardial infarction or angina pectoris (Doyle Gentry, & Stewart, 1985).*

experienced by cardiac patients for they begin at the point of strain, namely, CHD diagnosis. The present research examines CHD as a source of stress and identifies the stressors and strains generated by CHD. It thus proposes a stress-strain model which is applicable to research on cardiac patients. Past research indicates that aerobic exercise and Type A behaviour influence the stress-strain process generated by CHD (Dembroski, MacDougall, Costa, & Grandits, 1989; Holmes & Roth, 1983; Morris, Clayton, Everitt, Semmence, & Burgess, 1990; Öhman, Nordby, & Svebak, 1989). Consequently, components of aerobic exercise and Type A behaviour are included in the model. Motivation for the inclusion of these variables in the model is preceded by a discussion of the stress process.

The Stress Process

Cox (1988) defines psychological stress as an imbalance between an individual's subjective interpretation of objective demands and his / her ability to adjust to these demands under circumstances where failure to adapt will have deleterious consequences. That is, where the perceived inability to meet objective demands constitutes a threat to the individual's state of well-being (Edwards & Cooper, 1990). Within this conceptualisation, these objective demands constitute stressors, the subjective interpretation is the experience of stress, and the adverse psychological, physiological and behavioural reaction thereto, signifies strain (Pratt & Barling, 1988). Each component of the process is distinct from the other (Leventhal & Tomarken, 1987). In terms of this conceptualisation, stress is a multifaceted and dynamic process of interaction between individuals and the circumstances effecting them (Hinton, Rotheiler, & Howard, 1991; House, 1974).

The extent to which an objective demand is stressful is determined by the individual's perception of the stressor (Duckworth, 1985). As perception is influenced by individual characteristics (Beehr & Newman, 1978), the stress experience can be described as a subjective reaction to the nature of the objective demand, as moderated by individual characteristics (House, 1974). Thus, it is the response to the stressor, and not the stressor *per se* which generates the stress process (Leventhal & Tomarken, 1987).

Stress generates psychological, behavioural and physiological responses (Cox, 1985). The focal variables in the present research, namely, Type A behaviour and compliance with an exercise programme, can be described as behavioural responses to stress. The initiation of a response can be involuntary (e.g., physiological reactivity) or deliberate (e.g., a change in behaviour such as complying with an exercise regimen or modifying the negative aspects of Type A behaviour) (Cox, 1985). Both types of response can be described as measures of coping.

Coping is an action-oriented and intrapsychic attempt to combat the impact of specific internal and external stressors which threaten the individual's sense of homeostasis by exceeding or overtaxing his or her resources (Cohen & Lazarus, 1979; Johnston, Weinman, & Marteau, 1990; Kliewer, 1991). It is a process which is initiated either in anticipation of a stressful event or as a response to an existing stressor (Cohen, 1987). Further, it occurs as a conscious response to situations perceived as psychologically stressful (Kliewer, 1991). Individuals said to be actively coping are those who take deliberate steps either to avoid or alter the source of stress, or to overcome their emotional response to stress (Lazarus, 1966).

Deliberate coping can be either instrumental or palliative (Brown, 1988). With instrumental coping, the individual manipulates the environment to remove or modify the source of stress (Bluen, 1986). In the case of palliative coping, the individual attempts to accommodate stress by adjusting his or her internal environment accordingly (Lazarus, 1966). While both instrumental and palliative coping strategies are frequently employed in tandem (Bluen, 1986), a palliative response may be the only option in situations where the objective condition cannot be altered (Mechanic, 1962). Examples of such situations include terminal illness or acute anxiety.

Under circumstances that allow the individual to affect change, the choice of coping strategy will depend on the interaction between objective demands (e.g., CHD diagnosis) and individual characteristics (e.g., components of Type A behaviour) which influence the perception of the threats associated with the demands (Cooper & Marshall, 1976; French & Caplan, 1972; Johnston et al., 1990). Failure to summon the necessary resources to implement a coping strategy which effectively manages the objective demand will ultimately result in strain. Individuals less able to adapt, and consequently less able to cope with stress, are more likely to demonstrate psychological, physiological and behavioural manifestations of strain which culminate in disease (Fava et al., 1992; Taylor & Cooper, 1989). The relationship between stress and the development of disease is outlined below.

It is widely believed that numerous forms of psychosocial stress, be they occupational, interpersonal, or circumstantial, precipitate the development of disease (Edwards & Cooper, 1990; Kamarck & Jennings, 1991; Marmot & Madge, 1987; Russek, King, Russek, & Russek, 1990). The experience of stress is presumed to facilitate disease development by generating complex and potentially damaging, neural, hormonal and metabolic responses (Baum, Grunberg, & Singer, 1982). These responses result in a temporary disruption of physiological homeostasis (Godaert, 1990). Through activation of appropriate adjustment mechanisms, homeostasis is restored (Cohen & Lazarus, 1979). However, extreme or protracted stressors which impose too great a demand on adaptive mechanisms can result in an inappropriate stress response which, in turn, can lead to structural damage (Godaert, 1990). Chronic stress thus has the potential to transform relatively benign events into manifestations of psychological and physiological ill health (Krantz et al., 1985).

CHD is an example of such a manifestation and is frequently classified as a stress disease (Quick & Quick, 1984). The excessive physiological responses associated with behavioural stressors have been proposed as important factors in the pathogenesis of CHD (Suarez, Williams, Kuhn, Zimmerman, & Schanberg, 1991). Direct relationships between single stressors or stressful events and fatal and non-fatal myocardial infarction[6] have been established (Kamarck & Jennings, 1991;

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[6] *Myocardial infarction is the death, and subsequent build-up of scar-tissue, of a circumscribed area of heart muscle (i.e., myocardium) and is the result of an obstructed coronary artery's inability to supply the myocardium with blood (Stedman's Medical Dictionary, 1961).*

Selye, 1976; Willich et al., 1993). The manifestation of CHD is dependent on both acute precipitating factors and the degree of underlying atherosclerosis[7] (Matthews, 1988). An acute precipitating factor, such as the psychological response to stress, can initiate the neurophysiological activity which triggers a coronary event (Mittleman et al., 1993; Skinner, 1985; Willich et al., 1993).

Stress promotes the development of atherosclerosis through traditional risk factors. That is, stress raises blood pressure[8], heart rate[9] and cholesterol levels, and facilitates the physiologically unjustified release of potentially damaging catecholamines[10] all of which increase the rate of atherosclerotic development (Glass, 1977a; Johnston et al., 1990; Rozanski, Krantz, & Bairey, 1991; Specchia et al., 1991). Stress exerts a further negative influence through its association with Type A behaviour (Howse, 1974; Janisse & Dyck, 1989). Type A's are more susceptible to experiencing stress and demonstrate a more negative psychological and physiological stress response than Type B's (Brief, Rude, & Rabinowitz, 1983;

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7 *Atherosclerosis is the build-up of fatty deposits under the lining (intima) of the arteries which ultimately results in arterial occlusion and hence, impeded blood flow (Stedman's Medical Dictionary, 1961). Advanced atherosclerosis in the arteries of the heart impairs the supply of oxygen to the heart muscle which can lead to myocardial infarction or angina pectoris (Booth-Kewley & Friedman, 1987).*

8 *Blood pressure is the pressure of blood against arterial walls. Systolic blood pressure refers to the measurement of pressure during the rhythmical contraction of the heart which drives the blood through the aorta and pulmonary artery to the rest of the body (Stedman's Medical Dictionary, 1961). Diastolic blood pressure alternates with systolic blood pressure. During the diastolic phase, the heart chambers dilate to accommodate an influx of blood. Both diastolic and systolic pressure measures are of prognostic importance (Godaert, 1990).*

9 *Measurements of the heart rate provide a general physiological indication of the internal milieu, and a specific indication of psychological or emotional stress (Strasser, 1981).*

10 *Catecholamines comprise a class of transmitter substances which fulfill a vital role in the functioning of the central and sympathetic nervous systems (Carlson, 1981). Two of the primary catecholamines are epinephrine and norepinephrine (Carlson, 1981).*

Janisse & Dyck, 1989). Type A behaviour has also been empirically linked to the development of atherosclerosis (Blumenthal, Williams, Kong, Schanberg, & Thompson, 1978). Given that stress can act as an acute precipitating factor and also promote the development of atherosclerosis, CHD can be described as a manifestation of the strain arising from the physiological response to psychosocial stressors.

CHD: A Strain and Potential Source of Stress

Within the field of stress research, numerous attempts have been made to develop theoretical models which explain and predict the stress process (Antonovsky & Sagy, 1985). Many of these models can be criticised for terminating analysis of the stress process at the point of strain (e.g., House, 1974; Steinberg & Ritzman, 1990). Strain has the potential to generate further stress and render individuals vulnerable to other sources of stress by reducing their capacity for coping (Cox, 1988). It is possible that strain may act as a precursor of secondary stress and strain (Rawson, Bloomer & Kendall, 1994). Therefore, it is necessary to extend the stress process beyond the point of strain.

In the context of CHD, subjective responses to a CHD diagnosis may aggravate the progression of disease (Contrada & Krantz, 1987). When combined with reduced coronary reserves, the increases in oxygen demand, blood pressure and heart rate associated with the stress response may precipitate cardiac abnormalities in CHD patients (Specchia et al., 1991). The frequent occurrence of such abnormalities may advance the progression of CHD. This aggravation may occur through deleterious

physiological reactions to stress or through psychological reactions which generate negative behavioural responses to the stress implicit to the diagnosis of a life threatening disease (Contrada & Krantz, 1987; Terry, 1992). It is thus important to consider CHD as both a manifestation of physical strain, as traditionally regarded in the field of stress research (e.g., Holmes & Masuda, 1974; Kasl, 1983; Leventhal & Tomarken, 1987; Rosenman & Chesney, 1982) and as a source of psychological and physiological stress (Rejeski, Morley, & Sotile, 1985).

Based on the premise that CHD generates stress which in turn contributes to further strain, the present research examines psychological responses to the stress of CHD and the extent to which they culminate in strain. It is proposed that certain dimensions of cardiac patients' Type A behaviour will compound the experience of stress, and consequently contribute to subsequent psychological and physiological strain. A further proposition is that other dimensions of behaviour such as compliance with an exercise programme will reduce the incidence of strain, and hence, facilitate recovery. The following chapter explores CHD as a source of stress and subsequent strain.

CHAPTER 2

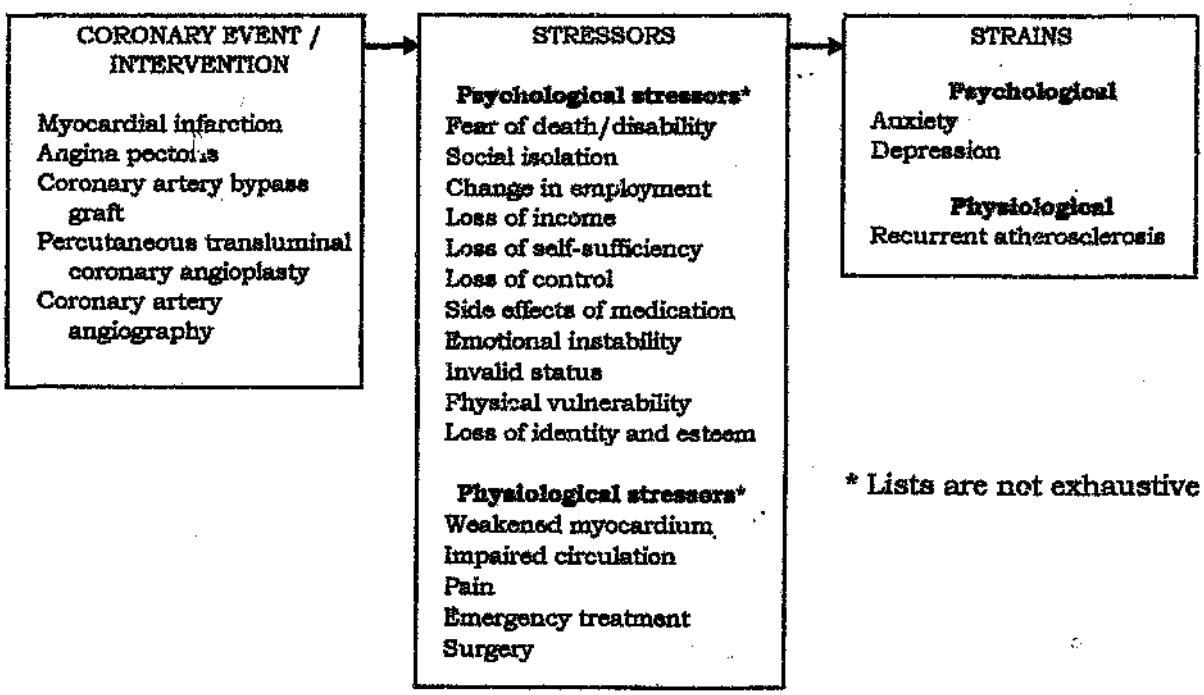
STRESSORS AND STRAINS OF CORONARY HEART DISEASE

The Stress of CHD Diagnosis

A coronary event constitutes an objective stressor (Rejeski et al., 1985; Terry, 1992). It threatens individuals' lives, their physical health, self-image, beliefs, social functioning and emotional stability (Cohen, 1981). In other words, it imposes psychological and physiological demands which require significant adjustment under circumstances where failure to adjust will have negative consequences (Cohen, 1981). Thus, though it is, in itself, a manifestation of strain, CHD is also the source of subsequent stressors and strain. The notion that CHD generates stressors and strain is conceptualised in the model illustrated in Figure 2.1.

Figure 2.1

Model of The Stressors and Strains of CHD



The psychological and physiological demands imposed by a coronary event start with the actual incidence of a myocardial infarction or angina pectoris and persist long into recovery. While some patients may experience the relevant stressors at different times, the sequence of stressors outlined below is generally representative of cardiac patients' experiences (*cf.* Erdman, 1990; Rejeski et al., 1985).

During the coronary episode, the immediate psychological stressor confronting the individual is the fear of death, while the physiological stressor is the body's adjustment to a weakened myocardium[11] and the decreased supply of oxygen resulting from impaired blood circulation (Rejeski et al., 1985). In the period directly following the episode, the individual is presented with a bewildering sequence of stressful events. These include the frightening experience of persistent and intense pain, the race to the hospital, the apparently frenetic activity of medical personnel, and emergency treatment or surgery, followed by imposed inactivity and loss of independence (Cay, Zaitsev, & Rudnicki, 1985; Erdman, 1990; Johnston, 1988). The acute nature of these events require rapid activation of adjustment resources (Danilewitz, 1987). However, the speed with which they occur prohibits patient adjustment and provides alarming evidence of the gravity of the patient's condition (Cay et al., 1985). Typically, these events generate considerable fear which not only concerns the prevailing fear of death but also the perceived impact the coronary event will have on subsequent lifestyle (Rejeski et al., 1985). Persistent symptoms,

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11 *The Myocardium is "...the middle of the three layers forming the wall of the heart. It is composed of cardiac muscle and forms the greater part of the heart wall..." (Oxford Reference Concise Medical Dictionary, III Edition, 1990, p. 450).*

shortness of breath, real or imagined angina pectoris[12], and overall physical weakness in the days following the episode may convince the patient that his or her disease is unalterable and permanent (Cay et al., 1985; Hackett & Cassem, 1975). However, the fears and beliefs generated during this period are so potent and overwhelming that the patient often denies their existence (Erdman, 1990).

The transition from high to medium hospital care places a new set of psychological demands on the cardiac patient (Erdman, 1990; Rejeski et al., 1985). With the immediate threat removed, the patient has more time and psychological resilience to reflect on the real significance of his or her recent cardiac experience (Hackett, 1985). On reflection, the patient realises the potentially fatal nature of his or her coronary episode (Erdman, 1990). For most, the recent confrontation with death is perceived as a personal attack (Erdman, 1990). This perception generates an imbalance between the patient's psychological resilience and the load imposed upon him or her (Hackett, 1985). The imbalance renders the patient vulnerable to the psychological stressors associated with the period of recovery which begins in the hospital and continues long after discharge.

In the recovery period, the constraints of structured hospital care are reduced or removed and the patient is compelled to assume personal responsibility for his or her condition (Rejeski et al., 1985). It is primarily during this period, that the

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[12] *Angina pectoris refers to brief and often recurrent pain in the center of the chest which occurs when the heart's demand for blood exceeds the coronary arteries' supply, invariably as a result of coronary artery degeneration (Oxford Reference Concise Medical Dictionary, III Edition, 1990). Angina pectoris is induced by exertion or emotion and relieved by rest or nitroglycerin (Eaker, Abbott, & Kannel, 1989). Nitroglycerin serves to restore blood flow by dilating the blood vessels of the heart.*

patient is confronted with the practical implications of his or her disease. If the practical implications of disease are perceived to be insurmountable, they are likely to be experienced as sources of stress (Froelicher, Myers, Follansbee, & Labovitz, 1993).

The psychological stressors during recovery are numerous and vary greatly between individuals. At the centre of these is still the fear of sudden death or reinfarction (Rejeski et al., 1985; Trelawny-Ross & Russel, 1987). While the threat of a second cardiac event may be imaginary (Budnick, 1991), the patient's possible change in employment, family and physical status and functioning is real and imposes additional stressors. The stressors generated by a change in employment include loss of income (Budnick, 1991; Rejeski et al., 1985) and the social isolation derived from reduced contact with colleagues and friends (Ruberman, Weinblatt, & Goldberg, 1984). Within the family structure, a loss of self-sufficiency and control and the family's perception of the patient as an invalid may constitute additional stressors (Budnick, 1991; Rejeski et al., 1985). So too may the side effects of drug therapy, such as insomnia, sexual dysfunction, depression, and mental confusion constitute additional stressors (Budnick, 1991; Rejeski et al., 1985). The patient may also experience psychological stressors in the form of sudden phobias, loss of concentration, emotional instability, unfamiliar feelings of powerlessness and hopelessness (Erdman, 1990) and an overriding loss of identity and esteem (Budnick, 1991; Froelicher et al., 1993; Wilson-Barnett, 1979).

Given the advances made by medical science and the sophistication and availability of rehabilitation, many of these stressors need not be chronic (Smith & O'Rourke,

1988). Despite this, many patients continue to perceive their condition as stressful, and experience the concomitant cardiovascular responses to stress (Smith & O'Rourke, 1988). The cardiovascular and neuroendocrine[13] responses to chronic CHD stressors may disrupt physiological functions and either repress normal bodily defences or nurture pathogenic change (Cox, 1985). Thus, the persistent experience of stress can contribute to a negative prognosis for recovery.

The Stress of CHD: Empirical Evidence

From past research it is evident that CHD is a profound stressor and that the resulting psychological disturbances persist long after physical recovery (Terry, 1992). Lloyd and Cawley (1982) observed that 65% of patients admitted to a coronary care unit had no history of psychological disorders and remained psychologically unaffected by their coronary event after one week of hospitalisation. However, four months later, 19% of these patients had developed psychological disorders (Lloyd & Cawley, 1982). Eleven percent of the patients who were affected at the four month follow-up were still experiencing psychological disorders 12 months after the initial event. Thus, there is evidence that psychological disorders continue after the CHD event and are not necessarily influenced by patients' premorbid personality (Terry, 1992).

The lack of premorbid influence is apparent when comparing retrospective and prospective studies (Contrada & Krantz, 1987). For example, the prospective

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[13] *Neuroendocrine reaction refers to "the system of dual control of certain activities of the body by means of both nerves and circulating hormones." (Oxford Reference Concise Medical Dictionary, III Edition, 1990, p. 464).*

Belgian-French Pooling Project (1984), which evaluated psychological health prior to the onset of disease, failed to find an association between myocardial infarction and psychological distress. The retrospective study of Thiel, Parker and Bruce (1973), on the other hand, established a relationship between the diagnosis of CHD and psychological distress. The comparison between prospective and retrospective studies suggests that post-coronary distress can be attributed to the coronary event itself and not, to pre-existing levels of stress.

There is also evidence to suggest that post coronary stress initiates deleterious cardiovascular reactivity[14] (Lassner, Matthews, & Stoney, 1994). Rozanski et al. (1988) assessed whether there was a causal relationship between laboratory induced acute mental stress and transient myocardial dysfunction among patients recovering from a coronary event. Of the 39 patients assessed, 23 (59%) experienced transient, functional myocardial abnormalities when subjected to acute stress. Similar results were yielded in a controlled study by Giubbini et al. (1991). In this study, 20 of the 24 myocardial infarction patients subjected to mental stress demonstrated cardiac abnormalities. Conversely, mental stress did not induce cardiac abnormalities in the 25 healthy subjects (Giubbini et al., 1991). Stress induced myocardial abnormalities can precipitate reinfarction (Dimsdale, 1977; Engel, 1971; Rozanski et al., 1988). This phenomenon is attributed to the

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[14] *Cardiovascular reactivity reflects the changes in cardiac and vascular responses (e.g., heart rate, blood pressure) between resting physiological levels and levels induced by intrinsic and extrinsic stimulation (Melamed, Harari, & Green, 1993; Vitaliano, Russo, Bailey, Young, & McCann, 1993). It is established by correlating the independent variable under examination (e.g., Type A behaviour) with changes in the above measures of cardiovascular arousal which occur in response to stressors (Ganster et al., 1991). Cardiovascular reactivity is considered to be stable over time and over a variety of conceptually relevant stressors (Lassner et al., 1994).*

compromised condition and function of the coronary arteries which results in vasoconstriction when patients experience mental stress (Rozanski et al., 1991). Vasoconstriction of the arteries results in ischaemia[15] (Rozanski et al., 1991). Indeed, in Specchia et al.'s (1991) sample of CHD patients, there was a greater occurrence of CHD fatalities, and non-fatal cardiac events among patients who demonstrated a heightened cardiovascular response to mental stress than among patients who did not demonstrate a hyperreactive response to stress. Thus post coronary stress has the potential to induce both persistent psychological distress and physiological dysfunction. Within the context of cardiac rehabilitation, post coronary stress has been found to induce anxiety and depression (Brown & Munford, 1983). Post coronary stress is also implicated in the further degeneration of the cardiovascular system as measured by maximal oxygen uptake. These forms of psychological and physiological strain hinder recovery from a cardiac event (Brown & Munford, 1983; Sami, Kraemer, & DeBusk, 1979). As an aim of the present research is to identify factors which predict prognosis for recovery, anxiety, depression and maximal oxygen uptake are discussed in greater detail below.

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[15] *Ischaemia refers to the insufficient flow of blood to a particular section of the body which is caused by constriction or obstruction of the supplying blood vessels (Oxford Reference Concise Medical Dictionary, III Edition, 1990). Myocardial ischaemia implies a deficiency of oxygen in a localized section of heart muscle caused by transient decreases in blood supply or increases in oxygen demand, or both (Kamarck & Jennings, 1991). Angina pectoris, and ultimately, myocardial infarction, occur as a result of ischaemia (Kamarck & Jennings, 1991; Oxford Reference Concise Medical Dictionary, III Edition, 1990).*

Psychological Strain

Post coronary anxiety and depression are frequently categorised as manifestations of the strain arising from coronary induced stress (e.g., Brown & Munford, 1983; Stern, Pascale, & McLoone, 1976). Indeed, some (e.g., Budnick, 1991; Cassem & Hackett, 1977; Gentry, Balder, Oude-Weme, Musch, & Gary, 1983; Terry, 1992) maintain that the emotional response to a coronary event is so predictable that coronary patients can expect to experience anxiety, followed by denial and depression, before reaching a state of psychological health. Psychological health is not, however, a given. Anxiety and depression following a coronary event are pervasive and are described as the most formidable problems confronting cardiac rehabilitation patients (Hackett & Cassem, 1975). Moreover, several studies have indicated that anxiety and depression are the most frequent determinants of persistent invalidism (Brown & Munford, 1984). For example, in a study conducted by Cay, Vetter, Philip and Dugard (1972), 56% of the sample reviewed remained depressed or anxious a year after their initial coronary event. In a follow up study, Singh, Singh, Singh, Singh, and Malhotra (1970), found that 34% of coronary patients continued to be anxious or depressed two years after their coronary event. Anxiety and depression exert a deleterious physiological effect (Garrity & Klein, 1975) and have the potential to hinder the rehabilitative process (Brown & Munford, 1984). Therefore, it is important that the anxiety and depression affecting cardiac patients be examined.

Anxiety

Anxiety is an emotional condition which is experienced as a debilitating state and characterised by tension, guilt-proneness, emotional instability, restlessness and suspicion (Schomer, 1985). Central to anxiety is the apprehension or dread of losing something which is perceived by the individual to be intrinsic to his / her existence as a personality (Drever, 1958; DSM-III, 1980). Anxious individuals frequently show a pervasive demoralisation which reduces self-esteem to such an extent that effective coping is impeded (Klein, 1981). A related impediment is the all encompassing psychological distress caused by anxiety which prevents the individual from concentrating on anything outside of his / her state of anxiety (Mathews, 1990). Often, anxiety is reflected in increased perspiration, respiration, blood pressure and heart rate (Budnick, 1991; Groenman, Vlaeyen, van Eek, & Schuerman, 1990). This hyperreactivity reflects the high level of vigilance concerning potential sources of danger and is characteristic of an anxious state (DSM-III, 1980; Mathews, 1990). Though anxiety frequently lacks a specific and realistic cause (Hackett & Cassem, 1978), it is often a product of stress (Dobson, 1982; Endler & Parker, 1990; Selye, 1976).

Anxiety as an outcome of CHD.

Anxiety following a coronary event is typically engendered by the fear of recurrence and its attendant threat of sudden death, persistent pain, the attributes the patient assigns to this pain (Groenman et al., 1990), the process of hospitalisation (Walding, 1991) and fears concerning the inability to return to work and maintain financial

stability and status (Cay et al., 1972; Wilson-Barnett, 1979). Anxious reactions to a coronary event tend to persist long after recovery (Guiry, Conroy, Hickey, & Mulcahy, 1987) and reflect a failure to adjust to the consequences of CHD (Garrity & Klein, 1975). Poor adjustment behaviour may be implicated in a negative prognosis (*cf.* Garrity & Klein, 1975). Anxiety is pervasive among cardiac patients (Brown & Munford, 1983). Guiry et al.'s (1987) review of 264 cardiac patients who had experienced a myocardial infarction a year previously revealed that 35.2% still suffered anxiety. In a sample of 63 myocardial infarction patients, Stern et al. (1976) found that 30% of patients had clinically significant anxiety six weeks after the infarction. Over 16% of the sample were still clinically anxious at the one year follow-up.

Other research has revealed an association between the failure to adjust to the consequences of CHD, as manifested by anxiety, and reinfarction. For example, the research of Garrity and Klein (1975), shows that defective adjustment behaviour following a coronary event significantly predicts the occurrence of reinfarction six months after the initial event. Garrity and Klein's results are supported by research detailing the relationship between psychological disorders and CHD mortality (e.g., Bruhn, Chandler, & Wolf, 1969; Segers & Mertens, 1977). It is apparent that anxiety is a typical response to a primary coronary event and endures long after the immediate threat has been removed. Its persistence reflects a failure to rationalise the threat of recurrence and the associated fear. Prolonged failure to rationalise these emotions may in turn, contribute to reinfarction (Rejeski et al., 1985). Therefore, anxiety is considered a manifestation of strain arising from the stressors intrinsic to CHD which has the potential to exert deleterious physiological and

psychological consequences. The present research measures the anxiety of a sample of cardiac patients following six months of cardiac rehabilitation and identifies the extent to which it is effected by components of aerobic exercise and Type A behaviour.

Depression

Depression is a pattern of affects, cognitions and behaviours and not a discrete entity (Gilbert, 1991). It is manifested by an abnormal state of excessive melancholy which is accompanied by loss of spontaneity and feelings of pessimism, nihilism, uncertainty, inadequacy and hopelessness (Beck, 1970; DSM-IV, 1994; Oxford Dictionary, 1982). Central to depression is the loss of enjoyment or interest in practically all usual activities (DSM-III, 1980). These features of depression can vary in intensity and combination (Gilbert, 1991). Depression is further associated with a generalised expectation that life outcomes are uncontrollable (Hildebrand-Saints & Weary, 1989). In terms of the learned helplessness model of depression, the underestimation of personal control over events can be attributed to depressives' distorted perception of response-outcome relationships (Seligman, 1975). The traditional model of mental health holds that depression stems from distortions of reality, cognitive biases and irrational beliefs (Tang & Critelli, 1990). Similarly, in his cognitive model of depression, Beck (1967; 1987) argues that the pessimistic view depressives take of themselves and the world is a product of an illogical interpretation of reality.

According to Selye (1976), depression is a primary response to stress. As a response to stress, depression may represent a positive coping mechanism. Depression may facilitate detachment from a stressful situation and thus provide an opportunity for individual growth and insight (Gut, 1989). Conversely, depression can be so debilitating as to prevent the depressive from making sense of his / her stress and consequently, pre-empt constructive coping (Endler & Parker, 1990; Gut, 1989). Therefore, depression can be both productive and counterproductive.

Depression as an outcome of CHD.

Depression is a typical response to the stress of a cardiac event (Brown & Munford, 1983). Cardiac patients' depression originates from fears concerning a perceived inability to function normally (Budnick, 1991; Wilson-Barnett, 1979) and a belief that the cardiac event will limit the amount of control they exert over their lives (Finnegan & Suler, 1985). A further source of depression among CHD patients is the belief that numerous changes are required and that they lack the resources or support necessary to implement them (Budnick, 1991). These fears and beliefs may be coupled with a loss of confidence, decreased libido, threat of dependency, sense of uselessness and insecurity, and a diminished sense of integrity (Cay et al., 1985; Hackett & Cassem, 1982). The loss of esteem resulting from imposed or imagined invalidism generates further depression which may contribute to reinfarction (Brown & Munford, 1984). Depression also disrupts CHD patients' physical, social and occupational activities and is associated with the physiologically unjustified stress and fear related to invalidism (Finnegan & Suler, 1985).

Severe post coronary depression is pervasive and affects over a third of cardiac patients (Guiry et al., 1987). While a spontaneous reduction in depression is likely to occur within six months of a coronary event, a substantial number of patients continue to be depressed after the six month to one year follow-up (Mayou, Foster, & Williamson, 1978; Stern et al., 1976). Kavanagh, Shephard and Tuck (1975) assessed the incidence of depression among 96 cardiac patients after 16 to 18 months of participation in a cardiac rehabilitation programme. The results indicated that 36% of the sample were severely[16] depressed while 25% were moderately depressed. The incidence of hypertension, elevated blood pressure and angina was greater among the severely depressed group (Kavanagh et al., 1975). Thus, depression was associated with negative health outcomes. A four year follow-up of 44 of the severely depressed patients found that 22 were still suffering severe depression (Kavanagh, Shephard, Tuck, & Qureshi, 1977). Twenty two percent of Stern et al.'s. (1976) sample of 68 cardiac patients reported symptoms of depression both at six weeks and six months after their myocardial infarction. After a year, significant depression was still evident in 70% of these patients (Stern et al., 1976). As a product of the numerous stressors of CHD, depression is a pervasive and persistent psychological manifestation of strain. The present research will identify the existence of depression following six months of participation in a cardiac rehabilitation programme. It will also examine the extent to which cardiac patients'

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[16] *Subjects were classified as severely depressed when their scores were in excess of 70 units (2 standard deviation points above the expected value) on the D scale of the Minnesota Multiphasic Personality Inventory (MMPI) (Kavanagh et al., 1975). In theory, scores in excess of 70 units on the D scale of the MMPI should only be achieved by 2.5% of the population (Kavanagh, Shephard, & Kennedy, 1977). Subjects with scores between 60 and 70 units were classified into an 'intermediate' or moderately depressed group (Kavanagh et al., 1975).*

depression is ameliorated or exacerbated by components of aerobic exercise and Type A behaviour.

Physiological Strain

Complications of underlying atherosclerosis form the foundation of CHD (Rosenman & Chesney, 1982). While there are numerous means of limiting the effects of atherosclerosis, persistent stress may result in the recurrent development of atherosclerosis. As will be discussed, the existence of atherosclerosis is indicated by maximal oxygen uptake. This discussion is preceded by a description of the process of atherosclerotic development. Atherosclerosis is initiated by the formation of a fatty sheen, comprised of cholesterol and fat, around lesions in the elastic layers of the arterial walls (Budnick, 1991; Goldberg, 1978). In time, the fatty sheen bursts, releasing its contents. Connective tissue is then generated to encompass the damaged area (Goldberg, 1978). Termed fibrosis, this process culminates in the formation of plaque, which extends into both the lumen and wall of the artery (Goldberg, 1978; Kamarck & Jennings, 1991). A build up of plaque ultimately deprives tissue of blood and, therefore, results in angina pectoris or myocardial infarction (Goldberg, 1978). Alternatively, the plaque may rupture and release debris and blood clots into the bloodstream, which may in turn, result in coronary thrombosis[17] (Goldberg, 1978; Kamarck & Jennings, 1991). Rupture of the plaque is thought to occur in response to acute vascular or blood pressure changes (Millar-

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[17] *Coronary Thrombosis occurs when a clot comprising blood and lipid elements occludes the coronary arteries (Kamarck & Jennings, 1991). The formation of the blood clot is invariably the result of degeneration of the arterial walls (Stedman's Medical Dictionary, 1961).*

Craig, Bishop, & Raftery, 1978). In turn, these changes are often a response to stress (Carlson, 1981).

A number of curative measures are employed to attenuate the effects of atherosclerosis. These include, diverting blood-flow from an occluded coronary artery to a vein which has been surgically grafted onto the heart (i.e., coronary artery bypass graft[18]), non surgical dilation of obstructed vessels (i.e., percutaneous transluminal coronary angioplasty[19]) and medication (Orha, Nikolaeva, & Velasco, 1985). These measures are curative rather than preventative and atherosclerosis may recur if the factors initiating its development persist. An example of this phenomenon is the persistent experience of stress. That is, stress generates extreme increases in the discharge of catecholamines and the reactivity of the cardiovascular system (Carlson, 1981; Matthews, 1988) which perpetuate the development of atherosclerosis (Eliot, 1979). Thus, if stress persists, then physiological strain, as indicated by atherosclerotic development, is likely to recur. The existence of atherosclerosis can be determined by measuring maximal oxygen uptake. The relationship between atherosclerosis and maximal oxygen uptake is outlined below.

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[18] *A coronary artery bypass graft is an invasive surgical procedure which involves the attachment of a section of healthy vein or artery to an occluded coronary artery at points before and after the site of occlusion. The procedure serves to bypass the occluded segment, and thus, restore blood flow to effected areas of the heart, relieve angina pectoris and reduce the risk of myocardial infarction (Oxford Reference Concise Medical Dictionary, III Edition, 1990).*

[19] *Percutaneous transluminal coronary angioplasty is a nonsurgical approach to dilating occluded arteries by mechanical force (Kent, 1987). Small collapsed balloons attached to flexible catheters, are directed by means of X-ray screening to the site of occlusion where they are inflated (Kent, 1987). Inflation of the balloon serves to dilate the artery. On completion of this procedure, the apparatus is withdrawn. Successful dilation is defined as a minimum of a 20% increase of the artery's diameter (Detre et al., 1988).*

Measurement of Recurrent Atherosclerosis: Maximal Oxygen Uptake (VO_2max)

Any disease within an organ or system reduces reserve capacity, which in turn, hinders the body's ability to respond to increasing physiological demands (Jones & Campbell, 1982). The organs and systems of the body generally have a substantial reserve and thus, clinical symptoms only become apparent with a significant loss of capacity (Jones & Campbell, 1982). This is particularly true of the cardiovascular system where substantial loss can occur before the system's capacity to meet the demands of everyday activities is compromised (Jones & Campbell, 1982). For example, arteries narrowed by atherosclerosis are still able to supply sufficient oxygenated blood during sedentary activities (Sharkey, 1984). Therefore, providing the cardiovascular system is not subjected to vigorous activity, the signs and symptoms of underlying atherosclerosis will not be detected (Sharkey, 1984).

In many cases underlying atherosclerosis is only evident when the heart is deprived of oxygen (Sharkey, 1984). Among people with CHD this can occur during vigorous exercise when the oxygen demands on the heart are increased (Jones & Campbell, 1982; Sharkey, 1984). This demand must be met by an increased supply of oxygen to the heart (Cornett & Watson, 1984). An increased supply of oxygen is achieved by an enhanced flow of coronary blood (Cornett & Watson, 1984). Atherosclerosis causes the occlusion of arteries which limits blood flow (Goldberg, 1978). Where coronary arteries are obstructed by a build-up of atherosclerosis, the resulting impeded flow of blood reduces the supply of oxygen to the heart (Crean & Fox, 1987; Posner, Gorman, Klein, & Woldow, 1986). Thus, an imbalance between the oxygen demands of the myocardium and the volume of coronary blood flow occurs (Cornett

& Watson, 1984). The point at which this imbalance materialises is termed the ischaemic threshold and is often experienced as angina pectoris (Cornett & Watson, 1984; Crean & Fox, 1987).

The severity of coronary disease and the ischaemic threshold are inversely related. As the severity of coronary disease increases so does the ischaemic threshold decrease (Cornett & Watson, 1984). In other words, the sooner the ischaemic threshold is reached during exercise, the greater the extent of atherosclerosis. This relationship translates into a poor prognosis because the presence of angina pectoris during vigorous exercise predicts an increased risk of future fatal and non-fatal myocardial infarction (Crean & Fox, 1987).

The ischaemic threshold can be determined by measuring maximal oxygen uptake which is symbolised as VO_2max (Cornett & Watson, 1984; Holloszy, 1983). VO_2max provides a measure of the greatest amount of oxygen used by an individual during physical exertion (Cornett & Watson, 1984; Crean & Fox, 1987). It reflects the maximal capacity to enhance blood flow and thus, the ability to meet the increasing oxygen demands of the body's tissues (Cornett & Watson, 1984; Fletcher et al., 1992; Jones & Campbell, 1982). Conversely, reduced maximum oxygen uptake during exercise will reflect an inadequate flow of oxygenated blood to the heart (Jones & Campbell, 1982) and consequently, the existence and extent of atherosclerosis (Posner et al., 1986). Because VO_2max provides an index of the cardiovascular system's ability to utilise oxygen (Jamieson & Lavoie, 1987), it is widely used to determine cardiorespiratory fitness (De Geus, Van Doornen, & Orlebeke, 1993; Desharnais, Jobin, Côté, Lévesque, & Godin, 1993; Pierce,

Weltman, Seip, & Snead, 1990; Weltman et al., 1990), the severity of CHD (Bruce, 1971) and prognosis for recovery or reinfarction (Sami et al., 1979).

While maximal oxygen uptake can be enhanced through aerobic exercise (Weltman et al., 1990), it will also be reduced if factors (e.g., stress) which initiate the development of atherosclerosis persist (Posner et al., 1986). A deterioration of maximal oxygen uptake will thus indicate the further degeneration of the cardiovascular system, and consequently, the presence of recurrent strain. Conversely, enhanced maximal oxygen uptake will reflect physiological recovery. Therefore, maximal oxygen uptake provides an index of physiological strain, namely recurrent atherosclerosis.

Prognosis for Recovery

Considerable advances in the treatment of CHD and the subsequent reduction of CHD related deaths have occurred within the domain of medicine (Smith & O'Rourke, 1988). The persistently high percentage of individuals left disabled by CHD does not reflect the advances made by medical science (Smith & O'Rourke, 1988). Nor too, do the numbers of people afflicted by secondary CHD. Indeed, 50% to 70 % of CHD mortality occurs among individuals with a history of documented CHD (Gordon & Kannel, 1971; Kamarck & Jennings, 1991). Among those patients who do survive an initial event, a significant number resist the necessary steps to recovery (Bass, 1984; Gundle, Bozman, Tate, Raft, & McLaurin, 1980; Heller, Frank, & Kornfeld, 1974) and remain, to varying degrees, physically, psychologically, and socially handicapped (Erdman, 1990). Clearly, there is a profound need to identify factors which predict prognosis (Powell & Thoreson, 1985).

While knowledge of the factors implicated in the development of disease has advanced considerably, the ability to predict prognosis is still limited (Marmot & Madge, 1987). The limit in extant powers of prediction can be attributed to an inadequate consideration of psychosocial factors in the pathogenesis of disease (Marmot & Madge, 1987). Given that research has identified at least 246 factors which relate to increased risk of CHD (Hopkins & Williams, 1981), it would be beyond the scope of the present research to consider all potential predictors. However, a review of the literature has revealed two factors which are particularly relevant to the prediction of prognosis, namely Type A behaviour and aerobic exercise (Dembroski et al., 1989; Holmes & Roth, 1983; Morris et al., 1990; Öhman et al., 1989). The present research will attempt to identify the components of both Type A behaviour and aerobic exercise which predict prognosis for recovery or reinfarction among CHD patients.

Conclusion

The stress theory and empirical studies reviewed in this chapter attest to the inadequacy of considering CHD exclusively as an endpoint measuring strain. A coronary event is also a discrete, objective stressor (Rejeski et al., 1985; Terry, 1992), which initiates a further series of potentially stressful events (Cohen, 1981). Moreover, the stressors associated with a coronary event are not confined to the initial danger period but extend into convalescence and beyond physiological recovery (Terry, 1992). Further, these responses, together with the patient's newfound physical status, require perpetual readjustment (Cay et al., 1985). Thus, both the actual coronary event and the process of adjustment and recovery are

sources of stress. These stressors have the potential to generate substantial psychological and physiological strains. specifically anxiety, depression and recurrent atherosclerosis.

Based on the premise that CHD generates psychological stress which, in turn, contributes to further strain, the present research aims to examine psychological and behavioural responses to the stress of CHD and the extent to which these responses predict strain. To this end, the present research will develop and test a model of predictors of CHD prognosis (see Chapter 8). The predictor variables included in the model will be components of exercise and Type A behaviour. Prior to developing the model of predictors, the present research will establish whether exercise has the potential to ameliorate the consequences of CHD by comparing the physiological and psychological levels of strain of exercising and non-exercising groups of cardiac patients (see Chapter 6). The present research will then identify the Type A components which characterise cardiac patients' behaviour (see Chapter 7). The Type A components relevant to cardiac patients will be included with the measures of exercise compliance in the model of predictors of prognosis.

CHAPTER 3

THE IMPACT OF EXERCISE ON THE STRESS-STRAIN PROCESS

Exercise Based Cardiac Rehabilitation

The World Health Organization (1964) defines cardiac rehabilitation as the "sum of activity required to ensure cardiac patients the best possible physical, mental, and social conditions so that they may by their own efforts regain as normal as possible a place in the community and lead an active, productive life" (Oldridge, 1988, p.43). It is a lifelong process which begins immediately following the cardiac event, or the treatment thereof (Wagner & Williams, 1987). Two fundamental goals underlie cardiac rehabilitation. The first is to remove any physical or psychological obstacles that hinder the patient's ability to resume a full and productive life, and the second, to reduce the patient's risk of suffering a second coronary event (Wagner & Williams, 1987). Central to most formal cardiac rehabilitation is an exercise programme which is conducted in a group setting, under medical supervision (Cay et al., 1985; Oldridge, 1988; Wagner & Williams, 1987).

Participation in an exercise based rehabilitation programme is a deliberate attempt to master the psychological and physiological disabilities caused by CHD. It is an active response to an objective demand that has been perceived as being stressful and as having potentially debilitating consequences (Soloff, 1978b). Involvement in a rehabilitation programme allows patients to participate actively in their own

recovery and gain confidence from the fact that supervised physical activity is both feasible and safe (Cay et al., 1985). Participation in such a programme restores a measure of control to the course of recovery and thus helps to reduce the depression typical of the post coronary period (Soloff, 1978a; 1978b). The ability to exercise also provides patients and their families with tangible evidence of patients' increased physical improvement (Naughton & Hellerstein, 1971). Confidence in their increasing physical mastery enhances cardiac patients' adjustment to CHD (Rejeski et al., 1985). Restoration of confidence in the ability to perform physical activities is vital to recovery from a coronary event (Allen, Becker, & Swank, 1990). The present chapter examines the effect of exercise on cardiac patients' psychological and physiological health and reviews the research pertaining to this relationship.

Aerobic Exercise Defined

Aerobic exercise is defined as the repeated "... movement of large muscle masses against little resistance" (Hammond, 1985, p.276). Performed regularly, aerobic exercise increases blood circulation, oxygen distribution, and the release of those metabolites[20] necessary for the dilation of blood vessels and ultimately, reduces peripheral resistance and improves maximal cardiac output[21] (Hammond, 1985). In addition, aerobic exercise reduces resting and sub maximal heart rates, blood pressure and catecholamine levels (Hartley et al., 1987). Regular aerobic exercise

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[20] *Metabolites are products involved in the process of metabolism which are usually concerned with the break down of chemical compounds and are frequently accompanied by the liberation of energy (Stedman's Medical Dictionary, 1961).*

[21] *Maximal cardiac output refers to the amount of blood ejected by the heart which in turn, determines the amount of blood which is delivered to the exercising muscles (Fletcher et al., 1992).*

improves the overall condition of the cardiovascular system in healthy individuals, and retards cardiovascular performance decrement in individuals with CHD (Fletcher et al., 1992; Schneider & Reed, 1985). Moreover, aerobic exercise induces a period of relaxation which serves to block the physiological arousal induced by stress (Ledwidge, 1980).

The Effect of Exercise on Physiological and Psychological Processes

Aerobic exercise modifies the psychological and physiological strain imposed by post coronary stress (Holmes & Roth, 1985). Exercise influences the experience of stress through both physiological and cognitive mechanisms. In terms of physiology, exercise augments adrenal activity, which causes an increased reservoir of the steroids needed to oppose the impact of stress (Michael, 1957). Habitual exercise reduces the physiological response to stressful stimuli (Cantor, Zillman, & Day, 1978; De Geus, van Doornen, De Visser, & Orlebeke, 1990) and increases immunity to a wider range of potentially stressful stimuli (Selye, 1974). Through mobilising adaptive responses and increasing resistance to powerful stressors, vigorous exercise serves to relax the individual and enables him or her to withstand psychological frustration (Seyle, 1976; Wood, 1977) and hence, manage stress (Folkins & Sime, 1981).

Increased fitness permits quicker physiological recovery from stress induced arousal and thus, enhances resistance to protracted, potentially damaging, sympathetic activity (McGlynn, Franklin, Lauro, & McGlynn, 1983). Indeed, exercise reduces cardiac rehabilitation patients' heart rates, blood pressure and serum cholesterol

and catecholamine levels and improves their functional capacity (Fletcher et al., 1992; Sharkey, 1984). All of these measures indicate a reduction in the stress response, namely, excessive sympathetic activity (Holmes & Roth, 1985; Widimsky' & Broustet, 1985).

Exercise also exerts an influence on cognition by occupying cognitive processes to the exclusion of anxiety provoking stimuli in CHD patients (Folkins & Sime, 1981). In this way, physical activity distracts the individual from the processing of negative, stress induced, emotional states such as the anxiety (Wood, 1977), anger and fear (Folkins & Amsterdam, 1977) typically experienced by CHD patients. Exercise provides a respite from stress and therefore, facilitates more effective coping (Brown, 1991). Further, the ability to perform repeated, incremental exercise quells anxiety concerning reinfarction for it demonstrates to the patient that he or she is able to exert substantial physical effort without being limited by cardiac symptoms (Soloff, 1978b).

Exercise also reduces affective disorders by enhancing body image (Goldberg & Folkins, 1974), sense of well-being (Folkins & Sime, 1981), self-concept and social adjustment (Layman, 1974), all of which accelerate CHD patients' return to work (Smith & O'Rourke, 1988). Return to such chosen activities as work is indicative of psychological recovery (Greenland & Briody, 1984). Thus, in addition to regulating the form and intensity of disturbances which exist within the cardiac patient's internal physiological environment (Folkins & Sime, 1981), exercise enhances cardiac patients' positive emotions.

Empirical Evidence of the Psychological Benefits of Exercise

While physiological and psychological theory supports the moderating effect of exercise on the stress-strain relationship, the empirical evidence is equivocal (Kinnaird, Yoham, & Kleval, 1982). On the one hand, there is considerable evidence of the positive consequences of exercise programmes for CHD patients. Folkins (1976) and Stern, Gorman and Kaslow (1983) have found that physical fitness reduced anxiety and depression among CHD patients. Shephard, Kavanagh and Klavora (1985) also report that cardiac patients' depression and anxiety was significantly reduced following a one year, exercise based rehabilitation programme. Decreases in cardiac patients' anxiety and disablement perception following a six month exercise programme have been demonstrated by Erdman, Duivenvoorden, Verhage, Kazemier and Hugenholz (1986). Schomer and Noakes (1983) have shown that participation in a six month exercise programme reduced cardiac patients' incidence of depression and tension and enhanced their emotional stability. Similarly, Valliant and Asu (1985) found that participation in a 12 week structured exercise programme increased self-esteem and reduced depression. Kavanagh et al. (1977) followed 44 severely depressed cardiac patients who were participants in an exercise programme over a period of four years. The results of the four year follow-up assessment indicated that the significant reduction in depression which occurred was related to participation in the programme. These studies suggest that aerobic exercise reduces the intensity of psychological manifestations of strain experienced by CHD patients.

Other studies yield less convincing results of the positive psychological effects of exercise programmes on CHD patients. For example, the results of the prerandomisation exercise programme of the National Exercise and Heart Disease Project (NEHDP) indicate that, while exercise reduced depression among CHD patients, it increased anxiety levels (Stern & Cleary, 1981). Van Dixhoorn, der Loos, and Duivenvoorden (1983) report similar results in a controlled study comprising myocardial infarction patients who participated in a programme which combined exercise with relaxation training. After participating in the programme, patients demonstrated a decrease in disablement perception and an increase in feelings of well-being. However, these patients' anxiety levels also increased (Van Dixhoorn et al., 1983). The increased anxiety in these samples supports the notion that exercise may actually accelerate stress hormone production and thus, exert negative psychological effects (Holloszy, 1983).

Given the existence of conflicting findings it is not possible to make categorical statements regarding the benefits of exercise on the degree of cardiac patients' psychological strain. Conflicting findings also serve as a reminder that extant knowledge is suggestive and incomplete (Holloszy, 1983; Widimsky' & Broustet, 1985). While research has consistently shown that exercise reduces depression, its position regarding the effects of exercise on anxiety is equivocal. Persistent anxiety hinders the process of psychological and physiological recovery from a cardiac event (Brown & Munford, 1983; Garrity & Klein, 1975). As aerobic exercise has the potential to ameliorate the negative consequences of anxiety (Folkins & Sime, 1981; Wood, 1977) the present research will investigate the relationship between exercise and CHD patients' anxiety.

Empirical Evidence of the Physiological Benefits of Exercise

Several studies report significant physiological improvements among cardiac rehabilitation patients following participation in an exercise programme. Grodzinski, Jette, Blümchen, and Borer (1987) found that patients assigned to a four week exercise programme demonstrated a significant improvement in maximal oxygen uptake. The control group, whose programme comprised relaxation classes combined with light exercise, showed a reduction in maximal oxygen uptake (Grodzinski et al., 1987). Bethell and Mullee (1990) report similar results in a controlled, randomised study of exercising and non-exercising cardiac patients. In their study, the maximal oxygen uptake of the 99 exercising patients was significantly greater than the 101 non-exercising patients (Bethell & Mullee, 1990). Moreover, the incidence of angina pectoris among the exercising group fell by 10%, but increased by 60% among the non-exercising group (Bethell & Mullee, 1990). The NEHDP results demonstrated a significant improvement in maximal oxygen uptake among post-infarction patients following participation in an exercise programme (Stern & Cleary, 1981).

The relationship between exercise and a reduced incidence of CHD mortality has also been examined. In a Finnish study, myocardial infarction patients were randomly assigned to either an intervention or a control group (Kallio, Hämäläinen, Hakkila, & Luurila, 1979). A primary component of the intervention was an exercise programme. At the three year follow-up, the incidence of CHD mortality among the control group was significantly higher than among the intervention group (29.4% versus 18.6%) (Kallio et al., 1979). Kellerman (1973) revealed a mortality rate of 6% for CHD patients participating in a long term exercise programme, compared with a

mortality rate of 22% for patients who dropped out of the programme and patients participating in a short term exercise programme. In a similar study, annual mortality rates of a control group were 4.3% as opposed to the 2.1% exhibited by the exercise group who had attended the programme for three years (Hellerstein & Franklin, 1984). Shephard, Corey and Kavanagh's (1981) retrospective review of exercise programme compliers and drop-outs established that drop-outs were more prone to reinfarction. Compelling evidence is also supplied by O'Conner et al.'s (1989) meta-analysis of 22 randomised exercise trials involving myocardial infarction patients. The analysis reveals that participation in an exercise programme is associated with reduced cardiovascular and all-cause mortality for three years after participation and a significant reduction in sudden death for the year following participation (O'Conner et al., 1989).

Indirect support for the beneficial effects of habitual exercise is provided by recent research conducted in Germany (Willich et al., 1993) and America (Mittleman et al., 1993). The two studies indicate that while sporadic exercise can trigger a myocardial infarction, regular exercise provides considerable protection against such occurrences. Each using over a thousand myocardial patients, these controlled, epidemiologic studies investigated the relationship between sudden strenuous physical activity (e.g., gardening, walking up stairs, housework) and the onset of myocardial infarction. The authors report that in comparison to less strenuous or sedentary activities, the risk of a myocardial infarction during, or shortly after strenuous exercise was two times greater in the German study (Willich et al., 1993) and six times greater in the American study (Mittleman et al., 1993). However, this ratio did not apply to individuals who habitually exercised four to five times per week (Mittleman et al., 1993; Willich et al., 1993). Indeed, little to no excess risk

was reported for these individuals (Curfman, 1993). Similarly, participants in both studies who had a known history of CHD did not demonstrate a higher risk than their previously healthy counterparts (Mittleman et al., 1993; Willich et al., 1993).

The studies reviewed above suggest that attending an exercise programme enhances CHD patients' physiological condition and reduces the incidence of fatal and non-fatal reinfarction. However, other studies ensure that the relationship between exercise and improved prognosis remains suggestive. For example, the Ontario Exercise-Heart Collaborative Study (OEHCS) failed to demonstrate a significant difference between the morbidity and mortality rates of a post coronary exercise training group and a non-exercising group (Pisa, Denolin, & Lamm, 1985). These findings highlight the inconsistency of research into the beneficial effects of exercise on CHD patients.

Limitations of Past Research

The conflicting results yielded by both psychological and physiological studies may be due to methodological flaws. First, several of the studies which failed to find a significant relationship between exercise and diminished strain (e.g., NEHDP, 1981; Valliant & Asu, 1985) included an exercise programme of insufficient length (i.e., six and 12 weeks, respectively). Significant changes in body chemistry require approximately four months of regular exercise training (Ismail & Young, 1977). Consequently, alterations in personality factors associated with body chemistry would not occur within a short space of time (De Geus et al., 1993; Folkins & Sime, 1981). As the therapeutic benefits of exercise only become apparent after six months of participation in an exercise programme (De Geus et al., 1990; Erb, Fletcher, &

Sheffield, 1979; Shephard, 1979), it is possible to conclude that longer programmes would have yielded significant results. Support for this possibility lies in the fact that those studies which reported an increase in anxiety (e.g., the NEHDP, 1981) included exercise programmes lasting six weeks, while those studies which included exercise programmes of three or more months (e.g., Stern et al., 1983) reported reductions in anxiety. Second, the absence of non-exercising control groups (e.g., Kavanagh et al., 1977; the prerandomisation exercise programme of the NEHDP, 1981) makes it impossible to determine whether any improvement was a result of maturation effects or of exercise *per se*.

A third weakness characterises several of the studies which compared the CHD morbidity and mortality rates of cardiac patients who participated in an exercise programme with those of patients who dropped out of the programme (e.g., Kellerman, 1973; Shephard et al., 1981). These studies failed to establish the drop outs' reasons for non-compliance. Thus, they have been unable to establish whether underlying CHD morbidity caused patients to drop out and subsequently, experience a secondary event, or whether, secondary events were the result of patients' failure to comply with the exercise programme. Thus, these studies are unable to establish the direction of the relationship between non-compliance and anxiety, depression and recurrent cardiac events.

Though the positive effect on global physical and psychological health has yet to be concluded, habitual exercise does reduce blood pressure and heart rate (Astrand & Rodahl, 1977). These reductions are associated with less ischaemia and an enhanced ability to perform more work without experiencing angina pectoris (Fletcher et al., 1992; Hartley et al., 1987). There is also a growing body of

suggestive evidence indicating that exercise is a natural bodily defence against atherosclerosis and the consequences thereof (Oberman, 1983). Moreover, numerous studies have shown that exercise promotes psychological health (Brown, 1991). It is perhaps for these reasons that exercise continues to be prescribed by physicians to combat anxiety (Dishman, 1985), depression (Simons, McGowan, Epstein, Kupfer, & Robertson, 1985) and stress (Crews & Landers, 1987) and constitutes a primary component of cardiac rehabilitation programmes (Oldridge, 1988).

To justify the continued inclusion of exercise in cardiac rehabilitation, the role of exercise should be clarified. Therefore, the aim of the first study of the present research is to establish if exercise reduces anxiety, depression and the incidence of recurrent cardiac events. This study is designed to overcome the aforementioned limitations of past research. This will be achieved by comparing cardiac patients who have participated in a six month exercise programme with a control group of patients who withdrew from the same programme. This study will supersede past research by determining the control group's reasons for leaving the programme. In identifying the group's reasons, this study will establish whether debilitating CHD symptoms preceded the decision to withdraw and were responsible for subsequent coronary events and increased levels of anxiety and depression. Alternatively, this study will show that the control group's incidence of anxiety, depression, and coronary events can be attributed to their failure to exercise. Thus, it will be able to establish the direction of the relationship between exercise and CHD related psychological and physiological ill health. The rationale for this study is discussed in greater detail in Chapter 5.

Measures of Compliance

Research concerning the frequency and duration of exercise required to reduce CHD risk is limited and inconsistent (Lakka et al., 1994). In order to clarify the role of exercise more precisely, research must extend beyond a mere comparison between exercising and non-exercising groups to include an investigation of the means by which exercise exerts a positive effect on psychological and physiological prognosis. This can be achieved by investigating the nature of compliance with prescribed exercise programmes. Past research has typically investigated compliance exclusively in terms of attendance (e.g., Blumenthal, Williams, Wallace, Williams, & Needles, 1982; Digenio et al., 1991; Oldridge & Jones, 1983). Enhanced cardiorespiratory fitness occurs as a result of the duration and frequency (i.e., attendance) of exercise (Pollock, Miller, Linnerud, & Cooper, 1975; Rejeski, Morley, & Miller, 1984; Seals, Hagberg, Hurley, Ehsani, & Holloszy, 1984). Indeed, to achieve a true training effect, patients need to exercise three times a week for 20 to 40 minutes at 50% of their maximal oxygen uptake (Oberman, 1983). Only under these circumstances will relevant systems be sufficiently activated to exert a physiological effect (Cupelli et al., 1984). Therefore, attendance figures alone, are insufficient for the prediction of prognosis. A comprehensive assessment of compliance should also include a measure of the duration of exercise performance (Sharkey, 1984). Thus, a further aim of the present research is to address this criticism of past research by including both measures in an assessment of the relationship between exercise compliance and cardiac patients' physiological and psychological condition.

Conclusion

Seen from a general perspective, aerobic exercise plays a definitive role in the regulation and enhancement of psychological functioning (Folkins & Sime, 1981). It simultaneously distracts from minor stressors, increases resistance to unavoidable stressors and improves stress management abilities (Selye, 1974; Wood, 1977). With respect to cardiac rehabilitation, exercise is believed to affect prognosis by retarding the development of atherosclerosis (Oberman, 1983), restoring cardiorespiratory fitness (Morris et al., 1990; Schneider & Reed, 1985; Shephard, 1985) and modifying the stress-strain relationship characteristic of the post-coronary period (Holmes & Roth, 1985; McGlynn et al., 1983). However, the existence of conflicting results regarding the effect of exercise on prognosis clearly indicates that this belief requires confirmation. A further indication is that any attempts to confirm and define the effect of exercise must include both measures of compliance, namely, the frequency and duration of exercise. An aim of the present research is to determine, in a controlled study, if exercise reduces the incidence of recurrent cardiac events, anxiety and depression (see Chapter 6.). A further aim is to establish the precise means by which exercise influences prognosis (see Chapter 8.). This will be achieved by examining the relationship between the two compliance measures and standard indices of physiological and psychological strain. As a concurrent aim is to examine the relationship between components of Type A behaviour and strain, Type A behaviour will be examined in the following chapter.

CHAPTER 4

THE ROLE OF TYPE A BEHAVIOUR IN CARDIAC REHABILITATION

Type A Behaviour Defined

CHD patients have long been observed as aggressive (Meninger & Meninger, 1936), hard-driving (Dunbar, 1943; Osler, 1892) and achievement oriented (Kemple, 1945). However, it was only in the 1950's that Friedman and Rosenman conducted systematic research into the relationship between behaviour and CHD (Friedman & Rosenman, 1959). The study followed Friedman and Rosenman's observation that their cardiac patients shared certain speech and behaviour patterns, later classified by the authors as Type A behaviour (Friedman & Rosenman, 1959). Type A behaviour has subsequently 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, and if required to do so, against the opposing efforts of other things or other persons (Friedman & Rosenman, 1974, p.67).

In contrast, Type B behaviour is characterised by a relative absence of Type A behaviour. Type B individuals are less competitive, more relaxed, and more easy going than Type A's (Jenkins et al., 1967). The incidence of mixed *type* is rare and possibly the result of error variance (Strube, 1989).

Type A behaviour is believed to be an extreme response to psychosocial stress (Kamarck & Jennings, 1991). Type A's are more vulnerable to potentially stressful situations than Type B's (Brief et al., 1983) and perceive more situations as being stressful (House, 1974). Thus greater vulnerability is reflected in the hyper-reactive sympathetic nervous system shown to characterise Type A behaviour (Janisse & Dyck, 1989). The heightened susceptibility to stress is believed to be a product of Type A's need for control (Glass, 1977b). In turn, this need is associated with Type A's style of self-evaluation which is based on the overriding need to prove oneself (Matthews, 1982; Price, 1982). The psychological, behavioural and physiological consequences of Type A's need for control and self-esteem are outlined below.

Psychological and Behavioural Consequences of Type A Behaviour

The need for control, even under seemingly uncontrollable circumstances, is pervasive among Type A individuals (Taylor & Cooper, 1989) and is the factor which underlies much of Type A's behaviour (Lee, Ashford, & Bobko, 1990). For the Type A, control facilitates the acquisitions and achievements (Byrne, Reinhart, & Heaven, 1989) needed to preserve and enhance self-esteem (Janisse & Dyck, 1989; Yuen & Kuiper, 1992). It also serves to reduce or avoid both internal and external forms of negative appraisal (Thoreson & Powell, 1992).

To maintain self-esteem, Type A's need to manipulate the environment in such a way that their level of achievement remains constant (Furnham & Linfoot, 1987; Keltikangas-Järvinen, 1990; Lee, 1992). To this end, Type A's expose themselves to an increasingly excessive number of occupational, social and personal challenges which carry the promise of achievement (Byrne & Rosenman, 1986; Ganster et al.,

1991). They attempt to master these incremental challenges, with increasingly higher standards of performance (Price, 1982; Thoreson & Powell, 1992; Yuen & Kuiper, 1992). This pattern demands constant, maximal effort (Fazio, Cooper, Dayson, & Johnson, 1981) and limits relaxation (Price, 1982). It requires the Type A to par- vities rapidly, suppress any subjective states (e.g., fatigue) and exhibit hostility toward anyone or anything that might interfere with his or her performance (Carver, Coleman, & Glass, 1976; Glass, 1977a; Hassmén, Stahl, & Borg, 1993; Keltikangas-Järvinen, 1990). Suppression also extends to physiological symptoms such as increased heart rate (Forgays, 1992).

The effort Type A's invest to complete tasks frequently leads to the self generation of stress (Heilbrun, Palchanis, & Friedberg, 1986; Jamal, 1990; Strube & Werner, 1985). Further, Type A's typically perceive their activities as stressful irrespective of the actual stress inherent to the task (Payne, 1988). This is possibly due to the emphasis Type A's place on achievement and the consequent experience of failure and loss of esteem if observable achievements are elusive.

The stress arising from the need to achieve is compounded by the often unreasonable demands Type A's place on themselves (e.g., multiple, unrealistic and diverse deadlines) (Gastorf, Suls, & Sanders, 1980). In the long term, this pattern of behaviour proves maladaptive (Taylor & Cooper, 1989; Yuen & Kuiper, 1992) and may be responsible for the greater incidence of self-initiated, distressing life-events experienced by Type A's (Järvikoski, & Härkäpää, 1988).

By attempting to control events as fickle as acquisition and achievement, and by making self-esteem contingent on these, Type A's set the stage for failure. As

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observable acquisitions and achievements fluctuate, so too does the Type A's sense of esteem (Price, 1982). The dependence of perceived self-esteem on achievement renders Type A's vulnerable to negative affects such as depression (Yuen & Kuiper, 1992). When Type A's attempts at establishing control over their acquisitions and achievements are frustrated, they demonstrate a greater degree of self-blame and self-directed anger than Type B's (Furnham & Linfoot, 1987; Taylor & Cooper, 1989). In these instances, Type A's typically resort to a sense of helplessness and give up their efforts (Glass, 1977a). Psychological helplessness yields a severe decrease of norepinephrine[22] and increase of cholinergic activity[23] (*cf.* Matthews, 1982; Verrier, DeSilva, & Lown, 1983). These abrupt shifts between the sympathetic activity associated with active coping and the parasympathetic activity associated with helplessness have been suggested as a cause of sudden cardiac death (Engel, 1970; Matthews, 1982; Verrier et al., 1983). It may be that as a socially acceptable, even masculine, manifestation of strain, CHD may present Type A's with the only acceptable response to the public humiliation they believe will be generated by their perceived failure (Price, 1982). Based on the above literature, it is possible to assume that Type A's are more threatened by a potential loss of control and esteem than Type B's and thus perceive the potential loss as more stressful (Dyck, Moser & Janisse, 1987). Indeed, as outlined below, research has consistently shown that

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22 *Norepinephrine (noradrenalin) is a principle transmitter substance of the adrenal medulla (Carlson, 1981). Liberation of norepinephrine causes the constriction of small blood vessels which serves to increase blood pressure and blood flow through the coronary arteries, enhance the rate and depth of breathing, slow the heart rate, and relax the smooth muscles of the intestinal walls (Oxford Reference Concise Medical Dictionary, III Edition, 1990).*

23 *Cholinergic activity refers to the activation of nerve fibres (Stedman's Medical Dictionary, 1961) involved in the stimulation of the reflexive functions (e.g., heart rate, blood pressure) (Carlson, 1981).*

there exists a positive relationship between Type A behaviour and self-reported levels of stress (Gamble & Matteson, 1992).

Suls and Wan's (1989) meta-analysis examined the relationship between Type A behaviour and chronic emotional distress. Their analysis was based on the belief that Type A individuals' incessant struggle to achieve more and more is a consequence of their chronic need for control and is the cause of greater emotional distress (Suls & Wan, 1989). As the need for control is pervasive among Type A's it was believed that such individuals would experience more emotional distress than Type B's. The meta-analysis of 27 studies yielded a significant, albeit, moderate, association between Type A behaviour and chronic emotional distress and a non-significant relationship between Type B behaviour and emotional distress (Suls & Wan, 1989). Thus, Type A's appear to experience more distress than Type B's (Suls & Wan, 1989).

Physiological Consequences of Type A Behaviour

Besides a possible role in acute coronary events, the perpetual need for control and to prove oneself is also considered responsible for Type A's heightened sympathetic nervous system activity (Matthews, 1982). Type A individuals' comparatively greater need for self-esteem, and the consequent heightened arousal, is clearly evident in settings where their sense of control is threatened (Houston, 1983). In such settings, Type A's are more aroused by the perceived threat than Type B's (Houston, 1983). This physiological arousal is more pronounced in settings which promise either accomplishment or failure (Houston, 1983), and to a degree, is limited to threats to the ego (e.g., competitive or evaluative settings) (Janisse & Dyck, 1989). The

physiological reactivity of Type A's is further enhanced when they are subjected to time pressure or harassment (Cinciripini, 1986). While the hypothesis linking the need for control and self-esteem to an exaggerated physiological response is incomplete, extant research permits a fundamental assumption. That is, Type A's sense of threat is reflected by an exaggerated physiological response, particularly in instances involving ego-threat (Janisse & Dyck, 1989).

The assumption that Type A's exhibit an exaggerated physiological response to stress has generated considerable support. Based on an examination of 37 studies comparing Type A and B reactivity to a variety of different challenges, Manuck and Krantz (1986) report that Type A's react to ordinary stressors with an excessive physiological response. Of the 37 studies reviewed, 26 demonstrated heightened physiological reactivity among Type A's (Krantz & Manuck, 1984). Suls and Sanders (1988) meta-analysis of the literature concerning reactivity also showed that Type A's experience higher systolic blood pressure reactivity than Type B's. This reactivity is not indiscriminate and reflects the valence Type A's attach to the stressor.

Type A's discrimination between stressors has been demonstrated in an experiment conducted by Malcolm, Janisse and Dyck (1984). Specifically, when exposed to both ego-threat and physical discomfort stressors, Type A individuals' cardiovascular response to the former was significantly greater than baseline measures, while the response to the latter was below baseline measures. In the same experiment, Type B's demonstrated a marginal increase in heart rate in response to both ego and non-ego threatening stressors (Malcolm et al., 1984). Thus, unlike Type A's, they did not differentiate between the two stressor types. Moreover, the Type B's did not experience the marked increases in physiological response which were evident

among the Type A's. Similar results were yielded by Ward et al. (1986) in an experiment which required 56 men to complete a series of challenging tasks. Compared to Type B's, Type A's demonstrated a significantly greater cardiovascular response during task performance (Ward et al., 1986). The difference between Type A and B responses was more pronounced in tasks involving ego-threat (e.g., decision making and problem solving under time pressure) and non-existent in tasks requiring passive waiting or tolerance of pain (e.g., timed, cold pressor tests[24]) (Ward et al., 1986). Type A's also demonstrated greater reactivity in response to laboratory induced cognitive stressors in a study by Schneider, Julius, and Karunas (1989).

Field studies of Type A and B's differential responses to stress have also been recorded. For example, a study of medical students during routine days and exam days showed that Type A's respond to mental stress with significantly greater increases in diastolic and systolic blood pressure than Type B's (Lovallo, Sausen, Altenüller, Everson & Wilson, 1991). A study conducted by Melamed et al. (1993) showed that Type A's respond to objective stressors in the work place with significantly greater increases in diastolic blood pressure and heart rate. Thus, Type A's demonstrate differential reactivity to stressors. Further, reactivity is more conspicuous when the stressor constitutes a threat to self-esteem.

While Type A's reactivity may be situationally determined, they do exhibit heightened 'baseline' arousal. In Schneider, Friedrich, Neus, and Ruddle's (1982) controlled experiment, pharmacological suppression of sympathetic activity was

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[24] *Cold pressor tests involve the submersion, into iced water, of the hand or foot for an approximate period of one minute (Kamarck & Jennings, 1991).*

accompanied by a decrease in the intensity of Type A behaviour. Similarly, Krantz, Arabian, Davia, and Parker (1982) report that the correlation between intensity of Type A behaviour and heightened cardiovascular reactivity continued to be significant even when patients were subjected to general anaesthetic during coronary artery bypass graft surgery.

There is also considerable evidence to show that reactivity patterns are stable intrapersonal characteristics (Sloan & Bigger, 1991). For example, Smith and O'Keeffe (1988) have demonstrated that the cardiovascular reactivity which occurred in response to the Type A interview significantly predicted the reactivity response to a mental rotation task which occurred two weeks later. The results of Smith and O'Keeffe's (1988) study suggest that cardiovascular reactivity is stable over time. Lassner et al.'s (1994) study provides partial support for the hypothesis that cardiovascular reactivity is stable over different stress eliciting situations. In this study, the cardiovascular reactivity elicited by an asocial (i.e., individual psychomotor tasks) stressor was significantly associated with the reactivity elicited by a social (i.e., conflict resolution within the family) stressor (Lassner et al., 1994). This association was only significant among the women, but not the men and boys, included in the study. The results of these studies indicate that there is, in fact, a stable psycho-physiological substrate of Type A behaviour and that it exists without the direct influence of the Type A individuals' environment and cognitive processes.

Given that there exists empirical support for the aforementioned assumptions, it is plausible that Type A's have a heightened sensitivity and pronounced reactivity to stress as a result of their extreme need to prove themselves. Providing these assumptions are correct, the chronic stress generated by Type A behaviour will

predispose Type A's to stress related diseases such as CHD. The following exploration of the hypothesis that Type A behaviour promotes CHD is based on the premise that Type A's do perceive more situations as stressful and react with a more pronounced cardiovascular response than Type B's. To assess the Type A-CHD hypothesis, the physiological processes which underlie stress and presumably contribute to CHD, will be examined.

Type A - CHD Relationship

Much of the argument for a relationship between Type A behaviour and CHD is located within the 'reactivity' hypothesis (Manuck & Krantz, 1986; Thomas & Friedman, 1990). This hypothesis holds that heightened cardiovascular reactivity, which occurs in response to the environment, generates chronic elevations of sympathetic nervous system activity which cause deterioration of the cardiovascular system and ultimately, leads to disease (Ganster et al., 1991; Melamed et al., 1993; Van Egeren & Sparrow, 1990). The relationship between exaggerated cardiovascular reactivity and disease is outlined below.

CHD occurs as a result of the prevalence of both atherosclerosis and acute precipitating factors (Matthews, 1988). An example of the latter would be the increased oxygen demand and cardiovascular reactivity resulting from psychological stress (Matthews, 1988; Mittleman et al., 1993; Willich et al., 1993). Type A's seek out potentially stressful situations, are prone to perceiving situations as threatening or challenging, employ stress-inducing cognitive strategies to cope with these, behave in a sufficiently aggressive manner to provoke conflict with others and

evaluate their performance and behaviour negatively (Smith & Anderson, 1986). All of these stress factors generate heightened physiological reactivity (Svebak, Knardahl, Nordby, & Aakvaag, 1992). Specifically, the perception of stress generates an increased discharge of the catecholamines, norepinephrine and epinephrine[25] (Carlson, 1981). The increased discharge plays a primary role in the activation of the sympathetic nervous system under conditions of stress (Ganster, Mayes, Sime, & Tharp, 1982). Under such conditions, the increased release of these catecholamines serves to generate sufficient energy to resist stress by mobilising glucose and fatty acids and elevating heart rate, blood pressure and blood flow (Ganster et al., 1982). However, the release of these catecholamines is also believed to expose the arteries to the infiltration of cholesterol (Jonisse & Dyck, 1989), to promote platelet aggregation[26] and accelerate the rate of damage to the arteries (Glass, 1977b).

A corollary of this belief is that frequent increases in the liberation of norepinephrine and epinephrine and the consequent sustained sympathetic activation have been found to cause the atherosclerotic lesions underlying CHD (Eliot, 1979; Haft, 1974; Wright, 1988). Certainly, extreme, protracted or periodic stimulation resulting from psychological stress can lead to the dysfunction of the cardiovascular system

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[25] *Epinephrine (adrenaline) is the chief hormone of the adrenal medulla. Liberation of epinephrine invokes constriction or dilation of the blood vessels and cardiac stimulation (Stedman's Medical Dictionary, 1961), increases the rate and depth of breathing, raises metabolic rate, improves the force of muscle contraction, delays the onset of muscle fatigue and reduces the flow of blood to the bladder and intestines while relaxing their muscular walls (Oxford Reference Concise Medical Dictionary, III Edition, 1990). The release of epinephrine occurs as a response to challenge (Svebak et al., 1992) and prepares the body for fight or flight (Oxford Reference Concise Medical Dictionary, III Edition, 1990). Epinephrine is a sensitive measure of response intensity (Rosenman, 1986).*

[26] *Platelet aggregation is the process whereby disc shaped structures (platelets) within blood bond to convert blood to a solid state (Oxford Reference Concise Medical Dictionary, III Edition, 1990). This occurs in response to vascular injury (Kamarck & Jennings, 1991).*

(Krantz et al., 1985). It follows then, that any psychological mechanism which enhances the perception of stress and consequently, increases the amount of circulating catecholamines, will play a significant role in the pathogenesis of CHD (Glass, 1977b). Type A individuals are prone to both cardiovascular and neuroendocrine hyperresponsivity to stress (Anderson et al., 1986) and to a slower recovery from stress induced cardiovascular arousal (Jamieson & Lavoie, 1987). The predisposition to hyperresponsivity and slower recovery translates to prolonged cardiovascular arousal which ultimately contributes to the development of atherosclerosis and CHD (Anderson et al., 1986; Ganster et al., 1991; Jamieson & Lavoie, 1987). The mechanisms by which this occurs are outlined below.

Type A behaviour is associated with excessive norepinephrine and epinephrine discharge (Dembrowski, MacDougall, Shields, Petitto, & Lushene, 1978; Friedman, St. George, Byers, & Rosenman, 1960). Periods of expressed anger and aggression liberate norepinephrine, while epinephrine is released during periods of suppressed anger and anxiety (Meltzer, 1973), and among Type A's in particular, during periods of stress (Lundberg & Forsman, 1975). Expressed and suppressed anger, anxiety and aggression are all characteristics of Type A behaviour (Rosenman & Chesney, 1982). An increased liberation of norepinephrine also occurs during the active coping which follows Type A's attempts to achieve control (Glass, 1977a). Excessive levels of norepinephrine and epinephrine initiate the release of fatty substances which contribute to the development of atherosclerosis (Crider, Goethals, Kavanagh, & Solomon, 1983). The release of fatty substances, together with the hypertension typically exhibited by Type A's, contributes to atherosclerosis and eventually to CHD (Friedman & Rosenman, 1974).

The psychological stress and concomitant increases of catecholamines associated with Type A behaviour exert a further deleterious influence through their relation to accelerated platelet aggregation and blood clotting (Friedman, Rosenman, & Carroll, 1958; Rosenman & Friedman, 1974). The acceleration facilitates the incorporation of platelets into arterial plaque and thus, expedites the development of atherosclerosis (Glass, 1977b). The acceleration is further compounded by the excessive insulin levels typically exhibited by Type A's (Goldberg, 1978). Due to high insulin levels, Type A's require three to four times as much time as Type B's to dispose of the cholesterol in their systems, thereby increasing the probability of blood clotting and platelet aggregation (Goldberg, 1978). Accelerated blood clotting and platelet aggregation relate reliably to CHD (Rosenman & Chesney, 1982). However, it must be emphasised that this relationship is not acute. Rather, it is the protracted and / or excessive cardiovascular reactivity which occurs in response to Type A behaviour which precipitates the development of CHD (Lassner et al., 1994; Taylor & Cooper, 1989). Considering that reactivity is a stable characteristic (Sloan & Bigger, 1991), and that over time, the habitual cardiovascular overreaction to stress, and the slower recovery thereto, contributes to the development of hypertension and CHD (Anderson et al., 1986; Engebretson, Matthews, & Scheier, 1989; Jamieson & Lavoie, 1987; Janisse & Dyck, 1989), it is plausible that Type A's are predisposed to these cardiac disorders. The following review of the relevant empirical studies explores the plausibility of the Type A - CHD relationship.

Empirical Evidence of the Type A-CHD Relationship

The relationship between Type A behaviour and CHD was first investigated by Friedman and Rosenman in 1959. The investigation showed the incidence of CHD to be seven times greater among Type A's than Type B's. Friedman and Rosenman's (1959) seminal study prompted the Western Collaborative Group Study (WCGS) (Rosenman et al., 1964a; 1964b). Designed to determine the existence of a relationship between Type A behaviour and the incidence of CHD, the WCGS assessed the development of CHD in 3 524 employed men aged 39 to 59 over a period of 8.5 years (Rosenman et al., 1964a; 1964b). The study was based on a double-blind design which meant that those assessing Type A behaviour were unaware of participants' physical condition while those determining risk of CHD were unaware of participants' behaviour type (Haynes & Matthews, 1988). Results of the 8.5 year follow-up showed that Type A men demonstrated an increased risk of 2.2 for CHD, 2.1 for myocardial infarction and 2.5 for angina pectoris when compared with Type B men (Rosenman et al., 1975). That is, Type A men had more than double the risk of contracting CHD (Rosenman et al., 1975).

The Framingham Study (Haynes et al., 1978), the second prospective inquiry into the Type A-CHD relationship, revealed comparable results. Incorporating 1 674 initially CHD free men and women aged 45 to 77, the Framingham Study followed subjects' progress for a period of eight years to assess the development of CHD (Haynes, Feinleib, & Kannel, 1980). A comparison between behaviour types after eight years yielded an increased risk of 1.8 for CHD, 2.1 for myocardial infarction and 1.8 for angina pectoris among Type A men (Haynes et al., 1980).

Subsequent studies confirmed the results of the WCGS and the Framingham Study. For example, the Belgian-French Pooling Project (1984) which incorporated 2 811 male civil servants and factory workers who were free of CHD at the outset of the study, showed that Type A's susceptibility to myocardial infarction was 1.6 times greater than Type B's. Similar results were yielded by other studies (e.g., Cramer, 1991; Haynes et al., 1980; Jenkins, 1976; Kannel & Gordon, 1974). Other research has established that Type A behaviour is not only associated with CHD events, but also with the development of atherosclerosis (Blumenthal et al., 1978) and the incidence of traditional CHD risk factors such as high cholesterol levels (Lundberg, Hedman, Melin, & Frankenhaeuser, 1989) and tobacco abuse (Glass, 1977a).

Booth-Kewley and Friedman's (1987) meta-analysis demonstrates that the results of the above studies are indicative of much of the research into the relationship between Type A behaviour and the development of CHD. Comprising 87 studies, the meta-analysis revealed a modest, though highly reliable relation between Type A behaviour and CHD. Together, these studies generated widespread acceptance of the Type A - CHD relationship in the 1970's and, in 1978, prompted the National Heart, Lung and Blood Institute (NHLBI) to acknowledge officially the existence and validity of the Type A - CHD relationship (Review Panel on Coronary-Prone Behavior and Coronary Heart Disease, 1981): Subsequent research indicates that the NHLBI's acknowledgement may have been premature.

Initial results of the WCGS (Rosenman et al., 1975) and of the Framingham Study (Haynes et al., 1978) served to establish Type A behaviour as a risk factor for primary CHD. However, the cogency of these findings has failed to withstand further investigation. A 22 year follow-up of 99% of the original WCGS sample included an

examination of the death certificates of the 584 subjects who died during the interim period (Ragland & Brand, 1988a). Of the 214 who had died of CHD, no association between Type A behaviour and CHD mortality was found (Ragland & Brand, 1988a). Similarly, a 20 year follow-up of a portion of Framingham Study subjects showed that, while Type A's had over twice the risk of developing angina pectoris, they were no more at risk of experiencing myocardial infarction and coronary death than their Type B counterparts (Eaker, Abbott, & Kannel, 1989).

The seven year Multiple Risk Factor Intervention study established that Type A behaviour was unrelated to CHD in a sample of 3 110 men (Shekelle, Gale, & Norusis, 1985). A non-significant relationship between Type A behaviour and CHD was also revealed in the eight year Honolulu Heart Study of approximately 2 200 men who were of Japanese extraction (Cohen & Reed, 1985; Cohen, Syme, Jenkins, Kagan, & Zyzanski, 1979). While the non-significant results in the Honolulu Heart Study could be attributed both to a low incidence of classical CHD risk factors among Japanese men and a cultural intolerance for the dimensions of Type A behaviour associated with CHD (Cohen et al., 1979; Haynes & Matthews, 1988), the research conducted on Caucasian samples indicates that Type A behaviour has no apparent relation to CHD.

The non-significant Type A-CHD association has been replicated in various studies (e.g., Case, Heller, Case, & Moss, 1985; Ruberman et al., 1984) and indicated by several meta-analyses (e.g., Matthews, 1982; 1988; Miller et al., 1991). Matthews' (1982) meta-analysis assessed the ability of various Type A measures to determine behaviour type in relation to cardiovascular reactivity. Of the 14 prospective studies utilising the Structured Interview, ten reported correlations between Type A

behaviour and cardiovascular reactivity to specific tasks (Matthews, 1982). Significant correlations between cardiovascular reactivity and Type A behaviour were reported in approximately half of the 21 prospective studies which employed the Jenkins Activity Survey (JAS), while the single psycho-physiological study using the Framingham Type A Scale revealed a non-significant association (Matthews, 1982). In a later meta-analytic review of the relationship between Type A behaviour and the subsequent onset of CHD, Matthews (1988) established that when the number of independent studies and the number of participants thereof were weighted, Type A behaviour failed to operate as a reliable predictor of CHD.

Conversely, Booth-Kewley and Friedman's (1987) meta-analysis of 87 prospective and cross-sectional studies established a moderate, albeit reliable, association between Type A behaviour and CHD. The positive association revealed in Booth-Kewley and Friedman's (1987) meta-analysis may be due to the authors' failure to weight the number of studies and participants and also, their inclusion of cross-sectional studies. While cross-sectional studies provide evidence of occurrence and association (Biddles, Slavings & Anderson, 1985), they prohibit statements regarding cause and effect relationships (Matthews, 1988). That is, they prohibit the determination of relationships between risk factors such as Type A behaviour and CHD (Haynes & Matthews, 1988).

Miller et al.'s (1991), more recent meta-analyses of 61 studies shows that while studies conducted before 1979 were significantly more likely to demonstrate a positive relationship between Type A behaviour and CHD, studies conducted in the succeeding decade failed to find an association between the two. Specifically, between 1978 and 1989, there was a four-fold likelihood of null findings reported in

studies using the Structured Interview and an eight-fold likelihood of null findings in studies based on self-report measures of Type A behaviour (Miller et al., 1991). The majority of the post 1978 studies were characterised by at least one of the following: a.) a high risk sample, b.) use of self-report measures to assess Type A behaviour or c.) use of a fatal myocardial infarction as the disease end-point. Miller et al. (1991) attribute the trend toward null findings to an increase in the above three characteristics in studies conducted after 1978. The authors maintain that the small sample sizes used in high risk studies have reduced statistical power and consequently, have lowered the correlations between Type A behaviour and disease. The extensive utilisation of self-report measures of Type A behaviour, which correlate poorly with CHD, would have increased the incidence of null findings (Miller et al., 1991). Miller et al. (1991) offer several explanations for null findings yielded by studies using a fatal myocardial infarction as the disease endpoint. First, they suggest that because Type A behaviour may accelerate the development of CHD, Type A cardiac patients might be younger than Type B's, and thus possess the youthful hardiness associated with a longer life expectancy. Second, they propose that Type A behaviour is associated with acute precipitating factors and not CHD itself. As a consequence, Type A cardiac patients may have less underlying CHD and survive longer than Type B cardiac patients. Third, the authors question whether a death certificate is a reliable indicator of a fatal myocardial infarction. It should be noted that despite finding a tendency toward null findings after 1978, Miller et al. (1991) found that on the whole, Type A behaviour, as assessed by the Structured Interview, was associated with CHD. Indeed, the authors found that 70% of CHD patients were Type A's.

Existing studies and meta-analyses concerning the Type A - primary CHD relationship are equivocal and therefore, undermine the construct's status as a predictor of primary CHD. This does not compromise the construct's significance and it remains the only psychosocial factor whose modification has resulted in a reduced severity of CHD (Räikkönen & Keltikangas-Järvinen, 1992). However, results of the few studies which have examined the predictive role of Type A behaviour on the development of secondary CHD (Ragland & Brand, 1988b; Rejeski et al., 1985) are less ambiguous. Ironically, secondary CHD research indicates that Type A behaviour does not contribute to the development of secondary CHD. The discrepancy between primary and secondary CHD research suggests that Type A behaviour may exert a differential effect on healthy individuals and CHD patients.

The Relationship Between Type A Behaviour and Secondary CHD

Research suggests that Type A behaviour discriminates between healthy individuals and CHD patients (Orth-Gomér & Undén, 1990). Healthy Type A's are known to deny the potential negative consequences of their behaviour, while Type A's with CHD are known to recognise and address these consequences (Wright, 1988). It is thus plausible that when diagnosed with CHD, Type A's take responsibility for the negative components of their behaviour and modify them accordingly (Evans, 1990; Wright, 1988). Modification of the negative components of Type A behaviour may result in a crucially different prognosis. That is, it may protect the Type A from future coronary events. Existing reinfarction research supports this possibility.

The 22-year follow-up of the WCGS examined behaviour type in relation to both short-term (within 24 hours) and longer-term fatal reinfarction (Ragland & Brand, 1988b). The follow-up study revealed that among subjects who contracted CHD during the 8.5 year period, approximately equal numbers of Type A and B's experienced a fatal reinfarction within the first 24 hours. In the longer term, however, the incidence of non-fatal reinfarction was significantly greater among Type B individuals while the incidence of fatal reinfarction was 42% less among Type A's than among Type B's (Ragland & Brand, 1988b). These results suggest that Type A's have a greater life expectancy after a first myocardial infarction than Type B's. In Powell et al.'s (1993) study of 83 women, a 4.5 year intervention designed to reduce Type A behaviour failed to diminish the incidence of CHD mortality. This study also revealed an inverse relationship between the global Type A measure and CHD mortality (Powell et al., 1993). Similar inverse relationships between Type A behaviour and secondary CHD have been reported by Barefoot et al. (1989), Dimsdale, Gilbert, Hutter, Hackett and Block (1981) and Sloan and Bigger (1991). Thus, numerous studies demonstrate that Type A behaviour is associated with a reduced risk of fatal reinfarction.

Other studies have failed to yield an association between Type A behaviour and secondary CHD. For example, in the Multi-Center Post-Infarction Project, Case et al. (1985) found Type A behaviour to be unrelated to reduced left ventricular ejection fraction[27] in a sample of 516 patients recovering from a myocardial infarction. The

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[27] *Ventricular ejection fraction measures the pumping action of the left ventricle of the heart. In that it reflects functional abnormalities (Kamarck & Jennings, 1991), the measure constitutes the most precise means of determining the severity of CHD (Case, 1988). Left ventricular dysfunction is the primary factor influencing fatal reinfarction (Powell & Thoreson, 1988).*

authors also established that Type A behaviour was unrelated to risk of CHD mortality (Case et al., 1985). The Aspirin Myocardial Infarction Study, which included 2 314 men and women who had experienced a myocardial infarction, failed to establish a link between Type A behaviour and reinfarction (Shekelle et al., 1985). These two studies suggest that Type A behaviour is unrelated to both the progressive development of CHD and the incidence of fatal and non-fatal reinfarction. Together with the results yielded by Ragland and Brand (1988b), Powell et al. (1993), Barefoot et al. (1989), Dimsdale et al. (1981), and Sloan and Bigger (1991) these studies imply that the impact of Type A behaviour on survivors of a coronary event may differ to that which occurs among individuals initially free of CHD (Abbott & Peters, 1988; Barefoot et al., 1989).

Limitations of Type A - Secondary CHD Research

While reinfarction research demonstrates that Type A behaviour imposes a differential impact on primary and secondary events, its value is limited by a general emphasis on global Type A behaviour (Edwards, Baglioni, & Cooper, 1990a). The predictive validity of the global Type A construct is poor (Evans, 1990) as only a subset of Type A components are believed to predict primary or secondary CHD (Dembroski et al., 1989; Grossarth-Maticek & Eysenck, 1990). Any attempt to correlate the Type A construct with manifestations of disease must include a thorough analysis of the multidimensional nature of that construct (Russek et al., 1990). While Price (1982) has identified 31 dimensions of Type A behaviour in the Type A literature, she maintains that many of these dimensions lack scientific plausibility. Further, analysis of all of 31 would be beyond the scope of the present research. Therefore, an assessment of the potentially differential impact of Type A

behaviour on secondary events requires the identification of key components of the Type A construct and their independent effect on health outcomes (Abbott & Peters, 1988; Ganster et al., 1991).

A Multidimensional Model of CHD patients' Type A Behaviour

Research has yet to determine the precise number and kind of Type A components which predict CHD (Heilbrun et al., 1986). However, various authors investigating the predictive capacity of Type A behaviour have isolated certain components of the behaviour pattern which relate to health variables more consistently than the global construct. Some of these (e.g., Bluen, Barling, & Burns, 1990; Helmreich, Spence, & Pred, 1988; Öhman et al., 1989; Spence, Helmreich, & Pred, 1987) proclaim Type A behaviour to be a bi-dimensional construct comprising impatience irritability and achievement striving. Those adopting the bi-dimensional approach to Type A behaviour suggest that impatience irritability is associated with health outcomes, while achievement striving is associated with performance. Others (e.g., Abbott & Peters, 1988; Friedman & Booth-Kewley, 1988; Siegman, Dembrowski, & Ringel, 1987) propose a multi dimensional construct wherein hostility and anger expression (anger-in and anger-out) are among the strongest predictors of disease (Matthews & Haynes, 1986). Thus, recent research identifies achievement striving, impatience irritability, hostility, anger-in and anger-out as key components of the Type A construct. An aim of the present chapter is to define achievement striving, impatience irritability, hostility, anger-in and anger-out and to motivate their classification as key components of Type A behaviour.

While the centrality of the five components has been acknowledged (e.g., Matthews & Haynes, 1986; Spence et al., 1987; Strube, 1989), no attempt has been made to reconcile all five in a model of Type A behaviour. Further, extant research has not examined the relationship between the five components and CHD. It has been argued previously that Type A behaviour discriminates between those who have CHD and those who do not. Therefore, models of Type A components which are applicable to healthy samples (e.g., Burns & Bluen, 1990) will not be suitable to a sample of CHD patients. It is probable that CHD patients' knowledge of their disease effects their behaviour and psychological status (MacDougall et al., 1985) and their methods of coping with disease (Martin & Lee, 1992). It may also effect their psychological assessment, particularly if this is based on self report measures (Evans, 1990). Knowledge of their physiological status and consequent greater risk, may alter patients' responses to measures of Type A behaviour (Evans, 1990). Relationships between the various components of Type A behaviour and health and performance are likely to be confounded by such effects.

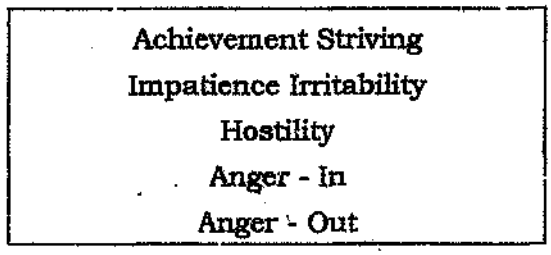
Individuals classified as Type A are known to deny their predisposition to Type A behaviour (Wright, 1988). Following a coronary event, Type A's are quicker to seek medical treatment than Type B's (Leikin, 1990) and show a greater willingness to acknowledge the ramifications of their behaviour (Wright, 1988). To enhance their chances of survival, Type A's may modify their Type A behaviour to the extent that their post-coronary behaviour classification does not reflect their pre-coronary classification (Haynes & Matthews, 1988; Matthews, 1988). This may result in Type A survivors of a coronary event being crucially different in character to Type A's who failed to survive (Evans, 1990). Indeed, Friedman et al. (1986) report a significant reduction in Type A behaviour during the period of recovery from a cardiac event. As

post-coronary behaviour modification may alter the differential effect of each component on performance and health, the Type A components which predict primary CHD cannot be assumed to predict secondary CHD. Therefore, before the differential effects of the Type A components on secondary CHD can be determined, it is necessary to develop a theoretical model which reflects the behaviour of a high risk sample.

The purpose of the present research is to develop and test a model of Type A components [illegible] for research concerning CHD patients. Specifically, the present [illegible] the Type A behaviour manifested by CHD patients in terms of a [illegible] model comprising achievement striving, impatience irritability, [illegible]-out and hostility (see Figure 4.1). While each component exerts a [illegible] on post-coronary behaviour (Jennings, 1984), they can be divided [illegible] broad categories, namely, benign and coronary-prone (Abbott & Peters, [illegible]). Achievement striving is believed to constitute a benign component (Öhman et [illegible]). The coronary-prone components are thought to be impatience irritability [illegible] et al., 1989), hostility (Matthews, 1988), anger-in and anger-out (Johnson & [illegible]ranan, 1987; Taylor & Cooper, 1989). Because little research has been conducted on the relationship between the components and reinfarction, their inclusion in the present model is motivated by research on healthy individuals. A discussion of each component follows.

Figure 4.1

A Multidimensional Model of Type A Behaviour



Achievement Striving

Achievement striving behaviour is a central component of Type A behaviour and is believed to generate many of the other components (Evans, 1990; Perry, Kane, Bernesser, & Spicker, 1990). Achievement striving reflects the hard-driving component included in Friedman and Rosenman's original conceptualisation of Type A behaviour (Furnham, 1990a). Individuals exhibiting achievement striving behaviour are typically hard-driving, competitive, achievement oriented, committed to their careers and extremely productive and successful (Spence et al., 1987). Those with a strong orientation towards achievement striving behaviour tend to have well defined goals and interests (Kliewer & Weidner, 1987) and to persist with goal oriented behaviour (McClelland & Burnham, 1976). This behaviour results in a greater number of actual achievements (Perry et al., 1990). The heightened acquisition of achievements and the corresponding fast pace of living are regarded by these individuals to be a key means of exerting control over the environment (Jamal & Baba, 1991) and maintaining self-esteem (Furnham & Linfoot, 1987). Individuals with an achievement striving orientation demonstrate many of the characteristics described by Friedman and Rosenman (1974) in their original

definition of Type A behaviour. However, their predisposition to achievement striving does not render them susceptible to ill health. Instead, achievement striving appears to contribute to performance excellence (Furnham, 1990a).

Significant correlations between achievement striving and performance have been yielded by the majority of studies examining the differential effects of achievement striving and impatience irritability. For example, in a sample of 362 male and 351 female undergraduates, Spence et al. (1987) found that achievement striving correlated significantly with the cumulative Grade Point Average (GPA) derived from two or more semesters. Achievement striving did not correlate with the negative health measures assessed in this study. A follow-up longitudinal study measuring academic performance over a minimum of four semesters replicated the original results and demonstrated the temporal stability of the achievement striving and impatience irritability dimensions (Spence, Pred, & Helmreich, 1989). In a further study, the same authors reanalyzed data collected by Matthews, Beane, Helmreich and Lucker (1980) and established a significant relationship between achievement striving and number of citations and publications among 118 eminent academic psychologists (Helmreich et al., 1988).

Several studies have advanced the work of Spence and colleagues by assessing the extent to which achievement striving and impatience irritability differentially predict other behavioural and health outcomes. In a study of 113 university students, Barling and Charbonneau (1992) replicated Spence et al.'s (1987) results showing significant correlations between achievement striving and GPA and nonsignificant correlations between achievement striving and measures of health. Barling and Charbonneau (1992) further established that achievement striving, and not

impatience irritability, predicted performance on a proof-reading task measuring attention and concentration. Bluen et al.'s (1990) research on life insurance brokers revealed that achievement striving predicted sales performance and job satisfaction, but not depression. In follow-up research comprising a portion of Bluen et al.'s (1990) original sample, achievement striving predicted the number and value of subsequent policies sold and the amount of commission earned while impatience irritability did not (Burns & Bluen, 1992). More recent support for Spence et al.'s (1987, 1989) research is provided by Lee (1992), who found that achievement striving significantly predicted class performance as measured by examination results in a sample of 142 undergraduate students and 39 part-time MBA students. This study showed that impatience irritability was again unrelated to performance. Thus, achievement striving, but not impatience irritability, relates consistently to measures of performance.

The Type A hard-driving component, which is conceptually analogous to achievement striving (Furnham, 1990a; Öhman et al., 1989), does not relate significantly to the incidence of both CHD and numerous other diseases (Rimé, Ucros, Bestgen, & Jeanjean, 1989). Indeed, Wright (1992) has established that in comparison to CHD patients, individuals hospitalised for a variety of other diseases score significantly higher on the hard-driving scale. The hard-driving component does not relate significantly to the number of visits to a physician (Suls & Marco, 1990). Further, Öhman et al. (1989) report that achievement striving does not correlate with heightened, task-induced, activation of the cardiovascular system among students. Heightened cardiovascular activity is characteristic of CHD (Rosenman, 1986). Given that the above research indicates that achievement striving is unrelated to extreme cardiovascular responses, it can be assumed that

individuals who exhibit primarily achievement striving behaviour are not predisposed to CHD.

Type A components have demonstrated relative stability over time (*cf.* Bergman & Magnusson, 1986). Further, cardiovascular reactions to behavioural challenges are a stable individual trait (Matthews, Rakaczky, Stoney, & Manuck, 1987; Taylor & Cooper, 1989). This suggests that individuals respond to particular behavioural challenges with a stereotypical level of cardiovascular reactivity which is stable over time (Lemmer et al., 1994). Thus, the level of cardiovascular reactivity which occurs in response to achievement striving prior to the development of CHD is likely to be similar to that which occurs after the disease has manifested itself. Should this be the case, the achievement striving behaviour exhibited by CHD patients may result in a positive response to rehabilitation but not to increasing deterioration of the cardiovascular system. Despite the fact that achievement striving might exert a beneficial influence on recovery, it would still appear to be a component of Type A behaviour, and thus should be included in the multidimensional model of the post-coronary Type A construct.

Impatience Irritability

Impatience irritability is a primary component of Type A behaviour (e.g., Matthews, Glass, Rosenman, & Bortner, 1977; Musante, MacDougall, Dembroski, & Van Horn, 1983; Palmer et al., 1992; Spence et al., 1987; Tett et al., 1992). Impatience irritability is characterised by a volatile temper, a sense of time urgency, intolerance, anger and hostility (Bluen et al., 1990). A need to maintain self-esteem through the achievement of goals and the mastering of challenges is thought to illicit impatience

irritability particularly if self-esteem needs are obstructed in any way (Price, 1982). Impatience irritability is believed to exert a deleterious effect on physical condition (Spence et al., 1987). In an attempt to master stressors which threaten their sense of control, individuals high in impatience irritability mobilise active coping processes (Öhman et al., 1989). These processes give rise to a discharge of norepinephrine and an increase in sympathetic activity (Matthews, 1982). As a direct result, a greater load than is metabolically justified is placed on the myocardium (Obrist, 1981). Frequent and sustained activation of such coping processes and the corresponding high levels of norepinephrine in the system increase blood pressure, expedite arterial and myocardial deterioration and facilitate the incidence of sudden death (Graff, 1974). Thus, the need to establish control over stressors (Glass, 1977a) may render impatient and irritable individuals vulnerable to both general ill health and the development of CHD.

Studies which have assessed the differential potential of achievement striving and impatience irritability to predict divergent outcomes have consistently yielded significant correlations between impatience irritability and measures of health but not measures of performance. Both Spence et al. (1987) and Barling and Charbonneau (1992) report that impatience irritability correlated significantly with negative health (e.g., headaches, respiratory problems) but not with academic performance (i.e., GPA). The relationship between impatience irritability and number of citations and publications in Helmreich et al.'s (1988) study was also nonsignificant. Nakano (1991) examined the effects of an operant self-control procedure on the impatient behaviour of Japanese men. The procedure required subjects to evaluate the influence of impatient behaviours and monitor their occurrence with the aim of developing a healthier repertoire of behaviours (Nakano,

1991). The results of this study indicated that modification of impatient behaviours resulted in a significant reduction in physical symptoms (Nakano, 1991). A correlational study of 206 female college students by Woods and Burns (1984) reveals that women who generated a high score on the speed impatience dimension of the JAS experienced 86% more chest pain and gastrointestinal, respiratory and sleep problems than women who scored low on the dimension. Speed-impatience, a Type A dimension which features a number of impatience irritability items and is conceptually similar to impatience irritability (Öhman et al., 1989), also predicted number of visits to a physician over an 18 month period in Suls and Marco's (1990) prospective study of 167 college students. Thus, impatience irritability is related to both self-reported and physician-diagnosed illness.

Other research reveals a relationship between impatience irritability and psychological ill health. For example, Forgays' (1992) study shows that women who demonstrate a strong orientation towards time pressed, impatient behaviour report significantly more maternal stressors and illness than women who do not (Forgays, 1992). Houston, Smith and Zurawski (1986) also demonstrate positive relationships between speed impatience and psychological distress. Positive correlations between impatience irritability and depression were found by Bluen et al. (1990) in one of the few studies to examine the relationship between impatience irritability and psychological manifestations of strain. In a study comprising 134 male medical practitioners and their wives, husband's impatience irritability predicted the marital dissatisfaction expressed by both themselves and their wives (Barling, Bluen, & Moss, 1990). Thus, there is empirical support for an association between impatience irritability and measures of psychological ill health.

More specific to the present research are reports of associations between impatience irritability and CHD. In a review of data yielded by the WCGS, Matthews et al. (1977) found that Type A individuals originally identified as being likely to develop CHD, and who subsequently did so, demonstrated a high degree of irritability at the outset of the study. In a sample of 1949 adults, speed-impatience was significantly associated with the self-reported incidence of a range of diseases which included CHD among them (Rimé et al., 1989). In Wright's (1992) study, the incidence of Speed-Impatience was significantly greater among a sample of patients hospitalised for CHD than among the sample hospitalised for other diseases.

Associations between impatience irritability and indices of cardiovascular reactivity have also been established. In a study comprising 40 extreme Type A students, Öhman et al. (1989) sought to determine whether certain Type A components had a greater relevance to cardiovascular reactivity than others. Subjects scoring high on impatience irritability experienced significantly greater increases in heart rate and pulse transit time[28] during stressful tasks than subjects demonstrating low levels of impatience irritability. Under the same conditions, heart rate and pulse transit time were unrelated to the hard-driving and competitive measures which the authors believe, are similar to the achievement striving factors discussed elsewhere (e.g., Spence et al., 1987). Both heart rate and pulse transit time are indices of cardiovascular reactivity (Öhman et al., 1989; Svebak et al., 1992). Jennings (1981)

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[28] *Pulse transit time reflects the speed of a pulse wave (Öhman et al., 1989) and is measured by means of an electrocardiogram and a dynograph (Svebak et al., 1992). Pulse transit time is taken as the lapse of time which occurs between the onset of the R - wave of the electrocardiogram and the peak of the pressure pulse measured at the earlobe (Svebak et al., 1992). The R-wave is the initial positive wave of ventricular activity during the cardiac cycle.*

found that participation in a task designed to arouse physiological change, increased the cardiovascular reactivity of Type A's high in impatience. As increased cardiovascular reactivity serves as a marker for hypertension and CHD (Rosenman, 1986), impatience irritability may contribute to the physiological changes which perpetuate CHD. Sveiak et al. (1992) also found that impatience irritability was significantly related to sympathetic reactivity when subjects were exposed to a challenging task known to induce cardiovascular changes. The results of these three studies suggest that impatience irritability initiates active coping processes associated with cardiovascular changes which place a metabolically unjustified load on the heart (Glass et al., 1989). Thus, impatience irritability, and not achievement striving, is associated with task-induced cardiovascular reactivity (Glass et al., 1989). Given that heightened cardiovascular reactivity is a CHD risk factor (Krantz & Manuck, 1984), these results lend support to the belief that impatience irritability is prognostic of CHD. It is thus plausible that, in the context of past coronary behaviour, impatience irritability represents the coronary prone and achievement striving the benign components discussed by Abbott and Peters (1988). Should this be the case, impatience irritability might counter the effect of rehabilitation and achievement striving, facilitate recovery.

Research on achievement striving and impatience irritability contributes to an explanation of the equivocal findings yielded by past Type A-CHD research. Those studies utilising the global Type A construct may have been insufficiently specific to determine the differential effects of the achievement striving and impatience irritability components of Type A behaviour in predicting health outcomes. Consequently, any such differential relationships between achievement striving, impatience irritability and health outcomes may have been obscured. Should this be

the case, benign components of the global construct would be partly responsible for the diminished predictive validity of Type A-CHD relationships (Wright, 1988). The differential effects of achievement striving and impatience irritability may also explain the discrepancy between primary and secondary Type A-CHD research. The impatience irritability component may dominate Type A's behaviour and consequently increase the risk of primary CHD. However, following the diagnosis of CHD, the Type A may modify impatience irritability behaviours to the extent that his or her risk of secondary CHD is reduced. While the achievement striving / impatience irritability studies help to resolve the equivocal findings of past research, they fall short of providing a conclusive explanation. The studies neglect to examine equally pathogenic components of Type A behaviour such as hostility and anger (Lee, 1992). The present research aims to overcome this weakness by expanding the Type A construct beyond achievement striving and impatience irritability to include hostility and anger in an assessment of the differential effects of Type A components on cardiac rehabilitation patients' health.

Hostility

Hostility has been considered a primary component of Type A behaviour since the time of the Type A construct's original definition (Smith & Pope, 1991). It is also considered to be one of the most pathogenic components of Type A behaviour (Blumenthal, O'Toole, & Haney, 1984; Greenglass & Julkunen, 1991; Matthews, 1988). Like Type A behaviour, hostility is multidimensional (Siegman et al., 1987). From the literature it is evident that hostility comprises a behavioural component characterised by overt aggression (aggressive-hostility) and a neurotic component characterised by cynicism and mistrust of others (cynical hostility) (Barefoot et al.,

1983; Siegman et al., 1987). However, in the original description of Type A behaviour hostility was defined as free floating and occurring in a response to anyone or anything that threatened to hinder Type A's progress (Matthews & Haynes, 1986). In terms of the original definition of Type A behaviour, it is aggressive, rather than cynical, hostility that is associated with the Type A construct (Engebretson & Matthews, 1992; Siegman et al., 1987). Individuals who show a strong tendency to exhibit hostile behaviour are believed to have a heightened predisposition to CHD (Suarez & Williams, 1989).

Coronary-prone, hostile behaviour can be described as a behavioural disposition characterised by animosity, and frequent acts of aggression toward anything which impedes behavioural freedom (Houston & Vavak, 1991; Rosenman, 1991; Williams et al., 1988; Williams, Barefoot, & Shekelle, 1985). Inherent to the period following a cardiac event are a number of factors which may threaten behavioural freedom. Examples include, physical and psychological limitations imposed by CHD and drug therapy, changes in employment and or family status, and loss of income and self-sufficiency (Budnick, 1991; Erdman, 1990; Rejeski et al., 1985). Type A's are more likely than Type B's to perceive factors such as these as threats to freedom and to respond with acts of hostile aggression (Check & Dyck, 1986; Strube & Werner, 1985). As hostile aggression is also a typical response to the behavioural limitations imposed by ill health or the treatment thereof (Rhodewalt & Fairfield, 1991), Type A cardiac patients are likely to display considerable aggressive hostility. In this context, aggressive-hostility may be directed at the rehabilitation programme and / or the disease itself and may result in noncompliant behaviour (Rhodewalt & Fairfield, 1991). Both hostility and non-compliance with a cardiac rehabilitation programme are associated with reinfarction (Koskenvuo et al., 1988; Shephard et

al., 1981). Further, patients with advanced CHD are prone to potentially dangerous cardiac arrythmias[29] (Koskenvuo et al., 1988). Among this population, acts of extreme hostile-aggression are implicated in the occurrence of fatal arrythmias (Koskenvuo et al., 1988). As the period following a cardiac event is characterised by a number of factors which limit behavioural freedom, and Type A's often respond to these with hostile-aggression leading to non-compliance, hostility is implicated in the incidence of secondary CHD. The role of hostility in the development of CHD is outlined below.

The precise means by which hostility engenders CHD is unclear (Diamond, 1982). However, there is some evidence that hostility occurs within a high arousal / attentional state which initiates increased sympathetic activity (Lundberg et al., 1989; Weidner, Sexton, McLellan, Conner, & Matarazzo, 1987). Increased activity results in the metabolically unjustified release of norepinephrine and mobilisation of lipids (Weidner et al., 1987) which, over time, damage the arterial endothelium[30] (Krantz & Manuck, 1984). Damage to the arterial endothelium constitutes the first step in the development of CHD (Ross, & Glomset, 1984).

Support for a link between hostility and increased sympathetic activity is provided by Dembrowski et al. (1978) who found that in response to experimentally aroused stress, hostility correlated significantly with systolic blood pressure and heart rate.

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[29] *Cardiac arrythmias refer to continuous or intermittent disturbances in the heart's normal rhythm which can, in severe cases, result in cardiac arrest (Oxford Reference Concise Medical Dictionary, III Edition, 1990).*

[30] *The single layer of cells lining the arteries (Oxford Reference Concise Medical Dictionary, III Edition, 1990).*

In a series of similar studies conducted by Suarez and Williams (1989, 1990), individuals high in hostility who were harassed while trying to complete an anagram task responded with more pronounced cardiovascular reactivity than individuals low in hostility. Comparable results were yielded by Hardy and Smith (1988) and Weidner, Friend, Ficarrotto and Mendell (1989). In Weidner et al.'s study, individuals demonstrating high levels of hostility evinced significantly greater diastolic and systolic blood pressure levels when attempting an unsolvable anagram task than those demonstrating low levels of hostility. Christensen and Smith (1993) report that in social interactions requiring the self-disclosure of personal information, individuals high in hostility demonstrated significant increases in diastolic and systolic blood pressure and heart rate. Ganster et al. (1991) also report that, when their sample of 568 healthy employed men and women were subjected to challenging stimuli, hostility correlated with both increased diastolic blood pressure and slower rates of physiological recovery. Increased diastolic blood pressure is considered to be the predominant precursor of cardiovascular distress (Ganster et al., 1991). Further, increased diastolic blood pressure is consistently related to hostility (Houston, 1986).

Together, these results suggest that hostility is the dimension of Type A behaviour which is the most compelling predictor of increased sympathetic activity (Ganster et al., 1991). This suggestion is strengthened by Svebak et al.'s (1992) research showing that hostility is significantly associated with increased sympatho-adrenal reactivity which is a risk factor for CHD (Krantz & Manuck, 1984). Thus, the hyperreaction to challenge and subsequent slower physiological recovery associated with hostility contributes to the deterioration of the cardiovascular system (Ganster et al., 1991).

A more direct link between hostility and the development of CHD has been established. In a 25 year prospective study focusing on physicians, Barefoot, Dahlstrom and Williams (1983) determined that hostility is associated with the severity of coronary atherosclerosis and an increased incidence of morbidity and mortality from CHD. Shekelle, Gale, Ostfeld and Paul (1983) and Williams et al. (1980) report similar results. As prospective studies (e.g., Haynes et al., 1980; Matthews et al., 1977) have established an association between hostility and CHD and hostility and mortality (Barefoot et al., 1989) it is probable that hostility constitutes a toxic component of Type A behaviour.

Significant relationships between hostility and the development of CHD have also been reported in studies which simultaneously failed to yield a significant relationship between the global measure of Type A behaviour and CHD. For example, MacDougall, Dembrowski, Dimsdale and Hackett (1985) reanalyzed data collected by Dimsdale et al. (1979) to assess the relationships between both global Type A behaviour and the extent of angiographically[31] determined CHD and hostility and the extent of CHD. The results of MacDougall et al.'s (1985) study confirmed the non-significant association between global Type A and the severity of CHD reported by Dimsdale et al. (1979). However, this study also revealed a significant relationship between hostility and the severity of CHD (MacDougall et al., 1985). Thus, retrospective studies have found hostility to be associated with

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[31] *'Angiographic' implies the use of angiography. An angiography comprises an X-ray examination of the coronary arteries (Kamarck & Jennings, 1991; Oxford Reference Concise Medical Dictionary, III Edition, 1990). An angiography provides the best measure of the extent of atherosclerosis (Loscalzo, 1990).*

angiographically determined atherosclerosis, in instances where Type A was unrelated to vessel disease (Demorowski, MacDougall, Williams, Haney, & Blumenthal, 1985). A meta-analysis (Matthews, 1988) of studies concerning Type A components and CHD also indicates that measures of hostility correlate with CHD even in cases where measures of global Type A behaviour do not. Clearly, hostility is independently associated with the incidence of primary CHD.

There is also some evidence to suggest that the hostility precipitating primary CHD also contributes to the incidence of secondary CHD. For example, Koskenvuo et al.'s (1988) study assessed the relationship between hostility and CHD in a sample of 3750 male twins. At the study's inception, the incidence of angina pectoris among subjects high in hostility was 2.88 times that of subjects low in hostility. A three year follow up failed to reveal an association between hostility and CHD morbidity and mortality among subjects who were free of hypertension and CHD at the study's inception. However, among subjects who were diagnosed with hypertension and CHD at the outset, hostility significantly predicted the incidence of subsequent CHD morbidity and mortality (Koskenvuo et al., 1988). Thus, while hostility did not predict primary CHD, it did predict secondary CHD. Further, hostility constituted the strongest predictor of reinfarction in a four year follow-up of the WCGS (Matthews et al., 1977). These results suggest that the hostility expressed by Type A CHD patients will increase the risk of reinfarction.

Anger

Like hostility, anger is considered a principal component of Type A behaviour (Palmer et al., 1992) and a strong predictor of CHD (Houston & Vavak, 1991; Taylor & Cooper, 1989). The literature concerning anger is fraught with ambiguity and confusion because anger is frequently, and erroneously, used interchangeably with the terms aggression and hostility (Spielberger et al., 1985). Anger is an affective emotional state encompassing subjective feelings of irritation, tension, aggravation, fury and rage (Spielberger, Jacobs, Russel & Crane, 1983). It is associated with a high tendency to perceive people or situations as challenging and threatening (Kennedy, 1992). Characterised by anger, aggression comprises overt, destructive or punitive behaviour (Feldman-Alschuler & Alschuler, 1984). Hostility, on the other hand, is an attitudinal dimension or remnants of the negative emotions which persist after anger has subsided (Feldman-Alschuler & Alschuler, 1984).

Anger occurs as a response to events which are appraised as frustrating, repugnant, or annoying and which elicit aggressive, antagonistic or withdrawn behaviour (Lopez & Thurman, 1986). Personal characteristics dictate that some individuals are more prone to appraise situations as provocative and hence, experience anger with a greater frequency (Kernis, Grannemann, & Barclay, 1989). Frequent provocation results in a state of chronic anger (Novaco, 1985). Individuals who are predisposed to chronic anger are typically combative in their response to threat, challenge, stress and adversity (Novaco, 1985). A combative orientation is an intrinsic characteristic of Type A behaviour (Friedman & Rosenman, 1974). Indeed, Type A's experience and express more anger than Type B's (Janisse, Edgeur, & Dyck, 1986).

The experience of anger is associated with augmented cardiovascular reactivity (Rosenman, 1986; Spielberger et al., 1985) and the release of epinephrine and norepinephrine (Walding, 1991). Of all Type A components, anger appears to illicit the greatest cardiovascular reactivity (Rosenman, 1986). It is through the increased cardiovascular reactivity implicated in the pathogenesis of CHD (Rosenman, 1978), that anger affects health (Johnson & Broman, 1987). More specifically, an habitual angry response to daily stressors raises blood pressure and ultimately contributes to hypertensive status (Harburg, Gleiberman, Russell, & Cooper, 1991; Maes et al., 1987). Anger also contributes to the development of atherosclerosis and CHD (Booth-Kewley & Friedman, 1987). Sustained hypertension is a precursor of reinfarction (Diamond, 1982). So too are atherosclerosis and CHD (Booth-Kewley & Friedman, 1987; Norris, Caughey, Deeming, Mercer, & Scott, 1970). Because anger is a typical response to the pain and discomfort of a coronary event (Fishman-Alschuler & Alschuler, 1984), and continues to exert an influence on the convalescing heart (Dembroski et al., 1985), anger is implicated in the development of secondary coronary events (Harburg et al., 1991).

Modes of Anger Expression

While anger is a relatively stable personality trait, its expression varies in intensity according to the demands made on the individual (Spielberger et al., 1983). As such, its expression constitutes a response to situationally determined demands (Fishman-Alschuler & Alschuler, 1984). This response can take one of two conceptually distinct forms, namely, suppressed anger (anger-in) and expressed anger (anger-out) (Harburg et al., 1991). Both forms of expression represent resentful, impulse controlled reactions which sustain the experience of anger

(Harburg et al., 1991). Again, both are reflected by the salient physiological manifestations of cardiovascular and endocrine arousal (Novaco, 1985). Anger-in, however, typically creates more psychological distress and therefore, initiates more pervasive physiological responses (Russek et al., 1990).

Anger-in.

Anger-in reflects a reluctance to express feelings of anger overtly even under circumstances where such expression would be warranted (Dembroski et al., 1985). It is particularly evident in situations where anger expression carries the potential for interpersonal conflict (Dembroski et al., 1985). Anger that is suppressed has to find expression elsewhere and is typically expressed physiologically (Budnick, 1991). The extreme effort required to suppress feelings of anger increases the reactivity of the cardiovascular system (Gilbert & McArthur, 1988). Chronic cardiovascular arousal ultimately leads to hypertension (Spielberger, Krasner, & Solomon, 1988) and CHD (Dembroski et al., 1985; MacDougall et al., 1985).

Research has consistently shown an association between anger-in and elevated blood pressure and hypertension among young and middle aged hypertensive and healthy individuals (e.g., Baer, Collins, Bourenhoff, & Ketchel, 1979; Falkner, Onesti, & Hamstra, 1981; Gentry, Chesney, Gary, Hall, & Harburg, 1982; Manuck, Morrison, Bellack, & Polefrone, 1985). As sustained hypertension is a risk factor for CHD (Jenkins, 1988), anger-in is associated with the development of coronary disease.

Empirical studies attest to a direct association between anger-in and the presence and severity of CHD. MacDougall et al. (1985) report a significant relationship between anger-in and the extent of CHD. Angiographic studies by Tennant and Langehuddecke (1985) show that anger-in predicts atherosclerosis. In a study comprising patients with minimal to severe CHD, Dembroski et al. (1985) report significant correlations between anger-in and angiographically determined severity of arterial occlusion, symptoms of angina and number of experienced myocardial infarctions.

A relationship between anger-in and both primary and secondary CHD has also been established. For example, the Framingham study showed that anger-in retrospectively predicted primary CHD among men and women (Haynes et al., 1980). Wright (1988), on the other hand, assessed the prevalence of Type A components in 40 cardiac patients with the aim of clarifying each components' role in the development of disease once traditional risk factors had been partialled out. Results showed anger-in to be a powerful predictor of recurrent CHD. Anger-in is associated with chronic cardiovascular arousal which is a precursor of hypertension and CHD. Anger-in is also significantly related to secondary CHD. Considering the relationship between anger-in and the development, maintenance, and recurrence of CHD, it is likely that the anger suppressed by the CHD patients in the present research will have deleterious psychological and physiological consequences.

Anger-out.

Anger-out is expressed verbally or physically and is directed at the source of provocation or at an allied person or object (Spielberger et al., 1988). The

behavioural limitations imposed by CHD elicit anger which is typically directed at the medical personnel involved in the process of rehabilitation or at the disease itself (Rhodewalt & Fairfield, 1991). The habitual expression of anger demands frequent bursts of exaggerated autonomic activity (e.g., accelerated heart rate and cardiac output) (Fishman-Alschuler & Alschuler, 1984). Exaggerated cardiovascular activity is believed to be the mechanism linking anger-out to CHD (Diamond, 1982). This belief is supported by the research reviewed below.

Harburg et al. (1991) revealed that among older black and white men, anger-out was significantly related to elevated systolic and diastolic blood pressure. Engebretson et al. (1989) demonstrated that anger provocation initiated heightened cardiovascular reactivity and that this was more pronounced when subjects were unable to use their preferred mode of anger expression. Subjects who typically expressed their anger exhibited significant elevations in blood pressure and heart rate when they were harassed but prevented from showing their harassment (Engebretson et al., 1989). Elevations persisted 25 minutes after the harassment procedure had terminated, suggesting that a substantial amount of time is required before the cardiovascular system returns to baseline functioning. Habitual and prolonged cardiovascular activation is dysfunctional, particularly as it is accompanied by the release of catecholamines (Krantz et al., 1985). An association between the arousal of anger and increased levels of the catecholamine, norepinephrine has been established (Funkenstein, King, & Drolette, 1954). Increased levels of circulating catecholamines are implicated in the development of atherosclerosis (Haft, 1974), and ultimately, CHD (Glass, 1977b). Therefore, the cardiovascular reactivity generated by anger-out plays a potential role in the development of CHD.

Other research has gone beyond linking anger-out to cardiovascular arousal to establish direct links between anger-out and general health, hypertension and CHD. Johnson and Broman's (1987) study of 1277 black adults revealed that anger expression predicted health problems independently of age, sex and traditional CHD risk factors. Other studies (Gentry, 1985; Spielberger et al., 1985) have established significant relationships between anger-out and hypertension and more specific relationships to secondary CHD have been reported by Wright (1988). In Wright's (1988) study, anger-out correlated with the incidence of CHD when statistically combined with time urgency and chronic activation. The correlations between anger-out and CHD occurred independently of the traditional risk factors and were higher than the correlations between the global Type A construct and CHD (Wright, 1988). A later study by the same author revealed that CHD patients demonstrated significantly more anger-out than non-CHD patients (Wright, 1992). These results suggest that anger-out may be a primary differentiating factor between CHD patients and patients who are suffering other diseases (Wright, 1992). Given that anger-out is associated with heightened cardiovascular reactivity, general ill health, hypertension and secondary CHD, it is plausible that the anger expressed by CHD patients in the present research will compromise their health.

Research on the components of Type A behaviour reveals that achievement striving, impatience irritability, hostility, anger-in and anger-out are characteristics of Type A behaviour which, with the exception of achievement striving, can result in sustained, intemperate reactivity, culminating in physiological processes which precipitate the development of CHD (Taylor & Cooper, 1989). Taken together, this body of research indicates that each component may exert a differential effect, be it on health or performance. Thus, this research provides support for a

multidimensional model of Type A behaviour which comprises both benign and coronary-prone components. It must be recognised however, that this body of research is undermined by the limitations discussed below.

Limitations of Type A Component Research

Type A component research is characterised by three fundamental weaknesses. First, as argued previously, the majority of studies discussed are confined to low-risk populations, and cannot be generalised to high-risk populations. Second, the majority of the Type A component studies are correlational in design. Correlational research generates data which is intrinsically ambiguous and possesses questionable internal validity (Lepore, Allen, & Evans, 1993). Consequently, it is difficult to determine whether measures of health and performance are influenced by the Type A components themselves, or by some third factor. In failing to consider, and hence control, factors which may ameliorate, diminish or exacerbate the effects of the Type A components, these studies have a limited ability to make definitive statements regarding relationships between Type A components and measures of health and performance. Theoretically, the number of third factors is infinite (Brown, 1991). However, one factor holds particular relevance to cardiac rehabilitation patients, namely, aerobic exercise. Regular exercise modifies the negative components of Type A behaviour (Barefoot et al., 1989). Therefore, in addition to enhancing cardiovascular health, exercise may also influence the relationship between Type A components and CHD patients' psychological and physiological strain.

Third, many of the Type A component studies make inadequate attempts to specify the physiological outcome of the Type A components. Those studies which do specify outcomes (e.g., B. ling & Charbonneau, 1992; Bluen et al., 1990), typically utilise self-report measures of ill health. Therefore, the task of measurement is left to the subject (Brown & Harris, 1978). Personality type imposes a profound influence on the perception, recollection and reporting of subjectively determined health measures (Rimé et al., 1989), and consequently, produces spurious associations (Marmot & Madge, 1987). For example, neuroticism, which is highly correlated with Type A behaviour, is related to the over-reporting of physical symptoms (Suls & Marco, 1990). In these cases, positive relationships between Type A behaviour and ill health may be a product of the Type A individual's predisposition towards neurotic over reporting and not to a higher incidence of disease (Suls & Marco, 1990). Moreover, it is naive to assume that individuals can translate their perceived state of ill health to a complex physiological condition with sufficient precision (Costa & McCrae, 1987). Research in this area (e.g., LaRue, Bank, Jarvik, & Hetland, 1979; Linn & Linn, 1980) has shown that subjective reports of ill health are only modestly correlated with objective indicators of ill health (e.g., physicians' ratings) (Suls & Marco, 1990). Thus, if self-report measures are used to establish psychological variables (e.g., Type A components), then objective measures should be used to determine ill health (Marmot & Madge, 1987). The use of different modes of assessment for psychological and ill health variables ensures that the amount of influence that the former exerts on the latter is limited (Marmot & Madge, 1987). The use of objective physiological measures is of particular importance when the variables under study are implicated in a stress-strain relationship which includes physical condition (Strasser, 1981). Such is the case with certain of the Type A

components.

The present research will overcome these weaknesses by means of the following: First, the relationship between the Type A components and health will be examined among a high-risk sample, namely, cardiac rehabilitation patients. Second, the present research will determine whether exercise plays an equally predictive role. Third, the impact of the five Type A components will be analysed in relation to a specific, objectively determined index of cardiac health (i.e., maximal oxygen uptake).

The examination of the Type A behaviour which characterises CHD patients will require the development of a Type A measure which reflects the components relevant to a high-risk sample. It is possible that associations between the behaviour pattern and cardiovascular reactivity, CHD morbidity and mortality may only occur with certain measures of Type A behaviour (Edwards, Baglioni, & Cooper, 1990b; Thomas & Friedman, 1990). Lack of precision of existing measures and the failure to calibrate independent components may cause non-significant associations between these variables (Chesney, Ekman, Friesen, Black, & Hecker, 1990).

Given that the multidimensional nature of Type A behaviour (Friedman & Rosenman, 1974) is inadequately represented by existing measures of the global construct, measures which reflect the full constellation of Type A components will have to be developed for this purpose (Edwards et al., 1990a; Meininger, Stashinko & Hayman, 1991). Only once these steps have been taken, can those components which increase prediction of recurring CHD be assessed (Evans, 1990). Therefore, one of the aims of the present research is to refine a multidimensional scale to

measure the five components of Type A behaviour outlined in this chapter, namely, achievement striving, impatience irritability, hostility, anger-in and anger out.

Conclusion

While research concerning the Type A-primary CHD relationship is equivocal, research into the association between Type A behaviour and secondary CHD suggests an inverse relationship. It is possible that the lack of consistency in results yielded by both primary and secondary CHD research can be attributed to the definition and measurement of Type A behaviour as a global construct. Global Type A behaviour has proved to possess inadequate predictive validity (Evans, 1990). Moreover, there is an accumulating body of research which indicates that the global Type A construct is heterogeneous in nature and comprises both benign and coronary prone components (Abbott & Peters, 1988). The benign component is believed to be achievement striving (Öhman et al., 1989) while the coronary prone components are thought to be impatience irritability, hostility, anger-in and anger-out (Johnson & Broman, 1987; Matthews, 1988; Öhman et al., 1989; Taylor & Cooper, 1989). In order to determine the impact of behaviour on prognosis, these components need to be differentiated from the global construct. Further, the health and performance consequences of each component should be evaluated individually.

Before such evaluation can occur it is necessary to identify precisely which of the above components characterise CHD patients' Type A behaviour. There is evidence to suggest that CHD alters the influence of Type A behaviour on health outcomes (Evans, 1990). Therefore, the nature of Type A behaviour as defined by component research concerning low-risk samples may not apply to a sample of cardiac

rehabilitation patients. The present research will attempt to define the Type A behaviour of CHD patients. This will be achieved by recording the responses of CHD patients on measures designed to assess achievement striving, impatience irritability, hostility, anger-in and anger-out. The construct validity of the resulting multidimensional model of CHD patients' Type A behaviour will then be assessed by factor analysing the data (see Chapter 7.). Subsequently, the components of Type A behaviour will be included and tested in a model designed to assess CHD patients psychological and physiological prognosis for recovery (see Chapter 8). Further, a criticism of past research will be addressed by including exercise in this model and determining its influence on cardiac patients' prognosis.

CHAPTER FIVE

AIMS AND RATIONALE OF THE PRESENT RESEARCH

From the preceding chapters it is evident that CHD patients' Type A and exercise compliance behaviour has attracted considerable theory and research. It is also evident that the research into these two areas of CHD patients' behaviour has yielded inconsistent and controversial results. In the present chapter, it is argued that the controversy and inconsistency surrounding research into CHD patients' behaviour can be attributed to the empirical and conceptual limitations of past research. By overcoming these limitations in the present research, it will be possible to identify factors which influence CHD patients' physiological and psychological recovery. The present chapter describes the aims and rationale of the three studies which have been designed to examine CHD patients' behaviour and its impact on physiological and psychological recovery.

Study 1: The Psychological and Physiological Consequences of Attending vs. Not Attending an Exercise Based Cardiac Rehabilitation Programme

CHD has typically been viewed as a negative physiological outcome of the stress process (Quick & Quick, 1984). Relatively little attention has been paid to the stress and resultant strain caused by CHD. In the present research it is argued that CHD generates numerous stressors which have the potential to culminate in psychological and physiological strain (Rejeski et al., 1985). Exercise moderates these stressors, and consequently, reduces the amount of strain experienced by

CHD patients (Folkins & Sime, 1981). It is thus plausible that exercise enhances the prognosis of CHD patients (Kinnaird et al., 1982). However, knowledge of the effect of exercise on CHD patients' prognosis is inconclusive and incomplete (Holloszy, 1983; Kinnaird et al., 1982; Widimsky' & Broustet, 1985). This can be attributed, in part, to the methodological limitations of past research outlined below.

A primary limitation of past research is the insufficient duration of the exercise programmes included in several key studies. For example, the six week exercise programme of the NEHDP (1981) and the 12 week exercise programme of Valliant and Asu's (1985) study did not provide enough time for exercise to exert an influence on prognosis (Ismail & Young, 1977). Other studies (e.g., Kavanagh et al., 1977; NEHDP, 1981) did not include non-exercising control groups and therefore, were unable to determine whether changes in prognosis were due to exercise or to the effects of time.

A further limitation concerns those studies which included a control group comprising CHD patients who chose to withdraw from the exercise programme (e.g., Kellerman, 1973; Shephard et al., 1981) but which failed to establish these patients' reasons for withdrawing from the programme. The prerandomisation trial of the NEHDP (1981) demonstrates that cardiac patients who drop out of an exercise programme tend to suffer more physiological and psychological ill health than those who remain in the programme. It is possible that the control groups of the above mentioned studies withdrew from the rehabilitation programme for reasons of psychological or physiological ill health. Given this possibility, the reported higher levels of strain are likely to be a product of ill health at the time of withdrawal and not a product of a sedentary lifestyle. Thus, in failing to determine the relevant

reasons for leaving, the studies conducted by Kellerman (1973) and Shephard et al. (1981) could not attribute the higher incidence of strain among the control group to a failure to exercise.

A similar flaw lies in the failure to establish any differences in the experimental and control groups' biographical and risk factor data (e.g., Kavanagh, Shephard, Doney, & Pandit, 1973). The results of past research suggest that CHD patients who drop out of an exercise programme have a different risk factor and biographical profile to patients who elect to remain in an exercise programme. For example, in the prerandomization trial of the NEHDP, CHD patients who dropped out of the programme were more likely to be categorised as middle vs. upper middle class and to be unmarried (Stern & Cleary, 1981). Blue collar workers suffer more CHD morbidity and mortality following an initial myocardial infarction than white collar workers (Hartley et al., 1987) and the incidence of reinfarction is greater among unmarried CHD patients (Stern et al., 1977). Therefore, it is possible that both social class and marital status influence prognosis for recovery. The drop-out group assessed in the NEHDP also reported significantly higher cholesterol levels and a greater tendency toward elevated blood pressure levels (Stern & Cleary, 1981). As the Coronary Drug Project Research Group (1974) has established that high cholesterol and blood pressure levels continue to exert independent effects on outcome following an initial infarction, it is likely that cholesterol and blood pressure also influence recovery. Given that drop-outs of an exercise programme have been found to differ from programme participants, it is necessary to control for any such differences by either including them as covariates in the analysis or by closer matching of groups.

The aim of the first study is to address the above criticisms and consequently, establish whether participation in an exercise programme affects cardiac patients' psychological condition and the incidence of subsequent cardiac events and medical interventions. It is believed that this aim will be achieved by overcoming the weaknesses of past research. Specifically, the present study will be based on a six month exercise programme, which, according to the American Heart Association (Erb et al., 1979), is the exercise period needed for the physical reconditioning of the CHD patient.

The study will also include a control group comprising CHD patients who dropped out of the exercise programme. By including a control group, the effects of time will be controlled. In order to eliminate the potentially confounding factor of pre-existing differences in psychological and physiological health, the control groups' reasons for leaving the programme will be determined. Those CHD patients who left the programme for reasons of ill health will be eliminated from the control group. This precaution will limit the possibility that the hypothesised significant difference between the levels of strain reported by the experimental and control groups at Time 2 is a result of a higher incidence of ill health among the control group and not the effects of the exercise programme. Thus, from Study 1 it will be possible to determine whether compliance with an exercise programme reduces physiological and psychological strain. The experimental and control groups will be matched on the basis of sex, race, marital status, education and occupational status thereby reducing the number of potentially confounding effects. Study 1 will also identify any pre-existing differences between the exercising and non-exercising groups' risk factor data. Variables reflecting a between-group difference will be included in the study as coveriates.

Unlike past research which has been criticised for using cross-sectional designs, and thus, has been unable to establish temporal relationships (Haynes & Matthews, 1988), Study 1 will be based on a longitudinal design. It is believed that in surmounting the methodological limitations of past research in Study 1, it will be possible to verify the influence of exercise on prognosis for recovery from CHD.

The present research is further concerned with the influence of Type A components on prognosis. Before this influence can be determined, it is necessary to conduct a second study to develop a valid measure of the Type A components manifested by CHD patients.

Study 2: Defining the Type A Behaviour of CHD Patients

While much research has been devoted to establishing Type A behaviour as a risk factor for primary CHD, very little research has focused on the role of Type A behaviour in the development of secondary CHD (Ragland & Brand, 1988b). Of those studies which have examined behaviour type in relation to secondary CHD, the majority have found that, contrary to expectations, Type A behaviour promotes a positive prognosis (*cf.* Barefoot et al., 1989; Case et al., 1985; Dimsdale et al., 1981; Ragland & Brand, 1988b; Shekelle et al., 1985). The contrary findings have been attributed to the possibility that, once diagnosed with CHD, Type A's modify the toxic components of their behaviour and thus, enhance their prognosis for recovery (*cf.* Evans, 1990; Wright, 1988). However, the value of these findings is limited by a flaw in Type A research. That is, these studies have erroneously examined Type A

behaviour as a global construct (Edwards et al., 1990a). Type A behaviour is a multidimensional construct (Friedman & Rosenman, 1974; Tett et al., 1992), comprising components which exert differential effects on illness and performance outcomes (Jennings, 1984).

To determine the impact of Type A behaviour on prognosis, it is necessary to take cognisance of the multifaceted nature of the construct. This requires the development of a model of Type A components. Existing models of Type A behaviour (e.g., Burns & Bluen, 1992) have been developed for low-risk populations. Perception and methods of coping with CHD are influenced by personality factors (Martin & Lee, 1992). Type A's are known to modify the toxic components of their behaviour following diagnosis of CHD (Evans, 1990; Friedman et al., 1986). Such modification may alter the composition and intensity of Type A components manifested by CHD patients (Haynes & Matthews, 1988; Matthews, 1988). As a result, the difference between the Type A behaviour manifested by healthy individuals and individuals afflicted with CHD may be substantial (Anderson et al., 1986; Evans, 1990). Given that CHD has been found to affect both the nature of Type A behaviour (Evans, 1990) and coping methods (Martin & Lee, 1992), these models are not suitable for research concerning CHD patients.

The criticisms of past research will be overcome in Study 2 by first considering Type A behaviour as a multidimensional, and not a global, construct. Second, the extent to which achievement striving, impatience irritability, hostility, anger-in and anger-out feature within the behaviour of cardiac rehabilitation patients will be examined in Study 2. Therefore, the aim of Study 2 is to develop and test a multidimensional model of Type A components which is relevant to people with CHD.

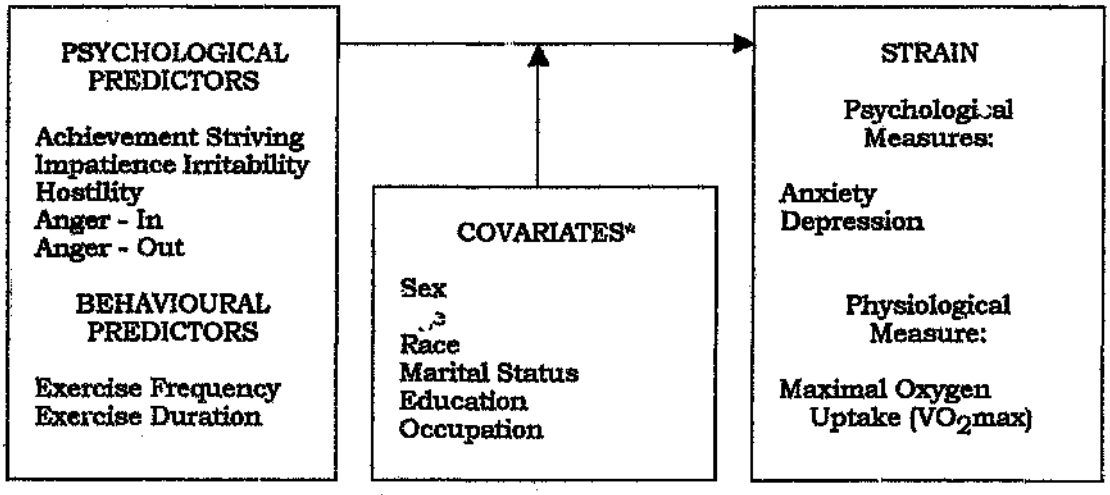
Study 3: Assessment of the Relationships Between Type A and Exercise Components and Indices of Psychological and Physiological Health

Aerobic exercise and the dimensions of Type A behaviour are believed to predict prognosis for recovery from CHD (Dembroski et al., 1989; Holmes & Roth, 1985; Matthews, 1988; McGlynn et al., 1983; Morris et al., 1990; Öhman et al., 1989). Research has examined the prognostic role of these variables in isolation (*cf.* Dembroski et al., 1985; Dimsdale et al., 1977; Grodzinski et al., 1987; Kallio et al., 1979; Matthews et al., 1977; Rimé et al., 1989; Shephard et al., 1981). As a result, little is known of the joint predictive capacity of aerobic exercise and Type A behaviour in the prognosis of CHD patients. Further, research which has examined exercise in relation to health outcomes has largely neglected a key variable, namely, the duration of exercise. Without including the measure of duration along with the usual measure of frequency, past research has been unable to demonstrate the precise mechanisms of exercise which predict prognosis (Sharkey, 1984). Research concerning Type A behaviour and prognosis can be criticised on a similar basis. That is, by considering Type A behaviour as a global construct, this research has limited the possibility of examining the various Type A components and the extent to which they differentially predict prognosis.

The aim of Study 3 is to develop a multidimensional model comprising psychological and behavioural variables which predict CHD patients' prognosis for recovery (see Figure 5.1). The psychological predictor variables included in the model will be the achievement striving, impatience irritability, anger-in, anger-out and hostility components validated in Study 2. The behavioural predictor variables will be the frequency and duration measures of exercise compliance discussed in Chapter 3. The predictor variables will be assessed in relation to psychological outcomes, namely, anxiety and depression and a physiological outcome which reflects changes in the cardiovascular system, namely, maximal oxygen uptake (Sami et al., 1979; Uhl et al., 1984). Study 3 will determine whether the components of Type A and exercise behaviour differentially predict changes in anxiety, depression and maximal oxygen uptake. In conclusion, it is hoped that by addressing the empirical and conceptual limitations of past research, the three studies comprising the present research will advance knowledge of the factors which influence CHD patients' psychological and physiological health.

Figure 5.1

Model of the Psychological and Behavioural Predictors of Prognosis



* As measured at Time 1

CHAPTER 6

STUDY 1: THE PSYCHOLOGICAL AND PHYSIOLOGICAL CONSEQUENCES OF ATTENDING VS. NOT ATTENDING AN EXERCISE BASED CARDIAC REHABILITATION PROGRAMME

CHD is physically and psychologically stressful (Rejeski et al., 1985). Consequently, it has the potential to generate physiological and psychological strain (Brown & Munford, 1984). This translates to a poor prognosis for recovery. Exercise is believed to affect prognosis by restoring cardiorespiratory fitness (Morris et al., 1990; Shephard, 1985) and modifying the stress - strain relationship characteristic of the recovery period (Holmes & Roth, 1985; McGlynn et al., 1983). Specifically, the exercise which improves prognosis is that which involves repetitive, dynamic movement and results in increased oxygen consumption, namely, aerobic exercise (Hammond, 1985; Morris et al., 1990; Sharkey, 1984). Regular aerobic exercise retards the development of atherosclerosis (Oberman, 1983) and the consequent cardiac performance decrement (Schneider & Reed, 1985). Thus, it reduces the physiological strain manifested by cardiac patients (Schneider & Reed, 1985).

From the studies reviewed below, it is evident that research concerning the physiological effects of exercise is equivocal. Several studies report significant physiological improvements among cardiac patients following participation in an exercise programme (e.g., Bethell & Mullee, 1990; Grodzinski et al., 1987; Kavanagh, Shephard, Pandit, & Doney, 1970; McGlynn et al., 1983; Schneider & Reed, 1985; Widimsky' & Broustet, 1985). An inverse relationship between exercise

and CHD mortality among both cardiac patients and healthy individuals has also been demonstrated (e.g., Cay et al., 1985; Kellerman, 1973; Paffenbarger, Hyde, Wing, & Steinmetz, 1984; Shaw, 1981; Shephard et al., 1981). However, other studies have failed to demonstrate significant differences between the morbidity and mortality rates of exercising and non-exercising groups of CHD patients (e.g., the OERCB, Pias et al., 1985; Stern et al., 1983). Therefore, research has failed to yield consistent support for the positive effect of exercise on the physiological health of CHD patients.

Aerobic exercise is also believed to affect prognosis by moderating the psychological states experienced by cardiac rehabilitation patients. Primary manifestations of CHD in cardiac patients are anxiety and depression (Brown & Munford, 1984). Exercise provides both a distraction from sources of anxiety and a respite from stress (Brown, 1991; Wood, 1977). The respite created by exercise facilitates coping behaviour which increases immunity to further anxiety provoking stimuli (Selye, 1974; Wood, 1977). This immunity is enhanced by the way in which individuals perceive their responses to such stimuli. Individuals attribute their current level of arousal to the most salient stimulus (Zillman, 1983). As individuals increase their level of fitness, they tend to interpret the high levels of arousal induced by anxiety as a product of the positive feelings of invigoration induced by exercise (Roth, Bachtler, & Fillingim, 1990). They thus learn to associate high arousal levels with a positive sense of vigour rather than with a state of tension (Roth et al., 1990). An increased level of fitness further serves to quell anxiety concerning reinfarction (Soloff, 1978b). By enhancing self-concept through improved body image and sense of well-being (Folkins & Sime, 1981; Goldberg & Folkins, 1974; Layman, 1974), and restoring a

measure of autonomy, exercise reduces the degree of depression typically experienced by cardiac patients (Soloff, 1978a).

Considerable research has established that exercise reduces cardiac patients' anxiety (Folkins, 1976; McGlynn et al., 1983; Schomer, 1985; Stern et al., 1983) and depression (Folkins, 1976; Kavanagh et al., 1977; Schomer & Neakes, 1983; Stern et al., 1983; Valliant & Asu, 1985). Conversely, the prerandomisation exercise programme of the NEHDP has shown that, although exercise reduces cardiac patients' depression, it increases anxiety (Stern & Cleary, 1981). The increase in anxiety levels has been attributed to the possibility that exercise may accelerate the production of stress hormones and consequently impose a negative effect (Holloszy, 1983). However, it should be noted that the NEHDP prerandomization trial comprised a six week exercise programme and that the studies reporting decreases in anxiety have utilised exercise programmes of a longer duration (e.g., Erdman et al., 1986; Shephard et al., 1985). It is possible that exercise only reduces anxiety levels in the long term. Nevertheless, when combined with the equivocal findings of the physiological benefits of exercise, this finding serves as a reminder that extant knowledge of the relationship between aerobic exercise and improved physiological and psychological health remains obscure (Holloszy, 1983; Widimsky' & Broustet, 1985).

Failure to demonstrate consistently the value of exercise can be attributed to several methodological flaws in past research. For example, the absence of non-exercising control groups (e.g., Kavanagh et al., 1977; the prerandomisation exercise programme of the NEHDP, 1981; OEHCS, 1985) makes it impossible to determine

whether improvements in health are the result of maturation effects or of exercise (Kerlinger, 1986).

Those studies which have included a group of non-exercising CHD patients as controls (e.g., Kellerman, 1973; Shephard et al., 1981) have not established the groups' reasons for not continuing their participation in the exercise programme. Lack of participation may have been due to a higher incidence of psychological and physiological morbidity among the control group. Therefore, it is difficult to ascertain whether any differences between the experimental and control groups' levels of strain are the result of participation in the exercise programme or of an intrinsically healthier experimental group. Existing research (Kavanagh et al., 1975) can also be criticised for failing to account for the potentially confounding effects imposed by biographical and risk factor data.

Other studies (e.g., Fletcher et al., 1992; Mittleman et al., 1993; Paffenbarger et al., 1984) can be criticised for inadequately manipulating degree of fitness. That is, these studies have relied on participants' own reports of participation in a variety of different leisure-time sports and physical activities (e.g., climbing stairs) to calculate the amount of exercise needed to reduce CHD risk. The effect of different exercise regimens varies considerably (Tunstall Pedoe, 1990). Without specifying the form of exercise or measuring the outcome in terms of precise objective measures, conclusive statements regarding the affect on prognosis cannot be made. Several studies (e.g., NEHDP, 1981; Valliant & Asu, 1985) have also utilised exercise programmes of insufficient length (six and 12 weeks, respectively). It is possible that statistical differences in disease severity may only be discernible after a three month time lapse (Suls & Marco., 1990). Regular exercise training for a minimum of four

months is needed to alter body chemistry (Ismail & Young, 1977). Changes in psychological states such as anxiety and depression, which are associated with body chemistry, are unlikely to occur within a short space of time (Folkins & Sime, 1981). Because the therapeutic benefits of exercise only become apparent after six months of participation in an exercise programme (Erb et al., 1979; Shephard, 1979) a longer programme would yield more convincing results.

The aim of Study 1 is to establish whether participation in an exercise programme affects cardiac patients' CHD related morbidity rates and their psychological condition. In designing the study it is intended to overcome the weaknesses of past research. First, the study includes a control group, comprising CHD patients who dropped out of the exercise programme. Second, the control group's reasons for leaving the programme are established. These will be used to determine whether any control group candidates left the programme for reasons of psychological or physiological ill health. Given that the inclusion of any such individuals in the control group would increase the possibility of spurious results, individuals who attributed their withdrawal to psychological or physiological ill health will be excluded from the study. Third, the experimental group will be established by randomly selecting an equal number of participants in a rehabilitation programme who match the control group on the basis of sex, race, and marital, occupational and educational status. Fourth, steps are taken to identify any pre-existing differences between the experimental and control groups' medical history and risk factor variables. Where differences exist, the relevant variables are included in the study as covariates. Therefore, every attempt will be made to eliminate potentially confounding factors. Fifth, objective, standardised methods will be used manipulate and measure exercise. That is, each participant's exercise prescription

will be calculated by means of a standardised formula and their degree of fitness will be measured by a standardised incremental stress test. Sixth, in keeping with the recommendations made by the American Heart Association (Erb et al., 1979), the study will be based on a six month exercise programme. Finally, the study utilises a longitudinal design. Longitudinal designs supersede cross sectional designs in that they determine temporal precedence and thus provide stronger evidence of causal inference concerning sequences and relationships (Biddles et al., 1985; Kasl, 1983). Longitudinal designs also provide the best means of validating the long term adequacy of a rehabilitation programme (Rahe, Ward, & Hayes, 1979). Thus, by using a longitudinal design, the present study will be able to infer causality regarding the impact of participation in an aerobic exercise programme on psychological and physiological well-being.

Hypothesis 1a: Controlling for any pretest differences, participants in the cardiac rehabilitation programme will experience significantly less anxiety and depression after six months of aerobic exercise than cardiac patients who withdrew from the programme.

Hypothesis 1b: It is hypothesised that following the control of pretest differences, participants in a cardiac rehabilitation programme will experience fewer cardiac events or associated surgical procedures than CHD patients who were accepted into the same programme but who chose not to participate in the exercise sessions.

Method

Setting

Participants in the present research (i.e., Studies 1, 2, & 3) were either current or past members of the Johannesburg City Health Department Cardiac Rehabilitation Centre (CRC). The CRC is a community based facility which offers a rehabilitation programme free of charge to eligible cardiac patients (Digenio et al., 1991). A cut off age of 70 is loosely applied. Admission to the CRC is based exclusively on medical referral and occurs eight to 12 weeks after hospitalisation (Digenio et al., 1991). Each participant must have experienced one or more of the following: recent myocardial infarction, coronary artery bypass graft, coronary artery angiography, percutaneous transluminal angioplasty, stable angina pectoris, or recent valvular or congenital heart disease surgery. Thus, each participant must have a confirmed diagnosis of heart disease.

The first six months of the CRC's programme comprises one hour, triweekly, exercise sessions held under close medical supervision. In addition, a group discussion, covering a variety of issues concerning CHD, risk factor modification and rehabilitation is held once a week. Pending satisfactory results from the psychometric and exercise tests conducted after six months, participants are allowed to proceed to the second phase of rehabilitation which consists of supervised exercise three times a week, only. The group discussion is eliminated from this phase. The second phase typically lasts for twelve months, after which participants exercise at home and are only required to submit an exercise report on

a monthly basis. Participants who exhibit a high risk of reinfarction are provided with supervised exercise on an indefinite basis.

Sample

Experimental Group

To reduce the bias inherent in non-matched samples (Kerlinger, 1964; Wildt & Ahtola, 1980) and to eliminate the number of plausible confounds, the experimental group was established by randomly selecting 20 cardiac patients whose race, sex and marital, educational and occupational status matched that of the 20 members of the control group (see Table 6.1). Attempts were also made to match the samples on risk factors. However, given the wide range of risk factor profiles, this could only be achieved with two of the five risk factors, namely, family history of CHD and treatment for high blood pressure. Members of the experimental group had been enrolled for a minimum of six months in the CRC's programme. They had thus participated in both the initial psychological and physiological assessment, the six month exercise programme and the six month follow-up assessment. Their exercise performance had been monitored for the six months in terms of their attendance and duration of effort.

Table 6.1

Summary of the Biographical, Risk Factor and Medical History of the Experimental and Control Groups

Variable		Experimental (N=20)	Control (N=20)	Chi^2
Sex	Male	20	20	0.00
Race	White	20	20	0.00
Marital Status	Married	20	20	0.00
Education	10 years	3	3	0.00
	12 years	9	9	0.00
	Technical Qualification	4	4	0.00
	Professional Qualification	2	2	0.00
	University Degree	2	2	0.00
Occupation	Professional	6	6	0.00
	Skilled	10	10	0.00
	Self-employed / Retired	4	4	0.00
High Blood Pressure	Yes	4	4	0.00
	No	16	16	
Family History	Yes	11	11	0.00
	No	9	9	
Smoking	Current	1	3	8.70*
	Never	2	9	
	Stopped	17	8	
High Cholesterol	Yes	12	6	3.64
	No	8	14	
Diabetes	Yes	2	1	8.70
	No	18	19	
Myocardial Infarction	Yes	13	12	0.11
	No	7	8	
Coronary Artery Bypass Graft	Yes	12	9	0.90
	No	8	11	
Percutaneous Transluminal Angioplasty	Yes	2	1	0.36
	No	18	19	
Coronary Artery Angiography	Yes	12	9	0.90
	No	8	11	

* $p < .05$

Control Group

All participants who fulfilled the criteria for admission to the CRC but who subsequently withdrew from the programme and who had undergone their initial physiological and psychological assessment six months previously were identified as candidates for the control group. Ninety four such candidates were identified. Questionnaires and a letter from the CRC were mailed to the 94 candidates. The

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Summary of the Biographical, Risk Factor and Medical History of the Experimental and Control Groups

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Marital Status	Married	20	20	0.00
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	12 years	9	9	0.00
	Technical Qualification	4	4	0.00
	Professional Qualification	2	2	0.00
	University Degree	2	2	0.00
Occupation	Professional	6	6	0.00
	Skilled	10	10	0.00
	Self-employed / Retired	4	4	0.00
High Blood	Yes	4	4	0.00
Pressure	No	16	16	
Family History	Yes	11	11	0.00
	No	9	9	
Smoking	Current	1	3	8.70*
	Never	2	9	
	Stopped	17	8	
High Cholesterol	Yes	12	6	3.64
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Percutaneous Transluminal	Yes	2	1	0.36
Angioplasty	No	18	19	
Coronary Artery	Yes	12	9	0.90
Angiography	No	8	11	

* $p < .05$

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All participants who fulfilled the criteria for admission to the CRC but who subsequently withdrew from the programme and who had undergone their initial physiological and psychological assessment six months previously were identified as candidates for the control group. Ninety four such candidates were identified. Questionnaires and a letter from the CRC were mailed to the 94 candidates. The

letter stated the purpose of the study and requested the candidate's participation. The letter emphasised that participation was voluntary and that confidentiality and anonymity were guaranteed. Also included was a form of informed consent which required the candidate's signature and the counter signature of a witness (see Appendix B). Candidates were encouraged to return the completed questionnaire in the accompanying stamped, self-addressed envelope.

Of the 94 questionnaires sent, six were returned address unknown. Regrettably, a further three were returned due to the candidate's death following withdrawal from the CRC programme. Complete questionnaires were returned by 31 candidates. The response rate was thus 36%. Five of these indicated that they had left the programme for reasons of ill health and were eliminated from the sample. To limit the number of confounding variables, the one black, one unmarried and three female respondents were excluded from the analysis. The final sample consisted of 20 married, white males who had withdrawn from the CRC six months previously for reasons other than ill health (see Table 6.1).

Procedure

The majority of the data used in Studies 1, 2, and 3 was drawn from the archives of the Johannesburg City Health Department Cardiac Rehabilitation Centre (CRC). On admission to the CRC programme, participants were subjected to numerous psychometric, anthropometric and exercise tests. Both the experimental and control groups completed the baseline tests. All psychometric items assessed in Studies 1,2, and 3 derive from the questionnaire included in Appendix A. A customised exercise prescription was compiled by a physiotherapist according to the results of the

participant's exercise test. Participants were expected to exercise at their prescribed intensity for 30 to 45 minutes of continuous, dynamic effort three times a week. After six months of participation in the rehabilitation programme, participants were again subjected to the same battery of psychometric, anthropometric and exercise tests. The experimental group's data derive from the psychometric and medical status (i.e., number of cardiac and associated surgical events) measures taken at Time 1 and Time 2.

Copies of the psychological battery of scales were sent to the control group six months after their initial psychological and physiological assessment. While subjecting the control group to physiological tests at Time 2 would have been ideal, the CRC was unable to grant permission to do so. Instead, the control group was asked to answer questions concerning any change in medical status or risk factors which had occurred since their last assessment at the CRC.

Permission to use the CRC's data was granted by both the CRC and the Johannesburg City Health Department's ethics committee and approved by the University of the Witwatersrand's ethics committee. Permission was conditional on the author's strict adherence to the confidentiality and anonymity guaranteed by the CRC. This was achieved by assigning case numbers to all CRC members' files prior to the data being analysed. Therefore, no data identifiable to a particular patient left the CRC's premises. The CRC also granted the author permission to identify members who had elected to withdraw from the CRC's progam six months previously (i.e., the control group candidates for Study 1). However, to protect the rights of these individuals, the Johannesburg City Health Department's ethics committee stipulated that the addresses of these individuals remain unknown to the

author. Thus, the questionnaires were sent out by, and on behalf of, the CRC. The committee further stipulated that the questionnaires be accompanied by a letter from the CRC outlining the purposes of the research and a form of informed consent (see Appendix F). The committee required that the forms of informed consent be signed by the candidate in the presence of a witness. Thus every effort was made to protect the privacy and rights of past and present members of the CRC.

Design

A longitudinal design (Christensen, 1985) was used to test the hypothesis that the control group would experience more cardiac and associated surgical events during the intervening six months and would report greater levels of psychological strain at Time 2 than the experimental group. The independent variables included in the design were attendance or withdrawal from the exercise programme. Changes in the psychological measures of strain (anxiety and depression) and change in physical health status (i.e., cardiac and associated surgical events which occurred between Time 1 and 2) constituted the dependent variables. The Time 1 measures of anxiety, depression and history of tobacco abuse were included as covariates.

Measuring Instruments

Personal Details

On admission to the programme, participants were required to provide biographical information and details of their medical and risk factor history. Biographical data included age, marital status, sex, race and education. Medical history referred to the type and frequency of cardiac and surgical events and risk factor history to the classical CHD risk factors, namely, the amount and duration of nicotine abuse, hypertension, hyperlipoproteinaemia and diabetes mellitus, and incidence of CHD in the participant's immediate family. Any changes in the above information were recorded every six months thereafter.

The control group was required to list on the covering page of the questionnaire, any changes in their biographical data, their medical history or their risk factor profile (see Appendix B). Information regarding their possible involvement in a supervised exercise programme was also requested. An open-ended question provided respondents with an opportunity to state their reasons for leaving the CRC (see Appendix B). This question stipulated that the CRC would not be notified of the respondent's particular reason for leaving, but would be given a summary of all responses. To establish what would be needed to encourage CHD patients to remain in rehabilitation, participants were also asked to list any suggestions they might have as to how the programme could be improved.

Anxiety

Anxiety was measured by means of the Illinois Personality Assessment Test Anxiety Scale (IPAT AS) (Cattel & Scheier, 1976). The IPAT AS was designed to measure changes in levels of anxiety over time (Greer, Ramsay, & Bagley, 1973). The scale measures components of anxiety, namely, tension, guilt proneness, emotional instability, suspiciousness, and low social integration (Cattel & Scheier, 1976). The IPAT AS is recommended for experimental designs (Mitchell, 1985). The scale consists of 40 items each of which is accompanied by three responses (i.e., "Always, Often, Seldom" or "True, In Between, False") which follow a Likert format (see Appendix A).

The IPAT AS has demonstrated test-retest reliability of .86 over a two week time period and has yielded a Kuder-Richardson coefficient of .80 (Auld, 1985). Comparison of the IPAT AS with the Taylor Manifest Anxiety Scale (Taylor, 1953), Eyseneck's Neuroticism Scale (1968) and Spielberger's Trait Anxiety Scale (1982) yielded an average correlation of .70 which attests to the scale's content validity (Auld, 1985). Further evidence of the scale's validity is provided by Greer et al.'s (1973) study of 31 out-patients of a teaching hospital. In this study, the IPAT AS was significantly correlated with clinical ratings of degrees of anxiety. Clinical assessment occurred prior to administration of the IPAT AS, and therefore, ruled out the potential for bias on the part of the clinician.

A standardisation sample of 9942 English and Afrikaans scholars of both sexes has been used to establish the South African adaptations and norms of the IPAT AS (Cattell, Scheier, & Madge, 1968). Cattell et al. (1968) report reliability measures for

the four groups namely, English girls and boys and Afrikaans girls and boys. Unless otherwise stated, reliability figures for the four groups are reported here according to the above order. Assessment of the IPAT AS yielded test-retest (two week interval) coefficients of .88; .87; .86; .83 and Kuder-Richardson coefficients of .83; .78; .78; .82. Spearman-Brown split-half correlations of .80 for Afrikaans girls and boys and .76 for English girls and boys are reported. The construct validity of the IPAT AS was determined by correlating the scale with the conceptually similar NB Adjustment Questionnaire published by the National Bureau of Social and Educational Research (South African Department of Education, Arts, & Science, 1967). In all instances, correlations between the two scales' components were significant ($p < .05$).

Reliability and validity figures reported by Cattell et al. (1968) demonstrate both the IPAT AS's psychometric properties and its ability to measure anxiety among South African samples and thus, justifies the inclusion of the scale in the present study. Furthermore, the IPAT AS has been used successfully by Schomer (1985) in a study designed to determine the moderating effect of exertion therapy on levels of anxiety in a sample of South African students. Although Schomer (1985) does not report psychometric figures for the IPAT AS, his successful use of the scale suggests that it is appropriate for research concerning the effects of exercise on anxiety among South African samples. The sound psychometric properties of the IPAT AS, together with evidence of its applicability to South African studies concerning the effects of exercise on anxiety, justify the scale's use in the present study.

Depression

The Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), was used to measure depression. Development of the BDI was based on both clinical and research sources (Welch, Hall, & Walkey, 1990) for the purpose of measuring the complaints, attitudes and symptoms related to an individual's current degree of depression (Sweetland & Keyser, 1984). Specifically, the scale reflects somatic, motivational, affective and cognitive symptoms of depression (Welch et al., 1990), and thus, provides a measure of the severity of depression (Lees-Haley, 1989). The BDI has been used widely in research concerning depression (Welch et al., 1990).

The BDI is composed of 21 items, each corresponding to a specific complaint, symptom or attitude (see Appendix A). Six of the 21 items reflect somatic / biological disturbances (Meakin, 1992). The items comprise four alternate, self-evaluative statements which are ranked on a zero to three scale of severity with higher scores reflecting greater levels of depression (Endler & Parker, 1990; Kauth & Zettle, 1990). The inventory adopts a four point Likert response format.

The original assessment of the BDI's internal reliability yielded a Spearman-Brown coefficient of .93 (Beck, 1970). Subsequently, a number of studies have assessed the reliability of the BDI. For example, Gallagher, Nies and Thompson (1982) assessed the BDI's split half, test-retest (three week interval) and coefficient alpha reliability among a sample of clinical and non-clinical middle aged subjects (M age = 68 years). Reliability figures yielded by Gallagher et al. (1982) were .84 (split-half), .90 (test-retest) and .91 (Cronbach's coefficient alpha). Hatzenbuehler, Parpal, and Matthews

(1983) report slightly lower test-retest figures of .83 (same day) and .60 (under one week). Schaefer et al. (1985) assessed the internal reliability of the BDI among two groups, the first being a group of 101 psychiatric ward patients and the second, a group of 99 chemical dependency ward patients. The study yielded an alpha reliability coefficient of .94 for the psychiatric group and a coefficient of .88 for the chemical dependency group. Welch et al.'s (1990) use of the BDI in three communit (non-patient) samples, one sample of depressed patients and one sample comprising members of an eating disorder patient group, yielded equally high measures of internal reliability. Specifically, they report reliability coefficients of .90, .89 and .91 for the three non-patient groups, .88 for the group of depressed patients and .88 for the group of eating-disorder patients. Beck and Steer (1984) also report an interna reliability figure of .88 for the BDI.

Gatewood-Colwell, Kaczmarek and Ames (1989) assessed the internal reliability of the BDI in a sample of white and Hispanic men and women aged 60 to 80 years and yielded a Cronbach alpha of .80. In Endler and Parker's (1990) assessment of the relationship between coping styles and depressive symptoms among a sample of undergraduate students, the BDI demonstrated internal reliability coefficients of .8 for males and .88 for females. In a more recent study of students, Endler and cohorts reported internal reliability figures of .91 for males and .87 for females (Endler, Cox, Parker, & Bagby, 1992). In a similar sample of university students, Gotlib (1984) reported an internal reliability coefficient of .82. Thus, the BDI has demonstrated internal and test-retest reliability in both clinical and normal samples.

The validity of the BDI has also been established for both clinical and normal populations (Welch et al., 1990). Comparisons between the BDI and psychiatric

ratings have produced correlation coefficients of .77 and .67 (Beck, 1970; Bumberry, Oliver, & McClure, 1978). Similarly, Schaefer et al. (1985) compared the BDI with clinicians' global ratings of depression and with an overall score of depression derived from the Diagnostic and Statistical Manual for Mental Disorders (DSM-III; American Psychiatric Association, 1980). The comparison with clinicians' ratings yielded significant ($p < .05$) correlations of .65 for psychiatric patients and .67 for chemical dependency patients. The authors report equally significant correlations of .69 and .68 between psychiatric and chemical dependency patients' BDI scores and the DSM-III overall depression score. These studies provide support for the construct validity of the BDI. Further evidence of construct validity derives from Welch et al.'s (1990) factor analysis of the BDI. This analysis yielded a replicable general factor across two patient and three non-patient samples and thus suggests that the BDI taps a global construct of depressive symptoms.

The discriminant validity of the BDI has been assessed by Kauth and Zettle (1990) in a study of one normative group of adolescents and two psychiatric groups of adolescents. The first of the psychiatric groups was diagnosed as clinically non-depressed and the second as clinically depressed. Diagnosis for depression was based on the independent assessment by each patients' therapist. The results of the study indicated that the BDI was able to differentiate between the normative, non-depressed and depressed samples with a high degree of accuracy. Kauth and Zettle (1990) also report that the BDI was able to make this differentiation where the Dysfunctional Attitudes Scale (Weissman & Beck, 1978) and the Adolescent Activity Checklist (Carey, Kelley, Buss, & Scott, 1986) were not. Similarly, Atlas and DiScipio (1992) report that the BDI was able to differentiate depression among adolescents suffering Posttraumatic Stress Disorder, adolescents suffering Conduct

Disorder and a control group of non-depressed adolescents. This study also provided evidence of the BDI's concurrent validity by yielding a moderately high Pearson correlation of .58 ($p < .01$) between the scores of the BDI and the Reynolds Depression Scale for the total sample of 58 adolescents. Correlations between the scores of the two measures for the Posttraumatic and Conduct Disordered groups were .73 ($p < .05$) and .48 ($p < .05$) respectively. Further evidence of the BDI's concurrent validity was provided by a significant Pearson product-moment coefficient of .79 between the BDI and Brink et al.'s (1982) Geriatric Depression Scale in Gatewood-Colwell et al.'s (1989) study.

Johnson and Sarason (1978) have demonstrated the utility of the BDI in research concerning the relationship between stress and depression. More importantly, the test's ability to distinguish between depressed and non depressed CHD patients has been demonstrated in a number of studies (e.g., Campbell, Burgess, & Finch, 1984; Carney et al., 1987; Sloan & Bigger, 1991). While the BDI has been used among samples comparable to the present one, a review of the literature failed to reveal any studies which have used the BDI on South African, CHD patients. Nevertheless, the BDI is thought to possess suitable psychometric properties and sufficient applicability to the aims of the present study.

Data Analysis

Multivariate analysis of covariance (MANCOVA) was selected to determine whether, due to their non-attendance, the control group experienced greater levels of strain than the experimental group. MANCOVA determines how two or more levels of an independent variable (e.g., group) differentially effect a set of dependent variables

(Olson, 1976; Tabachnick & Fidell, 1983). This occurs with the simultaneous control of covariates, namely, those variables which are extraneous and unassociated with the primary variables under study (Kerlinger, 1986) and the pretreatment baseline measures of the dependent variables (Tabachnick & Fidell, 1983).

According to Tabachnick and Fidell (1983), MANCOVA has three primary uses. First, it removes the variance generated by the covariates from the error variance and consequently, serves a 'noise-reducing' function. By including any between group differences in the baseline measures as covariates, MANCOVA is able to demonstrate that any significant differences in the posttest scores (i.e., dependent variables) are a product of the independent variable (i.e., the experimental and control groups which comprise the independent variable) (Kerlinger, 1986; Tabachnick & Fidell, 1983). Thus, MANCOVA is able to determine the effect of the independent variable on the dependent variables (Kerlinger, 1986). Second, where random assignment to groups is not possible, MANCOVA statistically matches all subjects' covariate scores thereby reducing sampling bias. Third, MANCOVA tests whether any significant group differences in a dependent variable are, in fact, the result of the independent variable, rather than the result of the other dependent variables included in the analysis. This is achieved by examining the impact of the independent variables on the dependent variable of interest while simultaneously adjusting the effects of the other dependent variables.

Tabachnick and Fidell (1983) further state that MANCOVA achieves the above by means of the following procedure. The variance among scores is divided into the variance associated with within group differences in scores and the variance associated with between group differences. As each subject has a score on each

dependent variable, it is not possible to create a simple set of scores. Instead S matrices of difference scores are formed. The S matrices comprise the sums of squares and cross products which have been adjusted for the intercorrelations between covariates. In the final step of the procedure, the effects of the independent variables on the linear combination of dependent variables is established by examining the generalised variance of each S matrix and the ratio of variance between S matrices. In this step, Wilks' Lambda criterion determines the existence of significant multivariate effects. A Wilks' Lambda value of zero indicates perfect discrimination while a value of one indicates no discrimination.

MANCOVA is suited to the design and purposes of the present study. By controlling covariates, MANCOVA permits the investigation of the variables of primary concern and reduces the probability of spurious findings. Thus, by incorporating the Time 1 measures of psychological strain as covariates, MANCOVA allows the assessment of between group differences at Time 2. With MANCOVA it is also possible to include as covariates variables which effect the dependent variables but are extraneous to the research question. Research using samples similar to the present one (e.g., Stern & Cleary, 1981), indicates that the risk factor and biographical profiles of CHD patients who fail to comply with a rehabilitation programme is different to that of patients who elect to comply with such programmes. Consequently, it is possible that differences between the present experimental and control groups' risk factor profile, medical history and physical condition at Time 1 may effect the dependent variables. Chi square tests will be conducted to determine any between group differences in these variables. Where differences exist, the variables will be included as covariates.

To further increase the precision of MANCOVA, change score analyses will be used to reduce the moderating effect of the six month period. The Time 1 scores will be subtracted from the Time 2 scores and subsequently included in the MANCOVA as dependent variables. Change score analyses will increase the stringency of the MANCOVA by eliminating the possibility of pretest-posttest covariation (Barling & Milligan, 1987). Thus, any significant differences between the two groups' measures of strain at Time 2 (i.e., the dependent variables) resulting from the MANCOVA will be due to the effect of attending, versus not attending, the exercise programme (i.e., the independent variables).

Underlying MANCOVA are the assumptions of reliability, homogeneity, linearity and multicollinearity (Lewis-Beck, 1980; Tabachnick & Fidell, 1983). Thus, these assumptions will be tested prior to conducting the MANCOVA. Cronbach's coefficient alpha (Cronbach, 1951) will be used to assess the internal reliability of the two scales. The reliability coefficient reflects that proportion of the test variance which is attributable to 'true' differences among individuals rather than that which is attributable to error variance (Cronbach, 1970; Guyatt, Walter, & Norman, 1987). Following Nunnally's (1967) recommendation, scales will be considered reliable if they yield a minimum alpha coefficient of .60.

The homogeneity assumption of MANCOVA requires that the variances in the different groups of the design are identical (Myers, 1979). This implies that the variances associated with each group have been drawn from a population with equal variances (Runyon & Haber, 1980) and that each group has the same relationship between covariates and dependent variables (Tabachnick & Fidell, 1983). Given that the design comprises more than one dependent variable, a further requirement is

that the intercorrelations (covariance) between dependent variables are homogenous across the different groups (Tabachnick & Fidell, 1983). An absence of homogeneity indicates interaction between the independent variables and covariates and renders MANCOVA an inappropriate method of analysis (Tabachnick & Fidell, 1983). Homogeneity is calculated by dividing the variance of the first group by the variance of the second. Taking the degree of freedom into account, the result is then assessed for significance by examining the critical values of the F table. If the resulting F ratio is not significant then the data is homogenous. This method of determining homogeneity will be used in the present study.

Linearity refers to the tendency of two variables to aggregate along a straight line when plotted against each other (Kerlinger, 1963). MANCOVA assumes that the relationships between all dependent variables and covariates in each cell of the design are linear (Tabachnick & Fidell, 1983). In the present study, linearity will be measured by plotting the residuals of each dependent variable with each independent variable and covariate on a graph. Linearity will be accepted if variables follow a straight line (Tabachnick & Fidell, 1983).

Multicollinearity occurs when the correlations between the dependent variables are too high and the majority of the variance in the matrices can be attributed to covariance (Tabachnick & Fidell, 1983). The existence of multicollinearity indicates that at least one dependent variable is a near-linear combination of the other dependent variables (Tabachnick & Fidell, 1983). This effectively means that the offending dependent variable reflects the same information as that reflected by at least one other dependent variable. The existence of multicollinearity can be determined by examining the intercorrelations between dependent variables on a

variance covariance matrix. Intercorrelations in excess of .80 violate the assumption of multicollinearity (Lewis-Beck, 1980).

Results

A between groups MANCOVA was performed on the dependent variables which comprised the change scores derived from the anxiety and depression scores. The aim of the analysis was to determine any between group differences in the anxiety and depression levels manifested by exercising (i.e., the experimental group) and non-exercising (i.e., the control group) cardiac patients.

The Time 1 measures of anxiety and depression and history of tobacco abuse where included in the MANCOVA as covariates. Inclusion of tobacco abuse as a covariate was based on Chi square tests which were conducted to determine the existence of any differences between the experimental and control groups' biographical data, risk factor profile and medical history at Time 1 (see Table 6.1). The results of the tests revealed significant differences between the experimental and control groups' smoking habits. Smokers have been found to be significantly more reactive to stress, and consequently, more sensitive to strain (Towner & Contrada, 1988). Further, among CHD patients, persistent tobacco abuse is associated with subsequent cardiac events and CHD related surgical procedures (Coronary Drug Project Research Group, 1974). Given its probable relationship to the present study's measures of psychological and physiological strain, history of tobacco abuse was included as a covariate. In addition, t tests were run on the continuous variables age and the Time 1 measures of weight and maximal oxygen uptake. No significant differences were found for these three, potentially confounding variables (see Table

6.2). The independent variable, group membership, comprised two levels, namely, experimental (participants in an exercise programme) and control (drop-outs from an exercise programme). Table 6.3 presents the means, standard deviations (SD) and Pearson correlations of the pretest, posttest and change scores of the experimental and control groups.

Table 6.2
Tests Between the Experimental and Control Groups' Measures of Age, Weight, and VO_2max

Demographic Variables	Independent Variable: Group Membership	n	M	t
Age (years)	Experimental	20	50.9	1.44
	Control	20	55.1	
Weight (kg's)	Experimental	20	84.95	0.77
	Control	20	81.80	
VO_2max (ml/kg/min)	Experimental	20	25.67	0.55
	Control	20	26.54	

Note. No significant differences were found between the experimental and control groups' age, weight and VO_2max.

As can be seen from Table 6.3 the Time 1 measures of the dependent variables are significantly correlated and thus, individual ANCOVA's are inappropriate for the present data (O'Leary & Turkewitz, 1978). To determine the combined effect of the dependent variables it was necessary to subject the data to a MANCOVA (O'Leary & Turkewitz, 1978). Prior to running the MANCOVA, the reliability, linearity and multicollinearity of the data was assessed. From Table 6.3 it is evident that for both the experimental and control groups, the BDI and IPAT AS yielded internal reliability coefficients in excess of the .60 cut off point. Thus, the reliability assumption of MANCOVA was met.

Table 6.3

Means, Standard Deviations, Cronbach's Alpha and Pearson Correlations of the Pretest, Posttest and Change Score Measures of Anxiety and Depression for the Experimental and Control Groups

		Experimental			Control								
Variables		M	SD	Alpha	M	SD	Alpha	1	2	3	4	5	6
Pretest	(1) Anxiety	29.40	14.12	.89	26.60	12.42	.84		.70**	.84**	.18	-.40	-.60*
	(2) Depression	7.75	6.10	.89	9.05	6.40	.78	.65*		.59**	.44*	-.27	-.71**
Posttest	(3) Anxiety	29.70	13.16	.88	23.00	11.41	.85	.77**	.46*		-.45*	.17	-.27
	(4) Depression	7.55	4.49	.86	6.75	4.48	.80	.61**	.47*	.52*		.44	.31
Change	(5) Anxiety	0.30	7.73		-3.60	8.13		-.45*	-.35	.23	-.20		.43*
	(6) Depression	-0.20	5.75		-2.30	5.84		-.25	-.74**	-.10	.25	.23	

\+ Decimal points omitted from the correlation matrix.

** $p < .01$

* $p < .05$

The experimental group's data are presented above the diagonal;

The control group's data are presented below the diagonal.

As can be seen from the following calculation, the variances of the two groups' measures of anxiety and depression showed no statistically significant deviation from homogeneity.

Depression $\frac{40.89211}{37.14474} = 1.10$. df = 1 thus, $F = 2.15$

Anxiety $\frac{199.4105}{154.1474} = 1.29$. df = 1 thus, $F = 2.15$

1.10 and 1.29 are below 2.15 and therefore, are not significant.

Examination of the plots of residuals showed that none of the scatterplots deviated from linearity, and therefore, that the variables could be retained in their original form (Tabachnick and Fidell, 1983). From the variance covariance matrix it was evident that all intercorrelations between dependent variables were below .80, and therefore, that there was an absence of multicollinearity. Thus, the data met the assumptions of MANCOVA.

Based on Wilks' Lambda criterion, no significant multivariate effect was found on the combined change scores (Huck & McClean, 1975) of the two dependent variables F (.9319) = 1.242, $p > .05$. Thus, the MANCOVA failed to yield a significant difference between the experimental and control groups' levels of anxiety and depression. Consequently, the hypothesis stating that CHD patients who participated in a six month exercise programme would experience less anxiety and depression than CHD patients who withdrew from the same programme was not supported.

A further aim of the present study was to determine whether there was a significant difference between the number of cardiac and associated surgical events experienced by the experimental and control groups. To this end, steps were taken to ensure that there was an absence of pre-existing differences in the two groups' physiological health and that the control group were not participating in an alternate supervised exercise programme. The control group's responses to the open ended question regarding their reasons for leaving the programme indicated that there were no pre-existing differences in the experimental and control groups' physiological condition. Specifically, the control group indicated that their reasons for leaving the programme were logistical rather than due to ill health. No member of the control group reported participating in a supervised exercise programme in the intervening six month period.

During the six month period under assessment, one member of the experimental group reported a change in medical status, namely, an additional coronary artery bypass graft. One member of the control group had undergone a coronary artery bypass graft since his last assessment at the CRC and a second had had a coronary artery angiography. A chi square test of the experimental and control groups' change in medical status showed that this difference was not significant ($p > .05$). Therefore, no statistical support was found for the hypothesis that the experimental group would suffer significantly fewer cardiac and surgical events than the control group.

Discussion

The present study compared the anxiety and depression levels of CHD patients who had participated in a six month exercise programme with the anxiety and

depression of patients who had dropped out of the same programme. No significant differences were found between the two groups' levels of anxiety and depression. The number of cardiac and surgical events experienced by the two groups in the intervening six months were also assessed. Again, no significant difference between groups was found. Thus, the present study failed to show that aerobic exercise reduces CHD patients' level of psychological and physiological strain.

In seeking an explanation for the absence of significant relationships between exercise and psychological health it is useful to examine the characteristics of the CHD population. There is some differentiation between patients who enrol in a rehabilitation programme and those who do not (Kavanagh et al., 1977). Severely anxious and depressed people demonstrate a tendency towards emotion-related coping behaviour and away from task oriented coping strategies of which an exercise programme would be an example (Endler & Parker, 1990). These individuals have also shown a greater orientation towards avoidance coping strategies (Rosenberg, Peterson, & Hayes, 1987). Thus, the listlessness generated by pronounced psychological ill health would prevent severely anxious and depressed cardiac patients from joining an exercise based rehabilitation programme (Kavanagh et al., 1977). This would result in a natural selection of programme participants whereby the more severely anxious and depressed cardiac patients do not attempt to join a rehabilitation programme. Conversely, those who elect to join a rehabilitation programme demonstrate a desire to confront their problems and seek information regarding a solution (Terry, 1992). Both active confrontation of problems and information seeking facilitate psychological adaptation to CHD (Terry, 1992). Thus, regardless of whether they subsequently chose to remain in the programme or not,

the majority of patients who enrolled in the exercise programme may not be severely anxious and depressed.

Based on their IPAT anxiety scale and BDI scores, the present samples do not fit into the category of the clinically anxious or depressed. A score above 38 is needed to classify an individual as anxious (Cattell et al., 1968) and a score in excess of 9 is needed for a diagnosis of depression (Endler, Cox, Parker, & Bagby, 1992). Both the experimental and control groups' mean anxiety and depression scores fall short of these cut-off points. Studies incorporating clinically anxious or depressed samples have demonstrated that participation in an exercise programme is significantly associated with dramatic improvements in psychologically well-being, while studies using nonclinical samples have failed to find consistent support for this association (Desharnais et al., 1993). Current research indicates that significant improvements in anxiety and depression are confined to subjects who experience clinical psychological disturbances (De Geus et al., 1993). Given the low levels of anxiety and depression demonstrated by both groups, it is not surprising that exercise failed to exert a significant influence on the outcome measures of these variables.

The present results are inconsistent with past research which has identified anxiety and depression as two of the most pervasive behavioural disorders among both general (Endler & Parker, 1990) and CHD populations (Soloff, 1978a). However, the process from psychological ill health to recovery is relatively predictable with the result that 70% to 85% of CHD patients are likely to overcome their feelings of anxiety and depression in the first six months of recovery (Brown & Munford, 1984; Kavanagh et al., 1977). Spontaneous recovery has been attributed to the process of adaptation which occurs as patients and their families come to terms with the

numerous changes implicit to the disease (Kavanagh et al., 1977). It is thus plausible that anxiety and depression fade over time (Roth et al., 1990). Given that the present study examines psychological strain after a period of six months, it is possible that the majority of both the exercising and non-exercising groups of CHD patients had recovered from their earlier feelings of anxiety and depression. Indirect support for this assumption is provided by Giese and Schomer (1986) who examined the mood states of a similar sample of South African CHD patients. In this study, there was no significant change in the mood state profiles of both the experimental group of exercising patients and the control group of drop-outs over the ten month period of study. Giese and Schomer attribute the lack of significant change to the relatively long time period of three months between the date of the myocardial infarction and the date of admission to the programme. Specifically, they assert that the intervening three month period provided sufficient time for patients to achieve a stable mood state (Giese & Schomer, 1986).

From the results of past research (e.g., Brown & Munford, 1984; Kavanagh et al., 1977) it is evident that there exists a sub group of cardiac rehabilitation patients which remains anxious and depressed regardless of any form of intervention. A normal distribution of these two segments of the CHD patient population in both the experimental and control groups would not generate significant differences between the anxiety and depression levels of the two groups.

A further consideration when interpreting the present results is that much of the evidence of a relationship between aerobic exercise and enhanced psychological condition derives from cross-sectional research which has yielded significant, though weak, associations between exercise and psychological characteristics (De

Geus et al., 1993). It is possible that such associations are equally evident in the population from which the present sample derives but that the small sample size limited the ability to detect such weak associations.

The present study also failed to reveal any difference between the number of cardiac and surgical events reported by the experimental and control groups. There is considerable evidence to suggest that daily performance of low-intensity activities such as gardening, housework and walking for pleasure can lower the risk of CHD (Fletcher et al., 1992; Mittleman, 1993). Normal activities may generate a conditioning effect on physiological condition (Kavanagh et al., 1970). It has been established that CHD patients who perform regular, though unsupervised, exercise are able to increase their maximal oxygen uptake by the same amount as patients who participate in supervised programmes (Greenland & Briody, 1984). Thus, it is possible that the unsupervised, daily physical activities of the control group would have been comparable to the supervised exercise performed by the experimental group and, consequently, would account for the non significant difference between the number of cardiac events and associated surgical interventions experienced by the two groups. To extrapolate from this result that supervised exercise programmes are unnecessary, would be to overlook the primary issue of safety. Aerobic fitness can be achieved with unsupervised exercise (Greenland & Briody, 1984) However, the risk of suffering a cardiac event during exercise training is lower among CHD patients whose exercise performance is supervised and monitored than it is among patients who perform unsupervised exercise (Haskell, 1978).

A further explanation for the present result lies in the use of hard disease endpoints as the measure of physiological strain. Pisa et al. (1985) maintain that research

based on hard disease endpoints is prone to error particularly in samples which contain as wide a variety of prognoses as cardiac rehabilitation samples typically do. It can be assumed that cardiac rehabilitation samples comprise both low risk patients, whose prognosis is positive regardless of treatment, and high risk patients whose prognosis is inherently negative (Pisa et al., 1985). The non existent relationship between the treatment and outcomes demonstrated by the low risk group may mask any positive effects the treatment has on the high risk patients' measures of strain (Pisa et al., 1985). A further consideration is that CHD develops slowly and often, without outward signs of its progression (Orha et al., 1985). It is possible that the control group did, in fact, have greater degeneration of the cardiovascular system than the experimental group, but as it had not culminated in a cardiac event or related surgical intervention, the present study failed to detect this exacerbated condition. The hard disease endpoints used in the present study may have been insensitive to changes in the level of physiological strain of the two groups. Moreover, given the slow development of CHD, it is possible that extending the study over a longer period would have revealed a significant difference between the two groups' levels of physiological strain.

That the use of hard disease endpoints may have contributed to the lack of significant results is supported by past research. For example, both the NEHDP (Shaw, 1981) and the OEHCS (Cunningham, 1981) used hard disease endpoints to measure strain and both, failed to yield significant findings. Conversely, studies (e.g., Bethell & Mullee, 1990; Grodzinski et al., 1987) which have utilised a more sensitive measure of strain, such as maximal oxygen uptake, report significant relationships between participation in an exercise programme and physiological strain. To determine whether exercise does in fact enhance prognosis, future

research should be based on a more sensitive measure of physiological strain such as maximal oxygen uptake.

Limitations of the Present Study

The present study is limited by several methodological weaknesses. First, it is possible that the size of the sample may have limited the power to detect differences in the two groups' measures of strain (Thoreson & Powell, 1992). Power of a statistical test is intrinsically related to the number of subjects (Runyon & Haber, 1980). This relationship is inverse, meaning that the smaller the sample size, the larger will be the error (Kerlinger, 1986). Studies comprising both small samples and low power are inherently biased toward achieving nonsignificant results (Powell et al., 1993). Consequently, non significant results hold little weight in such studies (Powell et al., 1993). Christensen (1985) suggests that 15 subjects per cell is sufficient for analysis of variance designs comprising two or more levels of the independent variable. The sample sizes in the present study fall short of this minimum requirement and consequently, may have resulted in deviant samples (Kerlinger, 1986). Restriction of the sample size to a number of 20 may have produced an over-emphasis of relatively healthy CHD patients among the control group, which in turn, may have biased the results. Indeed, questionnaires were returned on a voluntary basis rendering it impossible to determine whether the control group is representative of the population of CHD patients who dropped out of the exercise programme or whether they represent a healthier subgroup.

Second, the physical heterogeneity of the sample may also have compromised the results (Terry, 1992). No effort was made to determine whether the subjects were affected by coexisting illnesses or to limit the sample to subjects with the same manifestation and severity of CHD. Similarly, no attempt was made to establish how much time each member of the control group spent at the CRC prior to dropping out of the programme. Any benefits derived from time spent exercising at the centre are likely to have confounded the present results. It is possible that more precise results would have been yielded by a homogenous sample comprising subjects who had experienced a single cardiac event (Terry, 1992) and by ensuring that all members of the control group withdrew from the CRC's programme immediately following admission.

Third, all the patients comprising the control group indicated that they did not withdraw from the exercise programme for reasons of ill health, but rather, for logistical problems. While this suggests that there was not a pre-existing higher incidence of illness among the control group, it must be remembered that the reasons given for leaving the programme are based on the patients' subjective perceptions and not on a clinical assessment. In order to determine whether non-compliance with the exercise programme is related to number of secondary events, future research should incorporate medical personnel's' assessments of patients' physiological condition at the time of withdrawal.

Fourth, the present study did not establish whether the control group habitually performed low-intensity physical activities such as gardening and housework. As these have been found to reduce the risk of CHD (Fletcher et al., 1992), the control group's non-supervised physical activity may have had a similar risk reducing effect

as the experimental group's documented exercise performance. Future research should attempt to measure the daily and leisure time physical activities of subjects not participating in supervised exercise programmes. In so doing, future research will be able to differentiate between the effects of aerobic fitness and of daily exercise behaviour on psychological condition (De Geus et al., 1993). This limitation highlights a fifth weakness of the present study, namely, the measurement of exercise performance exclusively in terms of attendance.

Attendance figures alone do not provide an accurate estimate of the training effect derived from exercise. Achievement of a training effect is contingent on the frequency and duration of exercise performance (Hickson et al., 1981; Rejeski et al., 1984; Seals et al., 1984). As the present study only considered the experimental group's attendance of the programme, there is no way of knowing whether they exercised for sufficient duration to exert the training effect necessary to reduce levels of psychological and physiological strain. As it stands, the present study demonstrates that attending an exercise programme does not effect psychological and physiological outcomes. Therefore, it is necessary for research to extend beyond attendance figures to examine the role of duration of exercise in predicting psychological and physiological strain. Study 3 of the present research addresses this weakness by examining the frequency and duration of exercise in relation to psychological and physiological manifestations of strain.

A further weakness concerns the use of hard disease endpoints as measures of CHD patients' strain. The progression of CHD is slow and insidious (Orha et al., 1985) with the result that research based on hard disease endpoints is unable to detect subtle, albeit physiologically relevant, changes in cardiovascular strain. Thus the

small number of subjects who had surgical interventions to correct the progression of CHD limited the generality of the present findings (Sloan & Bigger, 1991). To examine the relationship between exercise and physiological strain with greater precision, it is necessary to incorporate a measure which is sensitive to subtle changes in the condition of the cardiovascular system. Maximal oxygen uptake is believed to provide a sufficiently sensitive measure of the severity of CHD (Bruce, 1971). Again, Study 3 will address this weakness by examining the relationship between the various measures of exercise and maximal oxygen uptake.

Conclusion

In examining the relationship between aerobic exercise and psychological strain, the present study overcame many of the weaknesses of past research. However, this study was undermined by several additional weaknesses. To clarify the effect of exercise on the CHD patients' levels of strain, these need be addressed by future research. In order to determine the precise means by which exercise effects prognosis it is necessary to assess compliance along more specific parameters. Attempts to establish the impact of exercise on prognosis should include an examination of the duration of exercise together with figures of attendance. In the interests of greater precision, it is also necessary to look beyond the hard disease end points examined in the present study to a more chronic, but less overt, sign of degeneration of the cardiorespiratory system, namely, reduced maximal oxygen uptake. Therefore, a further aim of the present research is to examine, in a subsequent study, the impact of the two measures of compliance on measures of maximal oxygen uptake over a period of six months (see Chapter 8). The study

outlined in Chapter 8 will also examine the role played by behavioural factors in determining prognosis.

Patients' perception of their health has a direct effect on their eventual prognosis (Desharnais, Godin, Jobin, & Valois, 1990). Health perception is in turn influenced by patients' personal and behavioural disposition (Brown & Munford, 1984) to the extent that disposition has a greater impact on perception than the actual severity of the illness (Wise & Rosenthal, 1982). A predisposition to Type A behaviour has been shown to contribute to elevated levels of anxiety and depression (Francis, 1981). More specific to the present research is the literature concerning CHD patients which suggests that Type A behaviour may influence psychological and physiological prognosis. It follows that an attempt to predict prognosis within the context of cardiac rehabilitation should account for the influence of Type A behaviour. Before the potential impact of Type A behaviour on prognosis can be examined, it is necessary to establish the precise nature of the construct and develop a measure which reflects that construct. Therefore, Chapter 7 is concerned with the identification of the dimensions of Type A behaviour manifested by cardiac rehabilitation patients and the development and validation of a multidimensional measure of Type A behaviour.

CHAPTER 7

STUDY 2: DEFINING THE TYPE A BEHAVIOUR OF CHD PATIENTS

The Type A - CHD association has been subject to considerable scientific inquiry over the past 30 years (Edwards & Baglioni, 1991; Edwards et al., 1990a; Taylor & Cooper, 1988). A process of inquiry typically yields conflicting research which challenges earlier findings (Haynes & Matthews, 1988) and consequently, leads to closer scrutiny of the underlying construct. Such has been the case with research concerning Type A behaviour. While the field of Type A research is characterised by equivocal results, it is still generally believed that Type A behaviour is implicated in the development of CHD (Matthews, 1982; Siegman et al., 1987; Thoreson & Powell, 1992). This persistent belief is based on the premise that Type A behaviour is a multidimensional construct comprised of various dimensions which can be assessed independently and which are likely to exert differential psychological, physiological and behavioural effects (Siegman et al., 1987). Hence, it is assumed that the conflicting results characterising Type A - CHD research are the result of the inaccurate conceptualisation and measurement of the Type A construct (Edwards et al., 1990a; Matthews, 1982; Palmer et al., 1992). Ambiguous definitions of Type A behaviour have generated a number of measuring instruments with questionable construct validity (Meininger et al., 1991; Palmer et al., 1992).

While Type A behaviour was originally conceptualised as a global construct (Edwards et al., 1990a) it is now believed that only a subset of Type A components predict primary or secondary CHD (Dembroski et al., 1989; Grossarth-Maticek & Eysenck, 1990; Lee, 1992) and that the global construct has limited predictive power (Evans, 1990). This limitation is attributed to the possibility that analyses based on a global orientation have concealed the more discrete yet crucial relationships between Type A components and outcome variables (Forgays, 1992; Lee, 1992; Steinkamp, 1990). A global orientation has also caused a loss of vital information and ambiguous explanations in studies regarding the impact of Type A behaviour (Carver, 1989).

The global construct defies the concept of individualism. Type A behaviour is a composite of personality dimensions rather than a global construct (Ganster et al., 1991). Individuals possess different combinations and degrees of these dimensions, each of which exerts differential effects (Jennings, 1984). Identification of the individual differences in Type A behaviour may reveal a Type A composite that is more predictive of CHD (Friedman, 1989). Given that a global orientation does not allow for the separate measurement of the dimensions, research concerning Type A behaviour and CHD should not be based on a global construct. Indeed, persistent use of the global construct is now considered scientifically invalid (Thoreson & Powell, 1992). Consequently, more recent Type A research has extended beyond the global conceptualisation to an examination of the impact of individual components on health (Taylor & Cooper, 1988).

An increasing number of studies have examined Type A behaviour as a bi-dimensional construct, comprising achievement striving and impatience irritability (*cf.* Barling & Charbonneau, 1992; Bluen et al., 1990; Helmreich et al., 1988; Lee, 1992; Öhman et al., 1989; Spence et al., 1987, 1989). These studies have consistently shown that achievement striving predicts performance, but not health related outcomes, and conversely, that impatience irritability predicts indices of health, but not performance. Several of these studies (e.g., Helmreich et al., 1988; Lee, 1992) concede that the body of achievement striving / impatience irritability research is limited by its failure to incorporate the equally important Type A dimensions of hostility and anger expression (i.e., expressed and suppressed anger). These dimensions hold particular relevance to research concerning CHD as they are believed to be the strongest predictors of heart disease (Greenglass & Julkunen, 1991; Matthews & Haynes, 1986). Therefore, measures of hostility, anger suppression (anger-in) and anger expression (anger-out) should be included in models of Type A behaviour used in research concerning the relationship between the construct and CHD (Abbot & Peters, 1988; Friedman & Booth-Kewley, 1988; Siegman et al., 1987). Despite the fact that none of the cited studies has reconciled all five of the above mentioned dimensions in a model of Type A behaviour, they have succeeded in demonstrating that different dimensions do exert differential effects on health and performance. Further, this research has confirmed the multidimensional nature of the Type A construct (Edwards et al., 1990a; Tett et al., 1992).

Based on past Type A research achievement striving, impatience irritability, hostility anger-in and anger-out can be considered central characteristics of Type A behaviour which, with the exception of achievement striving, can result in

sustained, negative physiological responses which enhance the likelihood of ill health (Taylor & Cooper, 1989) (see Chapter 4). It remains to be determined whether the five dimension model of Type A behaviour applies to people suffering CHD.

The bulk of component research has been conceptualised in terms of models of Type A components which are applicable to low-risk samples (e.g., Bluen et al., 1990, 1991; Burns, 1992; Weidner et al., 1989; Williams et al., 1980). The distribution of serious illness in a normal population is typically skewed with the result that the possibility of finding significant relationships is severely limited (Schmitt & Colligan, 1984; Suls & Marco, 1990). Further, research based on such populations may yield low correlations between predictor variables (e.g., Type A components) and relatively rare outcome variables (e.g., coronary events) (Schmitt & Colligan, 1984). There is also evidence to suggest that relationships between Type A components and CHD do not exist in all populations (Leon et al., 1988). Indeed, past research indicates that Type A behaviour discriminates between healthy individuals and CHD patients (Orth-Gomér & Undén, 1990) and may impose a differential impact on the two groups (Abbott & Peters, 1988).

A number of studies have revealed a significant association between Type A behaviour and primary CHD (e.g., Cramer, 1991; Haynes et al., 1980; Kannel & Gordon, 1974; Rosenman et al., 1975). Conversely, studies of high-risk samples (e.g., Barefoot et al., 1989; Case et al., 1985; Dimsdale et al., 1981; Powell et al., 1993; Ragland and Brand, 1988b; Shekelle et al., 1985; Sloan & Bigger, 1991) have found that Type A behaviour is unassociated with secondary CHD. From the results of the low-risk and high-risk studies, it is possible to conclude that Type A behaviour influences initial, but not secondary CHD (Matthews, 1988).

Evans (1990) proposes two possible explanations for the different influence of Type A behaviour on healthy and CHD patients' physical condition. Evans' first explanation is based on the premise that Type A behaviour is implicated in the atherosclerotic development and acute precipitating factors which jointly determine the occurrence of cardiac events (Matthews, 1988). He asserts that the extent of danger associated with acute precipitating factors are contingent on the severity of atherosclerotic development. If Type A behaviour exerts a greater influence on the occurrence of acute precipitating factors than it does on the development of atherosclerosis, then the extent of atherosclerosis found in Type A survivors of an initial event will be less than or equal to that which is found in Type B survivors (Kamarck & Jennings, 1991). Consequently, Type A and B survivors of an initial event will carry a similar risk of secondary CHD (Evans, 1990). In terms of Evans' argument, Type A's with severe atherosclerosis are more likely to suffer a fatal obstruction or blood clot while Type A's who survive an initial manifestation of CHD have less severe atherosclerosis and are less prone to developing atherosclerosis of significant severity.

Most high-risk studies have used inadequate measures of disease severity at study inception (Matthews, 1988). Therefore, the possibility that Type A behaviour exerts a greater impact on acute events than it does on atherosclerosis has yet to be assessed empirically. The possibility that Type A and Type B survivors' atherosclerosis is of similar severity has also not been assessed directly. However, Matthews' (1988) meta-analysis which reveals that Type A and B survivors have a similar risk of reinfarction or CHD related mortality provides indirect support for Evans' first explanation.

Evans' (1990) second explanation holds that the psychological impact of CHD may differentiate between Type A's and B's. Type A's are more prone to respond to CHD by re-evaluating their lifestyles and making the appropriate modifications to their behaviour than Type B's (Palmer et al., 1992). The ability to modify behaviour may be enhanced by the resilience associated with the relative youth of Type A CHD patients (*cf.* Eaker & Castelli, 1988; Williams et al., 1988). Type A behaviour accelerates the occurrence of cardiac events with the result that Type A CHD patients are typically younger than Type B patients (Miller et al., 1991). Consequently, Type A's may possess more of the youthful resilience necessary for adaptation and ultimate survival (Miller et al., 1991). Type A behaviour may be further modified by pharmacological treatment. For example, the beta-blocking drugs taken by many CHD patients have been found to reduce the intensity of Type A behaviour (Abbott & Peters, 1988; Powell, 1987). A further consideration is that physiological factors, such as an impaired cardiovascular system, are important predictors of prognosis and may override the contribution of Type A components (Palmer et al., 1992).

Given the possibility that CHD diagnosis may alter behaviour, models of Type A components which have been developed for low-risk samples may not reflect the Type A behaviour of cardiac patients (Palmer et al., 1992). Therefore, statements regarding the relationship between Type A components and CHD should be confined to specific populations (Leon et al., 1988). It is necessary therefore, to develop a multidimensional model comprising the Type A components, achievement striving, impatience irritability, hostility, anger-in and anger-out, argued for in Chapter 4, which is applicable to the behaviour manifested by CHD patients. Development of

such a model will require an appropriate measure of Type A behaviour (Hinkin & Schriesheim, 1989). Therefore, an aim of the present study is to develop a measure of Type A components which is appropriate to research concerning cardiac rehabilitation patients.

Measurement Error of Existing Self-report Measures of Type A Behaviour

A primary constraint of Type A research concerns the predominant emphasis on global measures of Type A behaviour (Edwards & Baglioni, 1991; Taylor & Cooper, 1988). Not only are these measures insensitive, but they are now considered conceptually invalid (Thoreson & Powell, 1992). Indeed, progress towards a refined, multidimensional theory of Type A behaviour is hindered by extant self-report measures of the construct, namely, the JAS (Jenkins, Zyzanski, & Rosenman, 1971), Framingham scale (Haynes, Levine, Scotch, Feinleib, & Kannel, 1978) and Bortner scale (Bortner, 1969). According to Edwards and Baglioni (1991) these scales combine multiple Type A dimensions into unitary indices. Therefore, they are subject to both the conceptual problems outlined in the preceding paragraphs and to psychometric weaknesses. That is, by their very nature, they violate both Type A and classical measurement theory (Edwards & Baglioni, 1991; Edwards et al., 1990a).

On a psychometric level, the above measures have failed to yield consistently satisfactory reliability and validity coefficients. The Bortner and Framingham scales have demonstrated moderate measurement error while the measurement error yielded by the JAS has been substantial (Bortner, 1969; Edwards et al., 1990a;

Haynes et al., 1978; Mayes, Sime, & Ganster, 1984). Without sufficient evidence of satisfactory internal reliability, the three measures can be said to lack precision.

The questionable internal reliability can be attributed to the fact that the scales comprise multiple dimensions but are operationalized as unidimensional measures (Edwards et al., 1990a). This translates to an empirical paradox whereby the fundamental principle of congeneric (unidimensional) measurement is simultaneously assumed and violated. Treating the scales as unidimensional, generates two additional problems. First, it confounds interpretation of results by disguising the relative contribution of the various dimensions to the overall score (Edwards & Baglioni, 1991; Edwards et al., 1990a; Gerbing & Anderson, 1988). Second, it does not allow for the identification of specific relationships between dimensions (Edwards & Baglioni, 1991; Edwards et al., 1990a). Without disentangling these dimensions and measuring them separately, examination of the Type A construct has limited psychometric and conceptual integrity.

There is also evidence to suggest that the validity of existing measures is questionable (Meininger et al., 1991). For example, while the JAS (Jenkins et al., 1971) has been found to be an effective predictor of some forms of CHD, there is still insufficient understanding of both the meaning of scores and of the underlying Type A construct measured by the instrument (Hansson, Hogan, Johnson, & Schroeder, 1983). Further, the JAS, like the Structured Interview, has been criticised for failing to adequately reflect three of the principle Type A components, namely, achievement striving, impatience and hostility (Leak & Flotte, 1987). Matthews (1982) shows that there is a low correlation between existing Type A assessment methods and self-reported Type A behaviour. Thus, though the JAS has demonstrated predictive

validity, albeit modestly, its construct validity is questionable (Hogan & Nicholson, 1988; Leak & Flotte, 1987).

A related issue is the comprehensiveness of extant measures. The three self report measures discussed previously each reflect different underlying constructs, while failing to consider dimensions key to the prediction of CHD, namely, anger and hostility (Booth-Kewley & Friedman, 1987; Edwards et al., 1990a; Evans, 1990; Julius, Harburg, Cottington, & Johnson, 1986; Lee, 1992; Matthews, 1982; Meininger et al., 1991). Any attempt to correlate the Type A construct with manifestations of disease must include a more specific and comprehensive analysis of the multidimensional nature of that construct (Russek et al., 1990). Only by including independent measures of the various Type A dimensions will it be possible to test directly the relevance of those dimensions implicated in the development of CHD (Matthews, 1982). Therefore, there is a strong need for a measure of Type A behaviour which includes key dimensions such as hostility and anger (Forgays, 1992; Lee, 1992).

Burns (1992) addressed this need by developing the Multidimensional Type A Behaviour Scale (MTABS). The MTABS has demonstrated satisfactory reliability and validity (Burns, 1992). The scale also includes independent measures of achievement striving, impatience irritability, hostility and anger. These components are defining characteristics of Type A behaviour (Matthews, 1982; Price, 1982; Wright, 1988) and have been identified in the present research as key components of CHD patients' behaviour. While the MTABS brings greater precision to the measurement of Type A behaviour, it is not suited to the present sample for the following reasons: First, the MTABS has been developed and tested on low-risk

samples. As discussed previously, CHD may alter the influence of Type A behaviour on health outcomes (Evans, 1990) with the result that Type A measures developed for low-risk samples may not apply to high-risk samples. Second, the achievement striving subscale of the MTABS contains items reflecting job involvement. As job involvement is not associated with CHD (Friedman & Booth-Kewley, 1988; Meininger et al., 1991; Wright, 1992), and the present sample comprises both employed and unemployed individuals, the achievement striving subscale of the MTABS is not suited to samples of CHD patients. Third, the MTABS does not include a measure of anger-in. The well documented relationship between anger-in and indices of CHD (Dembroski et al., 1985; Gilbert & McArthur, 1988; MacDougall et al., 1985; Tennant & Langeluddecke, 1985) and the research identifying anger-in as a powerful predictor of secondary CHD (e.g., Wright, 1988), suggests that any assessment of CHD patients' Type A behaviour should include a measure of suppressed anger. Thus, it is evident that existing measures of Type A behaviour are unsuited to the aims and sample of the present research.

Progress within the theoretical domain requires adequate Type A measurement technology (Hinkin & Schriesheim, 1989). To this end, Edwards et al. (1990a) recommend that new attempts be made to combine, modify and supplement existing Type A measures to form unidirectional measures of Type A components, which collectively show a high convergence with the multidimensional nature of Type A behaviour. Edwards and Baglioni (1991) maintain that this is best achieved by starting out with precise definitions of the dimensions under review, selecting items which reflect these, combining items into unidimensional scales and establishing the internal and external consistency of these scales. Ideally, this should be founded on an empirically validated factor structure (Forgays, 1992; Lee, 1992).

The present study aims to develop and validate a multidimensional scale measuring the Type A components achievement striving, impatience irritability, hostility, anger-in and anger-out. This scale will be suitable for research concerning the prevalence of Type A components among cardiac rehabilitation populations and the extent to which these components predict prognosis for recovery from CHD. Development of the scale is based on the assumption that achievement striving, impatience irritability, hostility, anger-in and anger-out are key components of Type A behaviour, each of which will exert differential effects. Further assumptions are that the five components are independent, but may have additive effects and that they can be quantified and assessed reliably by means of self-report measures (Bluen & Odendaal, 1988).

Toward the Development of a Valid Measure of Type A Behaviour

Validity concerns how well a test measures what it is supposed to measure (Anastasi, 1968). The validity coefficient represents the extent to which predictor and criterion scores are correlated (Lee & Foley, 1986). There are three primary categories of validity, namely, content, criterion and construct validity (Anastasi, 1968). Content validity refers to the sampling adequacy of a measuring instrument and indicates whether the instrument items are representative of all relevant aspects of the attribute under study (Kerlinger, 1986). Criterion validity reflects a measuring instrument's ability to predict behaviour under specified conditions (Anastasi, 1968). This form of validity is established by comparing the measuring instrument against an established and independent measure of the attribute under examination

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(Kerlinger, 1986). Construct validity is a more abstract, though durable, concept and reflects the precision with which a test measures a given theoretical construct (Anastasi, 1968). Further, it is a comprehensive procedure which accounts for both content and criterion validity (Anastasi, 1968; Hogan & Nicholson, 1988; Landy, 1986) and thus, renders the independent assessment of the latter two unnecessary.

Construct validity constitutes a primary link between theoretical concepts and psychometric principles (Kerlinger, 1986). It provides answers to the two questions critical to the advance and measurement of theory, namely, a.) Does the construct exist within the population under study?, and b.) Does the measuring instrument in question reliably measure individual differences in the construct? (Hogan & Nicholson, 1988). The aim of the present study is to develop a multidimensional measure of CHD patients' Type A behaviour comprising the dimensions, achievement striving, impatience irritability, hostility, anger-in and anger-out which were defined and discussed in Chapter 4. Central to this exercise will be the determination of the construct validity of the said Type A Component Questionnaire.

Method

Sample and Setting

Participants in the present study were 217 male and female CHD patients who had at one time, enrolled in the Johannesburg City Health Department Cardiac Rehabilitation Centre's (CRC) programme. The 217 participants were randomly selected from the CRC's archives. A summary of the demographic characteristics and medical history of the sample is presented in Table 7.1.

Table 7.1

Summary of the Demographic Characteristics of the Sample (N=217)

Variable	Number	Percentage
Sex		
Male	195	90 %
Female	22	10 %
Race		
White	186	88 %
Black	31	12 %
Marital Status		
Married	190	86 %
Unmarried	27	14 %
Employment Status		
Professional	99	46 %
Skilled	62	29 %
Self-employed	19	9 %
Retired	37	17 %
Education		
Ten Years (Std 8)	43	20 %
Twelve Years (Matric)	64	29 %
Technical Qualification	35	16 %
Professional Qualification	29	13 %
Undergraduate/Postgraduate Degrees	46	21 %
Medical History*		
Myocardial Infarction	147	68 %
Coronary Artery Bypass Graft	99	46 %
Percutaneous Transluminal Coronary Angioplasty	53	24 %
Coronary Artery Angiography	175	81 %
Congenital/Valvular Heart Surgery	3	1 %

* The majority of the patients included in the sample had experienced more than one of the cardiac events or associated surgical interventions listed above.

Design

A cross-sectional field design (Christensen, 1985) was used to explore the hypothesis that Type A behaviour is a multidimensional construct comprising achievement striving, impatience irritability, hostility, anger-in and anger-out.

Measuring Instruments

Personal Details

On being admitted to the CRC, participants provided their biographical details and information concerning their medical and risk factor history. Biographical data included age, marital status, sex, race and education. Medical history referred to the type and frequency of cardiac and surgical events, while risk factor history included the amount and duration of nicotine abuse, the presence of hypertension, hyperlipoproteinaemia and diabetes mellitus, and incidence of CHD in the participant's immediate family.

Achievement Striving and Impatience Irritability

The present study utilised items from the Jenkins Activity Survey (Form N) (Jenkins & Zyzanski, 1975) to construct scales comprising items considered to have face validity to the achievement striving and impatience irritability components (Leon et al., 1988). The 13 item Form N derives from the original 52 item Jenkins Activity Survey (JAS) developed by Jenkins et al. (1971) and has been validated against CHD criteria. Factor analysis of the 1971 version of the JAS revealed three independent factors namely, speed and impatience, hard-driving competitiveness, and job involvement (Zyzanski & Jenkins, 1970). The Form N scale was developed by eliminating the job involvement dimension, thereby rendering the scale applicable to employed and unemployed samples alike (Burke & Deszca, 1984). As job involvement is unassociated with CHD (Friedman & Booth-Kewley, 1988; Meininger

et al., 1991; Wright, 1992), the Form N is a more appropriate measure for samples of cardiac rehabilitation patients.

The Form N is an effective measure of Type A behaviour (Rosenman, 1978). The Form N has also demonstrated an ability to predict differentially the relationship between Type A behaviour and CHD and Type A behaviour and other diseases (Rimé, et al., 1989). Further, the Form N has proved successful in measuring the relationship between Type A behaviour and illness among a retired geriatric population (mean age: 81 years) (Kopac, 1990). The effectiveness of the Form N scale among older, unemployed samples of CHD patients is relevant to the present study which consists of both employed and unemployed CHD patients whose average age is 57 years and four months.

The 13 item, multiple choice scale is a self-report instrument designed to measure hard-driving competitive behaviour, speed and impatience (Jenkins & Zyzanski, 1975). These dimensions of Type A behaviour are also referred to as achievement striving and impatience irritability (Öhman et al., 1989). Eight of the 13 items measure achievement striving and reflect active, hard working and serious minded behaviour (e.g., How would your spouse or closest friend rate you currently? Definitely hard-driving and competitive - Definitely relaxed and easy going). The remaining five items measure impatience irritability and reflect intolerant and irate behaviour (e.g., How often do you actually "put words in the persons mouth" in order to speed things up?). Each item is accompanied by a two, three, or four point Likert response format (see Appendix A).

A review of the literature failed to yield any research which has assessed the psychometric properties of the achievement striving and impatience irritability subscales of the JAS Form N. However, two independent studies, incorporating samples of executives, have demonstrated internal reliability coefficients of .72 and .69 for the JAS Form N (Lipschitz, 1987; Moschides, 1988). Test-retest reliabilities of .90 (one week interval) and .61 (one year interval) reported by Holden and Hickman (1987) and Myrtek and Greenlee (1984) respectively, provide support for the scale's temporal stability. Comparisons between the Form N scale and the Thurstone Temperament Schedule's Activity Scale (TTSAS), and the Anger dimension of the Trait Anger Scale (TAS) have yielded significant correlations ($r = .35$, $p < .001$ and $r = .30$, $p < .001$, respectively) (Smith, 1985).

While the reliability and validity of the achievement striving and impatience irritability subscales has not been assessed in previous research, the psychometric properties reported for the Form N as a whole suggest that the subscales are reliable and valid measures. An aim of the present study is to assess the psychometric properties of the achievement striving and impatience irritability subscales. The items comprising the subscales have face validity to the achievement striving and impatience irritability dimensions. Indeed, the Form N contains many of the same items used in other factor analytic studies which have examined dimensions of achievement striving and impatience irritability (e.g., Edwards et al., 1990a; Gallant & Chrisjohn, 1985; Lee, 1992; Öhman et al., 1989). The successful application of the Form N scale to the South African context (e.g., Lipschitz, 1987; Moschides, 1988; Smith, 1985), its established ability to confirm the relationship between Type A behaviour and CHD (e.g., Carmelli, et al., 1988; Matthews, 1982; Rosenman &

Chesney, 1980), the use of its items in other component based research (e.g., Gallant & Chrisjohn, 1985), and its applicability to the present sample, justify the subscales inclusion in the present study.

Hostility

In the present study, hostility is measured by a subscale of the Hostility and Direction of Hostility Questionnaire (HDHQ) (Foulds, Caine, & Creasy, 1960). The HDHQ was derived from the Minnesota Multiphasic Personality Inventory (MMPI) by Foulds et al. (1960). The scale is based on the rationale that hostility is either directed toward the ego or self (intropunitiveness) or toward objects or other people (extrapunitiveness) (Foulds, 1965). The HDHQ yields measures of Total Hostility and Direction of Hostility. Subscales of self-criticism and guilt measure intropunitiveness, while acting-out hostility, criticism of others and projected or delusional hostility subscales measure extrapunitiveness (Spielberger et al., 1983). According to the authors, the facet of hostility tapped by each of the sub-scales is sufficiently different to justify their independent existence (Foulds et al., 1960).

The present study does not incorporate the scale in its entirety, but instead, utilises the acting-out hostility subscale. This subscale was selected on the basis that it provides the closest approximation of the hostility said to characterise Type A behaviour (*cf.* Friedman & Rosenman, 1974). The acting-out hostility measured by the HDHQ reflects a tendency towards expressed aggression which is externally directed at the perceived source of the hostile person's feelings of stress and frustration (Foulds et al., 1960; Hafner, 1974).

The acting-out hostility subscale comprises 12 of the 51 HDHQ statements and their concomitant true or false responses (see Appendix A). Few studies have used the subscales of the HDHQ as independent measures. Those that have (e.g., Philip, 1968; Verma, 1974) fail to provide details of the acting-out hostility subscale's psychometric properties. Therefore, with the exception of the subscale's reliability estimates reported in the test manual, only those psychometric properties which apply to the HDHQ as a composite measure could be found in the literature and are reported here.

The HDHQ has been standardised by Foulds et al. (1960) for normal and psychiatric populations alike (Hafner, 1974). In a sample of 15 men and 15 women drawn from a local voluntary society, Caine, Foulds and Hope (1967) report one year test-retest reliability coefficients of .51 for the Direction of Hostility measure and .73 for the measure of Total Hostility. The acting-out subscale yielded a one year test-retest reliability coefficient of .70 (Caine et al., 1967). The internal reliability of the HDHQ was assessed in Malan's (1989) study of a non psychiatric population and yielded an alpha coefficient of .72 for Total Hostility. Comparisons of the HDHQ scores yielded by medical, surgical, psychiatric and suicide patients show that the HDHQ is able to differentiate between the levels of hostility manifested by specific groups, thus providing evidence of the scale's construct validity (Vinoda, 1966).

Evidence of the HDHQ's discriminant validity is provided by Biaggio and Godwin (1987) who incorporated the scale in a study which assessed the relationship between depression, anger, and hostility. In this study, the HDHQ exhibited an

ability to discriminate between subjects high and low in depression by yielding a Wilks' lambda of .86 ($F = 12.41$, $p < .01$) (Biaggio & Godwin, 1987). Inclusion of the acting-out subscale of the HDHQ in the present study is based on the overall scale's psychometric properties and ability to predict physiological and psychological outcomes, the effective use of the scale on South African samples (e.g., Malan, 1989) and the subscale's articulation of the hostility said to be manifested by Type A individuals.

Anger

Feelings of anger can be expressed, inhibited or controlled and people vary considerably in the way they elect to process their anger (Greenglass & Julkunen, 1991). Both anger-in (Wright, 1988) and anger-out are correlates of Type A behaviour (Engebretson & Matthews, 1992; Friedman & Rosenman, 1974; Spielberger et al., 1988; Wright, 1988). Hence, the importance of a scale which differentiates between the modes of anger expression. The present study incorporates Spielberger et al.'s (1985) 20 item Anger Expression (AX) scale. The AX measures the degree of total anger expression, regardless of direction (Spielberger et al., 1985), and the components of anger expression, namely anger-in, anger-out and the control of anger (Schonwetter & Janisse, 1991). As an aim of the present study is to assess the mode of anger expression and not how anger is managed, only the anger-in and anger-out components were used. The anger-in dimension reflects the suppression of experienced anger which is synonymous with lack of assertiveness (Tennant, Langeluddecke, Fulcher, & Wilby, 1987), while the anger-out dimension concerns the extent to which the individual responds to angry feelings with

aggressive and hostile behaviour (Delamater & McNamara, 1987). The anger-in and anger-out subscales each comprise eight items accompanied by a four point Likert response format which allows respondents to indicate the frequency with which they feel certain emotions, or act in a particular manner (see Appendix A).

In the initial administration of the AX, Johnson (1984) assessed the scale's overall internal reliability among 1114 high school students and yielded alpha coefficients of .80 for males and .77 for females. Alpha coefficients for the anger-in and anger-out subscales were .84 (males) and .81 (females) and .73 (males) and .75 (females), respectively (Johnson, 1984). In the same sample, anger-in correlated significantly ($p < .01$) and negatively with the composite anger expression measure (-.83, males; -.70, females), while anger-out demonstrated a positive and significant ($p < .01$) correlation (.58, males; .64, females) (Spielberger et al., 1985). The AX has also been used by Greenglass and Julkunen (1991) in a study of hostility, anger and Type A behaviour among Finnish undergraduate students. While specific reliability figures are not provided, the authors indicate that the alpha's were in excess of .70 (Greenglass & Julkunen, 1991).

The AX's validity has been assessed by correlating the scale and its subscales with other measures of anger and personality. In this assessment, correlations were largely significant and in the predicted direction (Spielberger, et al., 1985). Delamater and McNamara (1987) included the AX scale in a study designed to assess the relationship between anger and assertive and aggressive behaviours. Analysis of the data yielded a significant ($p < .05$) correlation of .29 between anger-in and unassertiveness and a null association between the AX subscales (Delamater

& McNamara, 1987). These results provide further evidence of the AX scale's validity (Delamater & McNamara, 1987).

Support for the anger-in and anger-out subscale's construct validity is provided by Schill and Wang (1990) who correlated the AX with the MMPI-2 Anger Content Scale in a sample of male and female undergraduate students. Given that the MMPI-2 Anger Content Scale constitutes a measure of anger expression, it was predicted that the scale would correlate positively with the anger-out, but not the anger-in, subscale of the AX. Results of the study supported this prediction by yielding significant ($p < .05$), positive correlations of .69 for men and .43 for women between the AX anger-out subscale and the MMPI-2 Anger Content Scale. The correlations of .28 for men and .32 between the MMPI-2 Anger Content Scale and the AX anger-in subscale were not significant at the .05 probability level. Thus, the AX has been shown to possess construct validity.

Spielberger et al. (1985) assessed the anger-in and anger-out subscale's ability to identify the role of anger in the development of CHD and hypertension. Using the same sample as Johnson (1984), Spielberger and colleagues (1985) analysed the AX measures in relation to systolic blood pressure (SBP) and diastolic blood pressure (DBP). Male and female subjects' anger-in correlated significantly ($p < .001$) and positively with SBP and DBP (correlations ranged from .16 to .47). With the exception of female measures of DBP, correlations between subjects' measures of anger-out and blood pressure were significant ($p < .05$), but negative, and ranged between -.09 and -.13. These results are consistent with the literature (*cf.* Dembroski et al., 1985) which suggests that anger-in bears a stronger relationship

to elevated blood pressure than anger-out.

The AX has also been used by Janisse et al. (1986) to examine the relationship between anger and cardiac reactivity among Type A and B individuals. Engebretson et al. (1989) and Vitaliano et al. (1993) have successfully utilised the anger-in and anger-out dimensions of the AX to test the impact of anger expression on psychophysiological responses. Use of the AX by Kahn (1986), Malan (1989) and Burns (1992) demonstrates the subscales successful application to the South African context. The subscales have also been used in studies incorporating aims and samples similar to those of the present study (e.g., Janisse et al., 1986; Sloan & Bigger, 1991; Spielberger et al., 1985). The subscales' applicability to the aims and sample of the present study, together with their satisfactory psychometric qualities justify their inclusion in the present study.

Data Analysis

The present study attempts to establish that CHD patients' Type A behaviour is a multidimensional construct comprising achievement striving, impatience irritability, hostility, anger-in and anger-out. This will be achieved by administering and analysing a questionnaire composed of scales measuring each dimension of the Type A behaviour pattern. A primary objective of the analysis will be to ascertain the appropriateness of the Type A Component Questionnaire's five factor structure and validate each dimension by means of confirmatory factor analysis.

In as much as it identifies underlying psychological traits (Anastasi, 1968), factor

analysis constitutes a powerful means of establishing construct validity (Kerlinger, 1986; Martin & Lee, 1992). Factor analyses comprise a group of statistical procedures which facilitate the elimination of redundant variables, the grouping of correlated variables, and identification of the conceptual meaning common to the grouped variables (Briggs & Cheek, 1986). Within the realm of scale development, factor analysis serves to identify the fundamental properties of a measuring instrument, establish its construct validity and assess the extent to which it is factorially pure or complex (Kerlinger, 1986). That is, the procedure identifies the inherent factors and their respective loadings and reveals the factorial composition of the scales (Anastasi, 1968). The loadings reflect the correlation between scale items and factors which in turn, represents the scale's validity (Anastasi, 1968).

Factor analysis can be exploratory or confirmatory (Welch et al., 1990). Exploratory factor analysis is based on an initial pool of items and generates a synopsis of the pattern of intercorrelations between items and an indication of the source variables responsible for the observed variance (Welch et al., 1990). Thus, it provides an indication of the number of underlying factors. There are a number of limitations associated with exploratory factor analysis, not the least of which is that the results can only be regarded as tentative (Edwards et al., 1990a). Nevertheless, it is a useful tool in areas of research where insufficient theory is available to classify items on conceptual grounds (Edwards et al., 1990a). Where there is sufficient theory to justify the logical classification of items into conceptual groups, confirmatory factor analysis is indicated (Gerbing & Anderson, 1988). Certainly, the plethora of Type A theory allows for the formation of such hypothetical groups (Edwards et al., 1990a).

A fundamental purpose of confirmatory factor analysis is to test conceptual hypothesis (Briggs & Cheek, 1986), such as the five factor structure of the Type A construct. It has the capacity to confirm that the selected items tap the *a priori* operationally defined constructs (Welch et al., 1990). With confirmatory factor analysis, the number of factors are specified and standard extraction criteria are imposed at the outset. The data are then tested against the hypothesised factor structure for 'goodness of fit' (Briggs & Cheek, 1986). Based on the extraction criteria it is possible to discard those items which do not contribute to the fundamental properties of the questionnaire, and therefore, do not aid in the description of the underlying construct. As the procedures involved in test validation constitute hypothesis testing (Hogan & Nicholson, 1988; Landy; 1986), the results of a confirmatory factor analysis provide support for the construct validity of a measuring instrument. Thus, the resulting reduced and validated questionnaire can be taken as a direct measure of the observed data (Harman, 1967), namely, Type A behaviour.

Principle Component Analysis

Principle components with eigenvalues greater than one and subsequent varimax rotation (Kaiser, 1970) was selected as the central method of analyses. Possibly the oldest and most widely known methods of factor analysis, principle components analyses (PCA) was first described by Pearson (1901) and then later by Hotelling (1933) who suggested specific means by which PCA could be adapted to factor analyses (Harman, 1967; Jolliffe, 1986). It was only with the increased availability of computers, however, that PCA became as widely used as it is today (Harman,

1967; Jolliffe, 1986). Kaiser's Method of PCA or Kaiser's 'Little Jiffy' as it has come to be known, is one of the most pervasive methods of factor analysis (Cronbach, 1970; Kaiser, 1970). It is also a method of analysis used widely in research concerning both Type A behaviour and the psychological and behavioural aspects of CHD (e.g., Räikkönen & Keltikangas-Järvinen, 1992; Suarez & Williams, 1990; Tett et al., 1992; Wright, Carbonari & Voyles, 1992).

The primary aim of PCA is to reduce the dimensionality of a large set of interrelated variables to a core set of uncorrelated principle components which reflect the essential information contained in the original set of variables, and thus, increase interpretability (Jolliffe, 1986). This method of reduction both retains as much of the variation of the original data set as possible and orders the components in such a way that the bulk of the variation present in the entire original set of variables is accounted for by the first few components (Jolliffe, 1986). Thus, PCA achieves a parsimonious description of a given set of observed data (Harman, 1967).

With confirmatory PCA, the number of components is prescribed in accordance with a specified hypothesis. By applying established criteria to the analysis it is possible to provide empirical support for the factorial (or component) hypothesis. That is, fulfilment of established criteria provides empirical confirmation of the appropriateness of the hypothesised factor structure for the given data. The criteria applied to the present data include Kaiser's (1970) Measure of Sampling Adequacy (MSA), Kaiser's (1960) Little Jiffy criterion (i.e., eigenvalue greater than unity), the communality estimate (i.e., rejection of communalities below .20) (Cureton & D'Agostino, 1983), component saturation (i.e. acceptance of items loading in excess

of .40) (Kim & Mueller, 1978), and Cattell's (1966) scree test.

Kaiser's Measure of Sampling Adequacy

Kaiser's MSA reflects a particular variable's consistency with the set of variables from which it derives (Kaiser, 1970). As such, it indicates whether the data are suitable for factor analysis. Kaiser's MSA provides an index ranging from 0 to 1 where an MSA of 1 shows that every variable in the set will predict every other variable without error (Kim & Mueller, 1978). MSA results in excess of .80 reflect a high level of consistency (Kim & Mueller, 1978). Items with MSA's below .50 suggest that the data are unacceptable for analytic purposes (Kaiser & Rice, 1974). Therefore, in the present study, items which yield an MSA below .50 will be eliminated from further analysis.

Kaiser's Eigenvalue Greater than Unity Criterion

The factor extraction criterion employed by the majority of empirical studies is the Kaiser-Guttman eigenvalues greater than unity rule (Hakstian, Rogers, & Cattell, 1982; Zwick & Velicer, 1982). The Kaiser criterion (Kaiser, 1960) holds that the principle components which have eigenvalues greater than one reflect the same information as that which is present in the original, albeit larger, set of variables (Jolliffe, 1986). Those factors with variance below one possess less information than the original variables and, consequently, can be discarded (Jolliffe, 1986). In imposing the Kaiser criterion on the data, a lower bound for the number of statistically acceptable factors is set, and thus, the extracted factors with

eigenvalues greater than one will be greater than or equal to the number of factors responsible for the correlation matrix (Kim & Mueller, 1982). The present study will reject principle components which have eigenvalues less than one.

Communality Estimate

Central to factor analysis is the determination of the communality of a variable and its relative uniqueness (Gorsuch, 1983). The communality estimate refers to that proportion of the variance which an item has in common with all other items (Kerlinger, 1986; Kim & Mueller, 1978). An estimate of zero indicates that there is no correlation between the relevant item and all other items, while an estimate of one is indicative of a perfect correlation between the item and all other items. Thus, high communalities reflect a low level of uniqueness (Cortina, 1993). Following common practice, items which yield communality estimates below .20 will be rejected in the present study (Cureton & D'Agistino, 1983).

Component Saturation

A fundamental aim of all methods of factor analysis is to reduce a number of observed variables to a smaller set which still preserves most of the information represented by the original set (Guadagnoli & Velicer, 1988). According to Jolliffe (1986), it is invariably true that if the initial number of variables is large, then a subset of those variables will retain essentially all of the information present in the original set of variables. This is reflected by a minimal overlap of 9% in the variance between the variable and the factor, which is in turn, reflected by a factor loading of

.30 (Tabachnik & Fidell, 1983). Reduction of the original set is achieved by eliminating those variables which do not share sufficient variance with the factor to provide a salient interpretation of the underlying dimension (Tabachnik & Fidell, 1983). In PCA, items which yield loadings below .30 or .40 on any given factor are not considered salient and therefore, can be eliminated from further analysis (Guadagnoli & Velicer, 1988; Hinkin & Schriesheim, 1989; Kim & Mueller, 1978). While selection of an appropriate cut off point is a question of personal choice, a higher factor loading reflects a greater overlap between the variable and the factor (Tabachnik & Fidell, 1983). It follows that the greater the overlap, the more the variable can be regarded as a pure measure of the factor (Tabachnik & Fidell, 1983). That is, the more the item contributes significantly to the meaning of the factor. To limit the number of trivial loadings but maximise the number of variables sharing a proportion of their variance with a component (Zwick & Velicer, 1982), the upper .40 level will be used in the present analyses.

Cattell's Scree Test

Cattell's (1966) scree test is a widely used method of determining the number of factors (Hakstian et al., 1982), and will be employed as a further means of identifying the correct number of factors. The scree test is considered a reliable means of discerning the major common factors from the factors of minor importance (Kim & Mueller, 1986). The scree test provides a graphical representation of the appropriate number of factors which should be retained (Zwick & Velicer, 1982). Eigenvalues, representing the amount of variability accounted for by each factor, are plotted on a graph against the number of variables generated by the inter-item

correlation matrix (Welch et al., 1990). This process typically yields a plot resembling an 'elbow'. The number of appropriate factors is taken at the point where there is a break between the eigenvalues of the downward slope and the eigenvalues of the more horizontal slope (Kim & Mueller, 1978; Welch et al., 1990). This occurs at the point where the scree approximates a straight, though not necessarily horizontal, line (Jolliffe, 1986). Typically, the number of factors is taken as the factor to the immediate left of the scree as the onset of the scree represents trivial factors (Cattell & Vogelman, 1977). Thus, the present study will take the factors to the left of the scree as the correct number of factors.

Confirmation of the Five Factor Solution

To ensure that the five factor solution fulfils Harris' (1967) definition of a robust factor as being one which has a consistent pattern of two or more item loadings per factor across different methods of factor analysis, the data will also be analysed using alpha analysis, image analysis, and the maximum likelihood method. Alpha analysis considers the variables included in the analysis as samples of a universe of variables and constitutes a method of initial factoring (Kim & Mueller, 1978). Image analysis is also a method of obtaining initial factors. However, it decomposes the observed variation into partial images and anti-images, rather than into common and unique parts (Kim & Mueller, 1978). The maximum likelihood method determines the difference between the correlation matrix of the observed variables and the hypothetical matrix of the universe from which the variables derive (Harman, 1967).

Use of these methods also allows for the retesting of the five factor solution against the extraction criteria used in the Principle Components Analysis (i.e., Kaiser's (1960) rule, Kaiser's (1970) MSA, component saturation, the communality estimate, and Cattell's (1965) scree test) and the testing of the solution against the additional factor extraction criteria associated with the maximum likelihood method. These include, Bartlett's chi square, Akaike's (1974) information criterion, Schwarz's Bayesian criterion, and Tucker and Lewis' (1973) reliability coefficient.

Bartlett's chi square represents a test of the goodness of fit between the factor model and the observed data. Estimates of Bartlett's chi square which fall below a significance level of .05 suggest that the null hypothesis should be rejected and that the number of factors is appropriate for the data. Akaike's information criterion (1974) distinguishes the optimal and most parsimonious factor model from a class of competing models (Bozdogan, 1987). Thus, it highlights the model which presents both the highest information gain and the lowest complexity (Bozdogan, 1987). The number of factors which yields the smallest value of Akaike's information criterion is taken as the most appropriate factor solution. The same principle is applied to Schwarz's Bayesian criterion. However, this criterion is less inclined to yield trivial factors than Akaike's or Bartlett's criteria. Tucker and Lewis' (1973) reliability coefficient provides an index of the fit between the factor models and the observed data (Kim & Mueller, 1978). An acceptable fit is represented by a value of .9 or more (Edwards et al., 1990a).

It is believed that confirmatory factor analysis in the present study will provide empirical support for the five dimension hypothesis, identify the nature of these

dimensions and validate the Type A Component Questionnaire. Also necessary is an assessment of the reliability of the Type A Component Questionnaire.

Reliability

The concept of reliability refers to the consistent accuracy of a measuring instrument (Anastasi, 1968; Kerlinger, 1986). More specifically, it is defined as the ratio between the variance ascribed to the true differences between individual's scores on a single observation and the total variance (Guyatt et al., 1987). Thus, the reliability of an instrument reflects its stability from administration to administration and indicates that the measurement error apparent when differences occur between administrations are the result of random influences (Nunnally, 1978). Selection of the appropriate estimate of reliability is contingent on which source of error variance the research aims to identify. Two key estimates of reliability are internal consistency and test-retest stability (Kerlinger, 1986). Internal consistency identifies the measurement errors related to the use of different items, while test-retest estimates reveal error factors associated with lapses in time (Cortina, 1993).

Internal Reliability

Internal consistency reflects the homogeneity of test items (Anastasi, 1968; Kerlinger, 1986). Possibly the most frequently used measure of internal reliability is Cronbach's coefficient alpha (1951) (Cortina, 1993). This is particularly true of research concerning the psychometric properties of measures of Type A behaviour, namely, the JAS, Bortner Scale and the Framingham scale (Edwards et al., 1990a).

Alpha is equivalent to the mean of all split-half reliabilities and is a more generalised form of the Kuder Richardson coefficient of equivalence (Cronbach, 1951). It also constitutes the lower bound of a test's reliability (Novick & Lewis, 1967). That is, it provides the most conservative estimate of reliability. This can be attributed to the further assumption of Cronbach's coefficient alpha that test items are tau-equivalent (Cortina, 1993; Novick & Lewis, 1967).

Tau-equivalence is achieved when the true scores of test items correlate perfectly with each other and when the variance between items is homogeneous (Gilmer & Feldt, 1983). As perfect tau-equivalence is rare, the reliability estimate typically approaches the lower bound (Cortina, 1993). Scales composed of items containing varying response lengths may result in heterogeneous error variance between items. In turn, heterogeneity in error variance will generate differences in the variance of observed scores and consequently, violate the assumption of tau-equivalence (Gilmer & Feldt, 1983). When the assumption of tau-equivalence is violated, Cronbach's alpha deflates reliability (Feldt & Brennan, 1989).

Cronbach's alpha further assumes that items represent one underlying construct (i.e., that measures are congeneric), as in fact do most other measures of reliability (Nunnally, 1978). Violation of this assumption means that alpha reliability figures cannot be taken as a measure of true score variance (Edwards et al., 1990a). This assumption is less restrictive in practice as it accommodates measures which are composed of items that are unequal in length (Gilmer & Feldt, 1983).

The subscales derived from the factor analysis in the present study include items of

varying response lengths and consequently, violate the assumption of tau-equivalence (see Appendix C). This violation renders the use of Cronbach's coefficient alpha an inappropriate means of estimating the internal consistency f the five subscales of the Type A Component Questionnaire. That is, use of Cronbach's alpha will generate an estimate of internal reliability considerably lower than the true value. It is thus necessary to utilise a reliability formula based on the more accommodating assumption of congeneric parts.

Kristof (1974) developed a formula of reliability which is applicable to scales comprised of congeneric parts. The formula's accommodation of scales of unequal response lengths (Gilmer & Feldt, 1983) renders it the appropriate means of estimating internal reliability for the data in the present study.

The Kristof formula for internal reliability is as follows:

$$r_K = \frac{(\hat{\sigma}_{12}\hat{\sigma}_{13} + \hat{\sigma}_{12}\hat{\sigma}_{23} + \hat{\sigma}_{13}\hat{\sigma}_{23})^2}{\hat{\sigma}_{12}\hat{\sigma}_{13}\,\hat{\sigma}_{23}\hat{\sigma}^2_x}$$

(Gilmer & Feldt, 1983, p. 105).

Where r_K = Kristof's reliability estimate;

$\hat{\sigma}$ = the covariance coefficient;

12; 13; 23 = combinations of two of the three parts (i.e., part one and two; part one and three; part two and three) and;

$\hat{\sigma}2\,x$ = the total variance of the scale.

Kristof's formula only accommodates scales comprised of precisely three parts (i.e., items). This is not however, a limiting factor as scales comprising a larger number of parts can be reduced to three precise parts and then subjected to Kristof's formula (Gilmer & Feldt, 1983). To reduce the number it is necessary to first determine from the original scale, all possible combinations of three and second, to perform Kristof's formula on the various combinations. The resulting coefficients are then averaged (Gilmer & Feldt, 1983). The average coefficient is then taken as the estimate of internal reliability. The number of combinations is based on the number of observed parts (i.e., items) and can be determined using a formula developed by Kass (1975). The formula for determining the number of combinations of three for a given number of items is:

$$\sum_{i=0}^{r-1} (-1)^i \frac{(r-i)^c}{i!(r-i)!}$$

where r = number of required groups (i.e., three)

c = number of items.

(Kass, 1975, p. 108)

Unlike Cronbach's alpha, Kristof's formula does not constitute the lowest measure of reliability (i.e., the lower bound estimate of reliability). Instead, it provides an accurate and consistent estimate under circumstances in which the congeneric assumption has been met (Gilmer & Feldt, 1983). A small variance between the coefficients derived from the various combinations of three demonstrates that the formula is estimating the same underlying construct, and hence, that the assumption of congeneric measures is being met.

Determination of the minimum level of acceptable reliability is relatively arbitrary (Feldt & Brennan, 1989). While numerous authors reject coefficients which fall below .70 (Feldt & Brennan, 1989), Nunnally (1967) recommends that the cut off point be set at .60. Further, .60 is the cut off point employed in current Type A component research (e.g., Burns & Bluen, 1992). Therefore, the present study utilises the cut-off point of .60 for reliability estimates.

Test-retest Reliability

It is widely accepted that, for a method of reliability assessment to be of value, it must reflect the influence of all sources of measurement error (Feldt & Brennan, 1989). A primary source of measurement error derives from the day to-day variation in behaviour (Feldt & Brennan, 1989). The existence of this source of error measurement can be determined by readministrating the test after a prescribed lapse in time. Typically referred to as test-retest reliability, this approach provides estimates of the error variance of random fluctuations in performance between two different administrations of a test (Powell, 1987). Test-retest reliability constitutes the only method available which is able to reflect day-to-day error factors (Feldt & Brennan, 1989).

By definition, test-retest reliability reflects the extent to which test results are generalizable over time (Anastasi, 1968; Powell, 1987) and is referred to as the coefficient of stability (Cronbach, 1960). The higher the retest coefficients, the lower the chance that scores have been influenced by random changes concerning the subject or test environment (Anastasi, 1968). While reliability coefficients will decrease over time (Cronbach, 1960), it is not recommended that the interval

between tests exceed six months (Anastasi, 1968). Therefore, the stability of the Type A Component Questionnaire will be assessed by establishing its test-retest reliability over a period of six months. In the present study, a correlational matrix generated by a separate sample of cardiac rehabilitation patients' Time 1 and Time 2 scores will be used to determine the six month test-retest reliability of the five scales.

Results

Validity of the Type A Component Questionnaire

Principle components with eigenvalues greater than one and subsequent varimax rotation (Kaiser, 1970) was used to confirm the factor structure of the five dimension measure of Type A behaviour. Kaiser's (1970) MSA was the initial criterion applied to the data and served to determine the appropriateness of the common factor model. Four items failed to meet the .05 acceptability level and were eliminated from further analyses (i.e., items 65, 72, 74, & 79 in Appendix A).

The second factor extraction criterion applied to the data was component saturation. Twelve items were eliminated for failing to load on any of the five factors at the .40 level (i.e., items 62, 68, 70, 71, 75, 77, 80, 81, 83, 92, 93, & 98 of Appendix A). The final factor solution comprised 26 items (see Table 7.2). From Table 7.2 it can be seen that one item loaded on two factors. The literature indicates that the item, reflecting temper, is congruent with the conceptual meaning of both factors (cf. Carver et al., 1976; Gallant & Chrisjohn, 1985; Glass, 1977a; Williams et al., 1988). Thus, the multiple loading did not compromise the interpretation of the factors (Bluen & Donald, 1991).

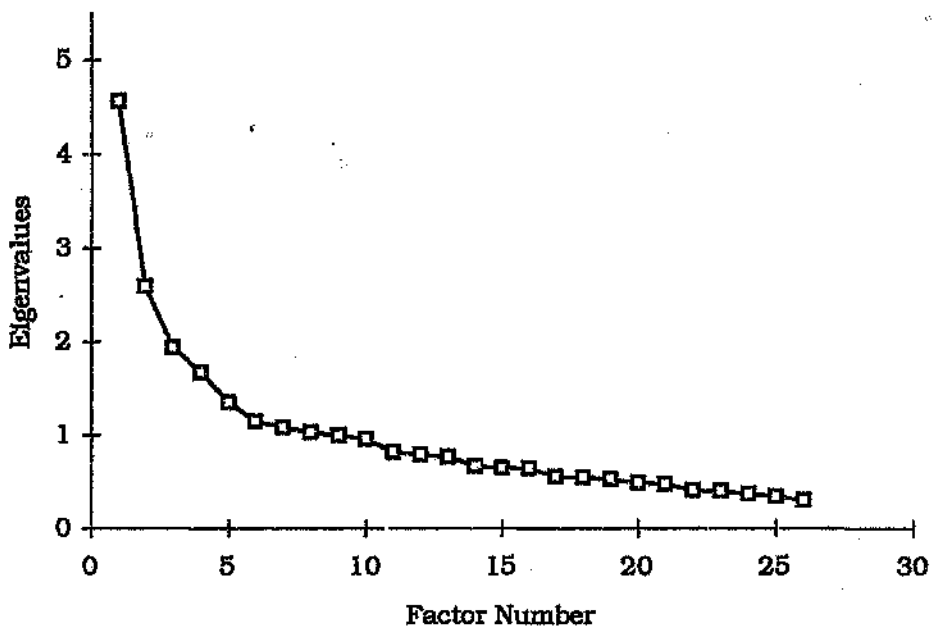
Table 7.2
Varimax Rotated Factor Loadings on Five Factors

	Scale Items	Factor Loadings 1	2	3	4	5	h^2	MSA
1	I pout or sulk.	.63	.11	-.18	.10	-.17	.48	.78
2	I withdraw from people.	.55	.01	.08	-.06	-.13	.33	.70
3	I boil inside, but I don't show it.	.59	-.31	-.04	-.02	.14	.47	.65
4	I tend to harbor grudges that I don't tell anyone about.	.57	.20	-.03	-.11	-.07	.39	.80
5	I am angrier than I am willing to admit.	.59	.06	-.13	-.29	.15	.47	.76
6	I am irritated a great deal more than people are aware of.	.67	.16	-.10	-.16	.03	.51	.82
7	I keep things in.	.39	-.63	-.01	-.05	-.0	.55	.77
8	I express my anger.	.06	.73	-.06	-.16	-.06	.57	.82
9	I argue with others.	.19	.56	-.02	-.26	.07	.41	.77
10	I strike out at whatever infuriates me.	.27	.63	-.11	.0	-.26	.55	.84
11	I say nasty things.	.33	.47	-.19	-.2	6	.45	.86
12	I lose my temper.	.30	.66	-.15	.01	.11	.56	.83
13	If someone annoys me, I am apt to tell him or her how I feel.	-.34	.58	-.05	-.16	-.13	.49	.76
14	How was your temper when you were younger?	-.07	-.11	.44	-.06	.51	.47	.67
15	I can easily make other people afraid of me, and sometimes do for the fun of it.	-.01	.04	.55	.19	.08	.34	.67
16	Sometimes I enjoy hurting persons I love.	-.02	.02	.67	.01	-.04	.46	.70
17	At times I have a strong urge to do something harmful or shocking.	.03	-.07	.52	-.12	-.17	.32	.60
18	I get angry easily and then get over it soon.	.01	-.14	.44	.20	.19	.28	.67
19	At times I feel like smashing things.	-.27	-.13	.60	-.17		.51	.76
20	At times I feel like picking a fist fight with someone.	-.18	-.17	.49	.1		.37	.75
21	When you listen to someone talking, and this person takes too long to come to the point, how often do you feel like hurrying the person along?	-.07	-.12	-.07	.79	.12	.66	.85
22	How often do you actually "put words in the person's mouth" in order to speed things up?	-.16	-.14	.01	.71	.21	.59	.78
23	I easily become impatient with people.	-.16	-.10	.33	.54	.09	.45	
24	When you were younger, did most people consider you to be (Definitely hard-driving and competitive? ... Definitely relaxed and easy going)?	.18	-.04	-.02	.20	.74	.62	.60
25	How would your spouse (or closest friend rate you currently (Definitely hard-driving and competitive? ... Definitely relaxed and easy going)?	-.02	-.16	.09	.25	.59	.45	.70
26	I approach life in general (Much more seriously ... Much less seriously).	-.22	-.00	-.18	.03	.53	.37	.59
	Eigenvalues	4.6	2.6	1.9	1.67	1.3		
	Percentage of total variance accounted for	18%	10%	8%	6%	5%		
	Over-all MSA							.75

The scale represented by the final factor solution demonstrated a satisfactory overall MSA of .75, with no individual item yielding a MSA below the .50 level of acceptability. Thus the overall and individual MSA's support the appropriateness of the common factor model (Kaiser, 1974) and indicate that the data are adequate for confirmatory factor analysis (Kim & Mueller, 1978). All items yielded communality

estimates above the .20 acceptability level. Given that the factor to the left of the scree reflects the correct number of factors (Cattell & Vogelman, 1977), the scree plot of the data supported the five factor solution (see Figure 7.1). The factors yielded by the analysis were consistent with the hypothesised five dimensions of Type A behaviour. Thus, PCA provided statistical and conceptual confirmation of the five factor structure.

Figure 7.1
Scree Plot of Eigenvalues derived from the Principle Components Analysis



On the basis of Kaiser's eigenvalue greater than unity criterion, eight factors were extracted using alpha factor analysis and six factors, using image components analysis. Of the eight factors extracted by the alpha factor analysis, two factors constituted 'doublets' and one did not yield a single loading above point .40. As Harris' (1967) criterion calls for the exclusion of 'singlet' and 'doublet' factors and the point of component saturation was set at .40 in the present study, this method

confirmed the five factor solution. Image component analysis extracted five robust factors and a sixth on which no item loaded above .40. In terms of Harris' criterion, the sixth factor could not be considered robust and thus, the five factor solution was again confirmed. The five factor solutions yielded by both alpha factor analysis and image components analysis replicated the factors yielded by the principle components analysis.

Maximum likelihood analyses was conducted with four, five, six, seven and eight factors to determine the appropriate number of factors as indicated by the extraction criterion associated with this method. The range of four to eight was indicated by the eigenvalues greater than one criterion of the PCA. Maximum likelihood with five factors yielded a significant Bartlett's chi square result. A significant result confirms the adequacy of the five factor hypothesis (Harman, 1967). Extraction based on Akaike's information criterion indicated a six factor solution. However, the scree test generated by the six factor specification supported a five factor solution, as in fact, did the scree tests for all other specifications. Schwarz's Bayesian criterion indicated a four factor solution, but the test for Bartlett's chi square for this specification showed four factors to be insufficient. Tucker and Lewis' reliability coefficient suggested that eight factors reflected the optimum fit between the correlations derived from the factor solution and the observed correlations. Of these eight factors, three violated Harris' criterion of 'singlet' and 'doublet' factors, while the fourth factor did not yield a single item with a loading above .40. Thus, Tucker and Lewis' reliability coefficient suggested a four factor solution. It should be noted, however, that the difference between the reliability coefficients generated by the five and eight factor specifications was marginal (i.e., .91 vs. .98). Further, a value of .91 is indicative of an acceptable fit between the observed data and the five factor model

(Edwards et al., 1990a).

The various methods of factor analysis, and their associated extraction criteria, confirmed that the five factor solution derived from the initial principle components analyses with varimax rotation (Kaiser, 1970) was compatible with the data. The five factors can thus be said to adequately account for the variation present in the original set of variables (Jolliffe, 1986).

As can be seen from Table 7.2, the five factors were consistent with the hypothesised five components of Type A behaviour. The five factor solution accounted for 46% of the total variance. The first factor explained 18% of the common variance and consisted of six of the eight items comprising Spielberger et al.'s (1985) anger-in subscale. Hence, the label of Anger-in for the first factor.

The second factor explained ten percent of the common variance and contained six of the eight expressed anger items of Spielberger et al.'s (1985) anger-out subscale and one item from the anger-in subscale by the same authors (i.e., "I keep things in"). This item yielded a *negative* loading. Given the negative value, this item was consistent with the concept of expressed anger and consequently, the second factor was labelled anger-out.

The third factor accounted for eight percent of the variance and was named acting-out hostility. Six of the items loading on this factor derived from the acting-out hostility subscale of the HDHQ while the seventh derived from the JAS (i.e., "How was your temper when you were younger?").

The fourth factor contained three items reflecting impatience irritability. Two of these derived from the JAS Form N (e.g., "When you listen to someone talking, and this person takes too long to come to the point, how often do you feel like hurrying that person along?"), and one from the acting-out hostility subscale of the HDHQ, namely, "I easily become impatient with people". The fourth factor, which accounted for six percent of the common variance, was named impatience irritability.

The fifth factor accounted for five percent of the variance. This factor was composed of four items derived from the JAS Form N. Three of the items reflected achievement striving behaviour. The fourth item (i.e., "How was your temper when you were younger?") measured temper. Given that the fifth factor was dominated by items reflecting achievement striving and had only a secondary emphasis on temper (Edwards et al., 1990a), it was given the name achievement striving.

Reliability of the Type A Component Questionnaire

The five subscales were subjected to Kristof's (1974) formula for reliability estimation. The variance between the different combinations' coefficients was minimal. Therefore, all subscales met the assumption of congeneric measurement. Further evidence of the congeneric status of the subscales' was provided by the low correlations between the five subscales (see Table 7.4). The correlations, which ranged between -.36 and .47, supported the independence of the five subscales. All five subscales yielded satisfactory internal reliability estimates (range: .65-.76; see Table 7.3).

Test - Retest reliability of the Type A Component Questionnaire was established over a six month interval for a separate sample of 31 cardiac rehabilitation patients (27 males, 4 females; M age = 55.5 years, SD = 12.37 years; 25 married, 6 unmarried; 28 white, 3 non-white; 16 professionals, 8 skilled workers, 4 self-employed, 3 retired; education level achieved - 2, ten years of schooling; 10, 12 years of schooling; 4, technical qualifications; 6, professional qualifications; 9 university degrees). The 31 patients constituted the total number of respondents to complete and return the questionnaire for Study 1 of the present research. All five of the test-retest correlations were significant at the .01 level (range: .61-.84; see Table 7.3). Therefore, the five subscales can be said to possess adequate temporal stability.

Table 7.3
Reliability Estimates for the Five Subscales of the Type A Component Questionnaire

	Reliability Estimate	
Subscale	Kristof's Formula	Test-Retest
Anger - In	.72	.74**
Anger - Out	.76	.69**
Acting Out Hostility	.67	.81**
Impatience Irritability	.71	.61**
Achievement Striving	.65	.84**

**p < .01

Table 7.4
Pearson Correlations Between the Five Components of Type A Behaviour (N = 31)

		Mean	S D	1	2	3	4	5
1	Anger - In	11.10	2.97					
2	Anger - Out	14.08	2.85	.35**				
3	Acting Out Hostility	13.22	1.73	-.23**	-.33**			
4	Impatience Irritability	4.86	1.42	-.28**	-.36**	.20**		
5	Achievement Striving	8.93	2.31	-.10	-.25**	.47**	.30**	

**p < .01

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3	Acting Out Hostility	13.22	1.73	-.23**	-.33**			
4	Impatience Irritability	4.86	1.42	-.28**	-.36**	.20*		
5	Achievement Striving	8.93	2.31	-.10	-.25**	.47**	.30**	

**p < .01

Discussion

The present study concerned the development and construct validation of the Type A Component Questionnaire - a multidimensional measure of CHD patients' Type A behaviour comprising the dimensions, achievement striving, impatience irritability, hostility, anger-in and anger-out. The initial step in the questionnaire's development involved the amalgamation of existing scales said to reflect the five components. The data derived from these was then subjected to PCA. The PCA yielded five factors reflecting achievement striving, impatience irritability, hostility, anger-in and anger-out. The five factor solution was verified using all techniques of factor analysis. Thus, support was found for the hypothesis stating that Type A behaviour is a multidimensional construct comprising achievement striving, impatience irritability, hostility, anger-in and anger-out and, also, for the construct validity of the Type A Component Questionnaire developed in this study.

The present study addresses a primary criticism of past Type A research, namely, the conceptualisation and application of Type A behaviour as a global construct. The majority of studies into the relationship between Type A behaviour and primary and secondary CHD have operationalised Type A behaviour as a global construct with equivocal results (Edwards et al., 1990a). Research based on the global construct would not have revealed the independent contribution of Type A components. Further, use of the global construct would also have eliminated the possibility of identifying the toxic and benign attributes of the different components. Research which has operationalised Type A behaviour as a multidimensional construct has established that the components exert differential effects, some benign and some toxic (e.g., Abbott & Peters, 1988; Helmreich et al., 1988; Öhman et al., 1989;

Spence et al., 1987). The collapse of these into a single measure is likely to have generated a cancelling effect between the benign and toxic components (Carver, 1989). In turn, this may have resulted in non significant relationships, where, in fact, significant relationships occurred (Carver, 1989). Measuring each component independently, as was done in the present study, makes it possible to identify the underlying structure of the Type A construct and therefore, increase the accuracy of Type A research (Edwards & Baglioni, 1991; Helmreich et al., 1988).

Past Type A research has also been criticised for being insufficiently comprehensive (Edwards & Baglioni, 1991). Specifically, past research has been criticised for failing to consider anger and hostility as central components of Type A behaviour (Edwards et al., 1990a; Matthews, 1982). Anger and hostility are implicated in both the original definition of Type A behaviour (Edwards & Baglioni, 1991; Price, 1982; Wright, 1988) and in the pathogenesis of primary and secondary CHD (Blumenthall et al., 1984; Matthews, 1988; Palmer et al., 1992; Taylor & Cooper, 1989). Therefore, anger and hostility are crucial to research concerning the relationship between Type A behaviour and CHD. The criticism pertaining to the lack of comprehensiveness of past Type A research was addressed in the present study by the inclusion of measures of anger and hostility in the Type A Component Questionnaire. The fact that the present confirmatory factor analysis revealed independent factors reflecting anger and hostility, supports the contention that both are central, but separate, components of Type A CHD patients behaviour.

The numerous forms of anger and hostility delineated by past research have contributed to the ambiguity surrounding the definition of Type A behaviour (Meininger et al., 1991; Palmer et al., 1992). The identification of the specific types

of anger and hostility relevant to research on Type A behaviour in the present study serves to reduce this ambiguity. Two conceptually distinct forms of anger have been identified in the literature concerning psychosocial predictors of CHD, namely anger suppression and anger expression. Anger suppression (i.e., anger-in) and anger expression (i.e., anger-out) are correlates of Type A behaviour (Wright, 1988). Past research has revealed relationships between anger-in, anger-out and primary and secondary CHD (Haynes et al., 1980; Johnson & Broman, 1987; Wright, 1988). Therefore, both modes of anger expression were accommodated in the present study. It is evident from the present results that anger-in and anger-out are, indeed, components of Type A CHD patients' behaviour. Therefore, both modes of anger expression should be considered in research into the Type A behaviour of CHD patients and its effect on their health.

Like anger, two primary forms of hostility have been identified by past research, namely, neurotic hostility and aggressive hostility (Dembroski et al., 1989; Engebretson & Matthews, 1992). From the literature, it is evident that it is aggressive, and not neurotic, hostility which is predictive of health outcomes (Carmody et al., 1989; Dembroski & Costa, 1987; Engebretson & Matthews, 1992). Aggressive hostility is also more consistent with the original definition of Type A behaviour than neurotic hostility (Friedman & Rosenman, 1974). As a consequence, the current trend in research concerning the relationship between Type A behaviour and health is to disregard neurotic hostility in favour of aggressive hostility (Engebretson & Matthews, 1992). Given that the hostility component derived from the factor analysis of the present study reflects aggressive hostility, the Type A Component Questionnaire complies with this trend.

Research which has examined anger and hostility as correlates of Type A behaviour has been criticised for failing to examine the equally relevant components of achievement striving and impatience irritability (Jennings, 1984). A number of researchers (e.g., Barling & Charbonneau, 1992; Bluen et al., 1990; Helmreich et al., 1988; Lee, 1992; Pred et al., 1986; Spence et al., 1989) have demonstrated that achievement striving and impatience irritability are primary components of Type A behaviour. The aforementioned research has shown consistently that achievement striving is not associated with measures of health. However, direct links between impatience irritability and health in general and the development of CHD in particular have been established by past research (e.g., Matthews et al., 1977; Rime et al., 1989). The differential effects of the two components revealed in past research clearly indicates the need to measure achievement striving and impatience irritability independently. The results of the present study support the inclusion of separate measures of achievement striving and impatience irritability in models of CHD patients' Type A behaviour.

Confirmatory factor analysis supported the hypothesised factor structure of the model of Type A behaviour. However, there is one result of the analysis which warrants further discussion, namely, the emergence of an item reflecting temper on the achievement striving factor. While this finding was unexpected, closer scrutiny of the Type A construct provides a plausible explanation. Evans (1990) maintains that achievement striving is a superficial attribute based on a profound sense of inadequacy and low self-esteem (Evans, 1990). Incessant striving behaviour is generated by the need to maintain self-esteem through perpetual achievement (Price, 1982). Situations which carry insufficient control to facilitate achievement frustrate

those Type A's who demonstrate achievement striving behaviour (Evans, 1990). Frustration of Type A's efforts at achievement typically results in outbursts of temper and animosity which are directed at the perceived source of interference (Carver et al., 1976; Glass, 1977a; Yuen & Kuiper, 1992). Paradoxically, the perceived loss of control associated with outbursts of temper would not necessarily result in a negative self-evaluation, for Type A's have been shown to rate their ideal selves as demanding and dominating (Henley & Furnham, 1989). In terms of their perception of the ideal, Type A's may regard a volatile temper as a necessary and indeed, desired aspect of achievement oriented behaviour. Thus, Type A theory supports the inclusion of an item reflecting temper in the factor measuring achievement striving behaviour.

There also exists empirical support for the inclusion of the temper item. In a factor analytic study aimed at determining whether the JAS reflected the multidimensionality of the Type A construct, Gallant and Chrisjohn (1985) report a factor almost identical to the achievement striving factor reported here. Specifically, their factor comprised five items, four of which reflected achievement striving behaviour and one reflected temper. It is of note that all four of the items in the fifth factor of the present study were identical to those yielded by Gallant and Chrisjohn's study. A similar factor is reported by Edwards et al. (1990a) who conducted factor analysis on the JAS, Framingham Type A Scale and the Bortner scale. In this study, an item delineating temper loaded with the achievement striving items. Thus, this finding is consistent with extant research.

The development of the Type A Component Questionnaire addresses several of the primary criticisms levelled at past research on Type A behaviour. First, the criticism

concerning the global conceptualisation and operationalisation of the construct was addressed by the provision of independent measures of the Type A components in the Type A Component Questionnaire. Second, existing measures of Type A behaviour have been criticised for their lack of comprehensiveness in general, and their failure to include measures of anger and hostility in particular. While achievement striving, impatience irritability, anger-in, anger-out and hostility are all central components of Type A behaviour (Taylor & Cooper, 1989), past research has failed to reconcile all five components in a model of Type A behaviour. This criticism was addressed by the inclusion in the questionnaire of five of the primary components of Type A behaviour, including measures of anger and hostility. The inclusion of measures of anger and hostility increases the comprehensiveness of the construct and renders it more consistent with the original definition of Type A behaviour. The ambiguity surrounding the definition of Type A behaviour which has elicited further criticism was reduced in the present study by the identification of the specific types of anger and hostility which are associated with the Type A construct. From the literature it is evident that insufficient provision has been made for the potential difference between the Type A behaviour of healthy individuals and the behaviour of individuals with CHD and its effect on health. Indeed, a review of the literature failed to reveal any models or measures of Type A behaviour which incorporate the five central components and which have been developed for samples of CHD patients. In the present study an attempt was made to overcome this insufficiency by the development of a model and questionnaire which reflects the specific behaviour of cardiac rehabilitation patients. Whether the Type A behaviour of CHD patients does exert a differential effect on health will be explored in the study outlined in the following chapter.

Theoretical Implications of the Present Study

The present study has both theoretical and practical implications for future research. The study confirms that achievement striving, impatience irritability, hostility, anger-in and anger-out are components of the behaviour manifested by cardiac rehabilitation patients and further, that they exist independently of each other. By identifying the underlying structure of the Type A construct, the present study increases precision of the conceptualisation of Type A behaviour (Ganster et al., 1991). The model developed in the present study provides a necessary tool for the examination of Type A components in relation to health outcomes and consequently, facilitates understanding of the mediating factors which underlie manifestations of the behaviour (Ganster et al., 1991). Further research will be required to determine the predictive utility of this model. To this end, the third study of the present research will incorporate the model of Type A behaviour in a model of the psychological and behavioural predictors of change in cardiac rehabilitation patients' levels of anxiety, depression and maximal oxygen uptake. By assessing the multidimensional model of Type A behaviour in relation to health outcomes, it will be possible to determine the predictive utility of the model.

The present study also addressed a primary weakness of past research, namely, the almost exclusive use of scales which are multidimensional in nature but which are used to obtain a unidimensional score. These scales violate the assumption of congeneric parts which is fundamental to classical measurement theory in general and reliability assessment in particular (Edwards et al., 1990a). The internal

reliability of existing scales is thus questionable. The present study overcame this weakness by constructing a multidimensional questionnaire comprising component specific scales of Type A behaviour. The scales were found to be congeneric and therefore, their respective reliability coefficients could be taken as a true reflection of their internal consistency. Thus, the questionnaire can be said to be based on sound psychometric principles.

Practical Implications of the Present Study

Determination of construct validity is an infinite process with the result that a measuring instrument's validity can never be declared conclusive (Nunnally, 1978). At best, it can be said that there exists strong support for a measuring instrument's construct validity (Hinkin & Schriesheim, 1989). In as much as the extraction criteria associated with the PCA demonstrated that the five factor model was appropriate for the observed data, the present study yielded support for the construct validity of the five dimension Type A Component Questionnaire. Further support for the questionnaire's construct validity was provided by the consistent emergence of a five factor solution across different methods of factor analysis. The questionnaire also demonstrated satisfactory internal and temporal reliability. It is widely held that providing reliability and validity have been established, a measuring instrument is ready for use in experimental studies (Guyatt et al., 1987) Given that the Type A Component Questionnaire demonstrated internal and external consistency and temporal stability, it can be considered to possess sufficient psychometric properties to justify its use in future research concerning the Type A behaviour of CHD patients. This justification is strengthened by the fact that the questionnaire addresses the conceptual and measurement weaknesses of past

research. A primary weakness was addressed by disentangling the key dimensions from the global construct. These being, achievement striving, impatience irritability, acting-out hostility, anger-in and anger-out.

The fact that the five dimensions were so clearly and consistently represented by the observed data demonstrates the utility of examining Type A behaviour as a multidimensional construct. A further advantage of developing independent scales is that it provides future research with the possibility of identifying specific relationships between dimensions. This possibility is enhanced by the fact that the questionnaire developed in the present study included all dimensions relevant to research concerning the relationship between dimensions of Type A behaviour and CHD. Existing scales have been criticised for failing to include all relevant dimensions and consequently, for hindering attempts to examine the impact of Type A behaviour on prognosis (Powell & Thoreson, 1985). The questionnaire developed in the present study supersedes extant scales by including measures of anger and hostility.

From the literature, it is evident that anger and hostility are two of the most pathogenic components of Type A behaviour (Blumenthal et al., 1984; Lee, 1992; Matthews, 1988). Significant relationships between anger, hostility and recurrent CHD have been reported in past research (e.g., Koskenvuo et al., 1988; Matthews et al., 1977; Wright, 1988). Relationships between anger, hostility and indices of psychological ill health have also been reported (e.g., Biaggio & Godwin, 1987; Edwards & Baglioni, 1991; Spielberger et al., 1985). Therefore, it is imperative that anger and hostility be considered in research concerning CHD patients prognosis for physiological and psychological recovery. The Type A Component Questionnaire

includes measures of anger and hostility and consequently, provides future research with a tool with which to determine the existence of anger and hostility and the extent to which these components predict prognosis.

The present study provides a reliable and valid self report measure of Type A components which is both an adequate measure of the Type A construct and appropriate for use with samples of cardiac rehabilitation patients. By including independent measures of the Type A components, the scale provides an opportunity for establishing which of the components are toxic and which are benign. It therefore facilitates the identification of subgroups within the cardiac rehabilitation population who are at greater risk of contracting secondary CHD and suffering protracted psychological strain. With the Type A Component Questionnaire it will be possible to not only screen those patients who are at greater risk but also to monitor any changes in their behaviour and the corresponding changes in the relationship between the toxic components of behaviour and health outcomes. Provision of a self report measure also eliminates the time and personnel constraints imposed by more objective measures such as the Structured Interview and supports the continued research into the impact of Type A components on secondary CHD.

Limitations of the Present Study

The psychometric properties of the scale may be compromised by the following factors. First, the Type A Component Questionnaire was validated on a single sample. It is argued that factor extraction based on mathematical criteria (e.g., Kaiser's rule, Kaiser's MSA and Cattell's scree test), may generate over factoring and fragmentation of the real factor structure and consequently, that the most robust

and meaningful factor structure is one that can be replicated across samples (Welch et al., 1990). As replication provides the most acceptable form of validation (Grossarth-Maticek & Eysenck, 1991) it is necessary to replicate the five factor solution in an additional sample of cardiac rehabilitation patients. The study outlined in the following chapter will address this limitation by subjecting a second sample of cardiac rehabilitation patients' responses to the 26 item questionnaire to PCA and comparing the factor structures of the two groups. This comparison will determine whether the Type A Component Questionnaire is consistent across samples and possesses external validity within the South African context. It will remain the task of future research to prospectively validate the questionnaire (Tett et al., 1992).

Second, the scale has been developed for a sample of predominantly white, male cardiac rehabilitation patients and therefore, may be less sensitive to assessing the Type A behaviour of other groups of rehabilitation patients. As CHD is a gender linked disease, and in South Africa, is prevalent among the white male population and relatively rare among the black population (Wyndham, 1979), the present sample approximates the demographic distribution of CHD in South Africa. However, as the incidence of CHD is also high among South African Indian males, and past studies have shown that both Type A behaviour and Type A related CHD risk are equally distributed among black and white samples (Thoreson & Powell, 1992), future research should assess the generalisability of the Type A Component Questionnaire to other race groups.

Third, the generalisability of the Type A Component Questionnaire to samples of individuals free of CHD may be questioned. CHD diagnosis is believed to exert a

moderating effect on behaviour to the extent that multidimensional models of Type A behaviour developed for low risk samples may not reflect CHD patients' behaviour (Palmer et al., 1992). Given the potential disparity between the behaviour of high and low risk samples and the effects thereof (Abbott & Peters, 1988; Orth-Gomèr & Undèn, 1990), the development of Type A measures should be population specific (Leon et al., 1988). The aim of the present study was to develop a model of Type A behaviour which was specific to the behaviour of CHD patients and not to Type A behaviour in general.

Fourth, reports of the scale's temporal consistency are based on only one test-retest administration on a single sample (Bluen & Barling, 1987). Given that Type A components reflect relatively stable personality traits (*cf.* Bergman & Magnusson, 1986), this is unlikely to undermine the reliability of the scale. Nevertheless, the temporal consistency will be assessed on the additional sample described in Chapter 8. Finally, further research is needed to assess the predictive power of the scale. To explore the scale's potential fully, such research should be based on a longitudinal, multivariate design (Bluen & Donald, 1991). Study 3 of the present research addresses this limitation by examining the extent to which the Type A Component Questionnaire is able to predict prognosis for recovery from CHD over a six month period.

Conclusion

Ganster et al. (1991) highlight the futility of attempting to define Type A behaviour by correlating imperfect scales with imperfect models of the construct. By developing

a Type A measure which is based on a theoretically sound model of Type A behaviour and possesses adequate psychometric properties, the present study contributes to a more precise definition of the construct. Existing definitions of Type A behaviour have evolved from observations of people who developed CHD with the result that there is insufficient understanding of how the behaviour interacts with the disease (Abbott & Peters, 1988). In redefining the Type A construct in the light of both classical and recent Type A literature, the present study yields a reliable and valid self-report measure that takes cognisance of the multidimensionality of the construct by including independent measures of the five key dimensions of Type A behaviour. Having developed and validated a multidimensional measure of Type A behaviour it is possible to examine more closely the extent to which the five dimensions differentially predict recurrent CHD or recovery. That is, it is possible to assess the interaction between Type A behaviour and CHD. Different individuals are likely to display different combinations of the five dimensions which will either increase or decrease their risk of recurrent CHD (Matthews, 1982). It is believed that the more toxic components of Type A behaviour individuals possess, the more likely they will be to suffer further manifestations of CHD (Taylor & Cooper, 1988). Given this, increasingly widespread, belief, it is important that those dimensions which relate consistently to CHD be isolated with the ultimate view of identifying means by which they can be modified (Dembroski et al., 1985). The ability of the five components, and the combinations thereof, to predict either recurrent CHD or recovery will be examined in the study detailed in the following chapter.

CHAPTER EIGHT

STUDY 3: ASSESSMENT OF THE RELATIONSHIPS BETWEEN TYPE A AND EXERCISE COMPONENTS AND INDICES OF PSYCHOLOGICAL AND PHYSIOLOGICAL HEALTH.

CHD is a primary cause of death and disability in most industrialised countries (Fuster, 1993). The resultant cost to society is considerable (Palmer et al., 1992). So too, is the psychological impact of the disease on the surviving individuals (Palmer et al., 1992). It is for these reasons that the identification of CHD risk factors and appropriate intervention strategies have become of paramount importance and indeed, have resulted in a substantial investment into CHD research (Palmer et al., 1992). While a portion of CHD research has been directed at identifying factors which influence adaptation to CHD (Carmody et al., 1989; Desharnais et al., 1990), there is still insufficient attention paid to the survival and adjustment patterns of CHD patients (Johnson & Morse, 1990; Martin & Lee, 1992). Personality factors influence both the perception and the consequent methods of coping with CHD (Martin & Lee, 1992). Participation in an exercise based rehabilitation programme has also been shown to influence psychological and physiological adaptation to CHD (Bethell & Mullee, 1990; Brown, 1991; Erdman et al., 1986; Layman, 1974; Selye, 1976). While adaptation is now thought to be a product of the complex interaction between cognitive, affective and behavioural responses, the precise nature of these relationships has yet to be determined (Desharnais et al., 1990).

The majority of CHD studies which have examined the relationship between psychosocial factors and CHD have focused on the impact of Type A behaviour, and the components thereof, on the development of the disease (Carmody et al., 1989). Insufficient attention has been paid to the impact of Type A components on

prognosis for recovery (Johnson & Morse, 1990). From the studies which have examined the impact of CHD patients' behaviour on prognosis, it appears that the relationship between Type A behaviour and primary CHD is different to the relationship between Type A behaviour and secondary CHD (Anderson et al., 1986). Thus, there is a need to determine the specific effects of the components of CHD patients' Type A behaviour on their psychological and physiological health.

Like much of the early work on Type A behaviour, research into the behaviour of CHD patients has been based primarily on the global Type A construct. A number of studies have shown that the Type A construct comprises subcomponents (Cramer, 1991). It is now believed that some components of the Type A construct, such as achievement striving, are not associated with health, while others like impatience irritability, hostility, anger-in, and anger-out are predictive of CHD (Ganster et al., 1991). No attempt has been made previously to combine all five of these components in a model of CHD patients' behaviour and to assess the differential effects of these on patients' psychological and physiological health. As a consequence, important relationships between the Type A components and different outcomes of CHD diagnosis may have been obscured in past research (Steinkamp, 1990). The present study will address the limitations of past research by developing and testing a model which proposes that achievement striving, impatience irritability, hostility, anger-in, and anger-out will differentially effect psychological and physiological outcomes of CHD.

An attempt will also be made in the present study to determine the differential effects of exercise frequency and duration on the psychological and physiological outcomes of CHD. A number of studies have sought to determine whether attending an exercise based rehabilitation programme reduces CHD patients' anxiety and depression and increases their maximal oxygen uptake (e.g., Bethell & Mullee, 1990; De Geus et al., 1993; Erdman et al., 1986; Gorman & Kaslow, 1983;

Grodzinski et al., 1987; Kavanagh et al., 1977; Shephard et al., 1985; Stern & Cleary, 1981). It is believed that the ability to increase the frequency and duration of their exercise performance enhances patients' confidence in their physical condition and overall adjustment to CHD (Rejeski et al., 1985). Successful adjustment is associated with a reduction in the affective disorders anxiety and depression (Layman, 1974). Indeed, significant reductions in anxiety and depression following participation in an exercise programme have been reported (e.g., Erdman et al., 1986; Folkins, 1976; Kavanagh et al., 1977; Schomer & Noakes, 1983; Shephard et al., 1985; Stern et al., 1983; Valliant & Asu, 1985). However, other studies (e.g., De Geus et al., 1993; Pauly, Palmer, Wright, & Pfeiffer, 1982; Stern & Cleary, 1981; Van Dixhoorn et al., 1983) have failed to reveal a significant relationship between exercise, anxiety and depression. A possible explanation for these non-significant results lies in the use of attendance as the single measure of compliance. As stated previously, cardiorespiratory fitness is contingent on the frequency and duration of exercise (Pollock et al., 1975; Rejeski et al., 1984). Therefore, any assessment of changes in personality factors associated with increased fitness must consider both measures of exercise compliance. Inclusion of both measures of compliance may shed light on the cause of the non significant relationships revealed in some studies.

The results of research concerning the relationship between attending an exercise programme and enhanced maximal oxygen uptake are more compelling, with the majority of studies revealing a significant relationship between exercise and improved maximal oxygen uptake (e.g., Bethell & Mullee, 1990; Grodzinski et al., 1987; Stern & Cleary, 1981). However, maximal oxygen uptake is not only a product of the frequency of exercise but also of exercise duration (Pollock et al., 1975; Rejeski et al., 1984). Therefore, knowledge of the relationship between exercise and physiological outcomes will be enhanced if it can be determined whether the duration component of compliance is equally associated with maximal oxygen uptake. The present study will incorporate the frequency and duration measures of

exercise compliance in the model of predictors of psychological and physiological outcomes of CHD. Thus, the first aim to be achieved in the present chapter is the development of a model comprising some of the primary predictors of CHD patients' psychological and physiological health. The second aim is to test the model empirically on a sample of CHD patients.

Development of a Model of Psychological and Physiological Outcomes of CHD Patients' Type A and Exercise Behaviour

In the present chapter, a model of the psychological and physiological outcomes of CHD patients' Type A and exercise behaviour is proposed (see Figures 8.1 and 8.2). Included in the model are the five components of CHD patients' Type A behaviour which were identified in Chapter 4 and tested in Chapter 7, namely, achievement striving, impatience irritability, hostility, anger-in and anger-out. Also included are the frequency and duration of exercise compliance. The rationale for incorporating both measures of exercise compliance is based on the results of Study 1, in which it was shown that when taken alone, attendance figures do not predict changes in psychological and physiological outcomes. It is proposed that the aforementioned seven variables will be independently and differentially associated with changes in anxiety, depression and maximal oxygen uptake. Specifically, it will be argued that achievement striving is not associated with changes in anxiety, depression and maximal oxygen uptake. It will also be argued that impatience irritability, hostility, anger-in and anger-out will predict an increase in anxiety and depression, but a decrease in physiological health as measured by maximal oxygen uptake. Conversely, the frequency and duration of exercise compliance will predict a decrease in anxiety and depression but an increase in maximal oxygen uptake. The relationship between each predictor and each outcome variable is described below.

Figure 8.1
Model Of the Hypothesised Positive Relationships Between Type A and Exercise Compliance Components and Changes in Psychological and Physiological Health.

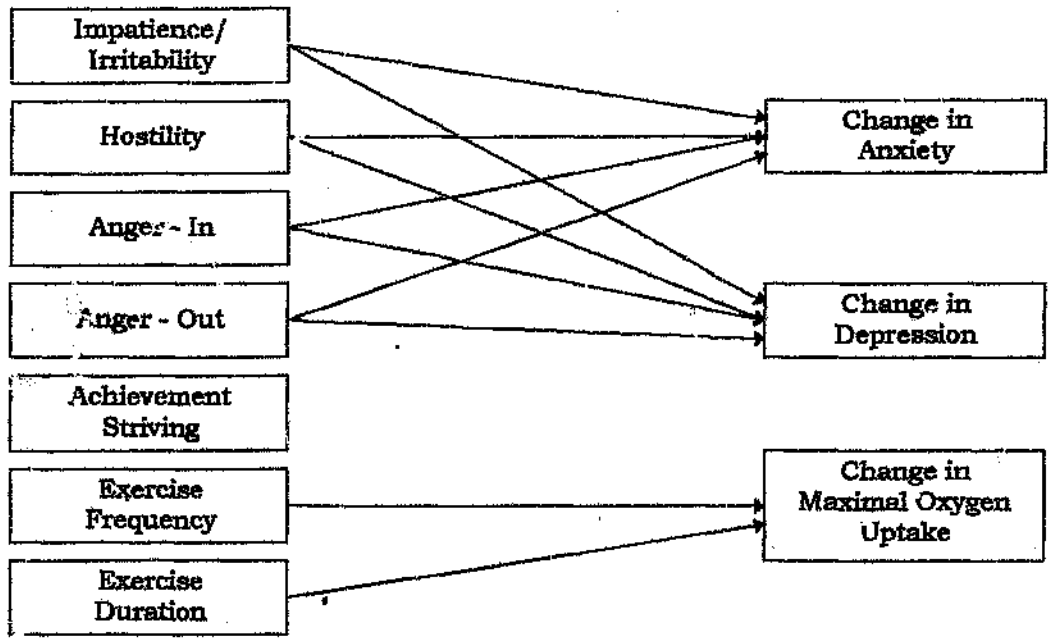
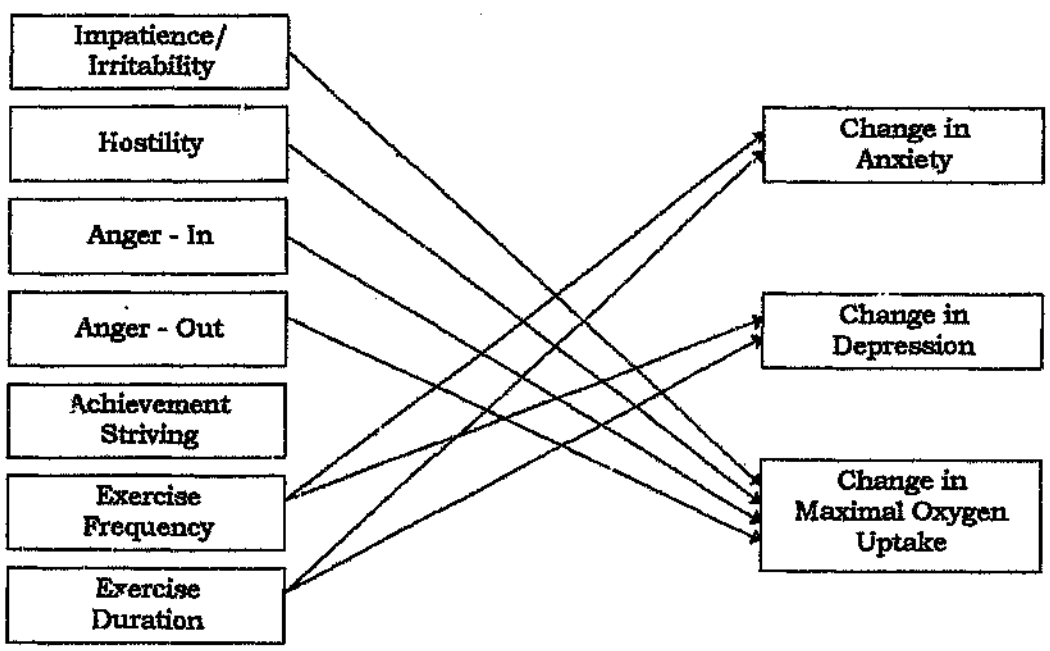


Figure 8.2
Model Of The Hypothesised Negative Relationships Between Type A and Exercise Compliance Components and Changes in Psychological and Physiological Health.



Type A's possess unrealistically high expectations of their ability to control the success rate of their endeavours (Furnham & Linfoot, 1987). With the maintenance of their self-esteem being subject to something as erratic as achievement, Type A's leave themselves vulnerable to failure (Price, 1982). While Type A's are thought to suffer more disappointment in the face of perceived failure than Type B's (Furnham & Linfoot, 1987), little effort has been made to investigate the psychological consequences of this failure. Anxiety and depression are two possible consequences of Type A's perceived failure. CHD patients typically respond to daily stressors with anxiety and depression (Suarez et al., 1991). The present study will attempt to bring greater specificity to past research findings by examining the relationship between the stress inducing components of Type A behaviour (i.e., impatience irritability, anger-in, anger-out and hostility) and changes in CHD patients' anxiety and depression.

Change in Anxiety

CHD generates anxiety concerning the finiteness of life, a sense of powerlessness and dependency, and the ability to function normally in the future (Erdman, 1990). Type A's are anxiety prone (Evans, 1990). In compatison to healthy individuals, CHD patients demonstrate higher levels of anxiety (Martin & Lee, 1992). Therefore, it is likely that Type A CHD patients are particularly vulnerable to feelings of anxiety. The present assertion that the toxic components of Type A behaviour will predict anxiety is based less on empirical findings than on the nature of Type A behaviour.

Achievement Striving, Impatience Irritability and Change in Anxiety

A characteristic of Type A behaviour is the need to maintain self-esteem through acquisitions and achievements (Perry et al., 1990). Frustration with their physical vulnerability and inability to maintain the acquisitions and achievements at pre-CHD levels results in Type A CHD patients demonstrating impatience irritability (Erdman, 1990). Type A CHD patients' perpetual need to maintain a high level of acquisitions and achievements and the impatience and irritability when this need is unfulfilled, may lead to high levels of anxiety. However, it must be emphasised that among Type A's, it is the fear of losing something that is perceived to be intrinsic to esteem and not achievement striving *per se*, which generates anxiety (Drever, 1958). Thus, while the impatience irritability associated with the perceived inability to achieve may generate anxiety, achievement striving will have no impact on levels of anxiety. Empirical support for this contention is provided by Edwards and Baglioni (1991) who found that impatience irritability, but not achievement striving, predicted anxiety. Partial support is provided by Edwards et al. (1990b) who report that the Bortner equivalent of the impatience irritability scale was positively related to anxiety in one of the three samples assessed in their study.

CHD patients high in impatience irritability are likely to demonstrate an anxious response to exercise based cardiac rehabilitation. With exercise training, impatient irritable individuals' need for swift achievement is frustrated by the nature of the task. The exercise prescribed for CHD patients requires steady effort over a lengthy period of time. The potential risk of reinfarction necessitates that progress is slow (Fletcher et al., 1992; Kannel, 1982). Because this process denies individuals high in impatience irritability the possibility of achieving the goal of recovery with minimal time and effort, it constitutes a source of frustration (Feather & Volkmer, 1991). Individuals characterised by impatience irritability avoid tasks which require effort at a slow pace (Feather & Volkmer, 1991). Where they are unable to avoid

such tasks, these individuals experience considerable anxiety (Feather & Volkmer, 1988). It is plausible that, in the context of cardiac rehabilitation, impatient irritable individuals' anxiety is a response to the inability to avoid the slow pace of exercise training.

Hostility and Change in Anxiety

Among samples of CHD patients, hostility is associated with anxiety (Cleveland & Johnson, 1962). The association between hostility and anxiety can be attributed to the hyper-vigilance and insecurity which form the foundation of hostile behaviour. The mistrust and suspicion characterising hostility (Houston & Vavak, 1991; Smith & Frohm, 1985) causes hostile individuals to observe vigilantly their social environment for threats of mistreatment at the hands of others (Christensen & Smith, 1993). Hyper-vigilance also occurs in response to perceived threats to the environmental control so intrinsic to Type A behaviour (Hart, 1984). This hyper-vigilant state is associated with anxiety (Mathews, 1990). According to the DSM-IV, (American Psychiatric Association, 1994) elevated vigilance is a criterion used to diagnose clinical anxiety. As CHD constitutes a considerable threat to control (Budnick, 1991; Rejeski et al., 1985), it is possible that the period of recovery will increase hostile CHD patients' hyper-vigilance and in turn, their level of anxiety.

Also central to hostile behaviour is a pervasive sense of insecurity (Houston & Vavak, 1991; Williams et al., 1985). CHD patients experience considerable insecurity regarding life expectancy, employment, financial and family status, self-esteem, identity and the ability to control their altered physical status (Budnick, 1991; Rejeski et al., 1985; Wilson-Barnett, 1979). It is possible that the insecurity which often occurs in the period of recovery, is manifested as hostility and results in increased anxiety (Cay et al., 1972; Groenman et al., 1990; Wilson-Barnett, 1979). That Type A's experience more anxiety than Type B's (Furnham, 1990a), is

attributed to the Type A's insecurity regarding their abilities (Yuen & Kuiper, 1992). Past research (e.g., Cleveland & Johnson, 1962) has demonstrated a significant relationship between CHD patients' hostility and anxiety. Further, the excessive discharge of norepinephrine initiated by hostile, Type A behaviour is related to increased anxiety (Braestrup, 1982; Weidner et al., 1987). Therefore, research provides a conceptual and empirical link for the association between hostility and a limited reduction in anxiety.

Anger and Change in Anxiety

Type A's hold themselves responsible for uncontrollable outcomes and demonstrate anger when unable to overcome such outcomes (Furnham & Linfoot, 1987). Given the predisposition to take responsibility for all outcomes, it is likely that Type A's will respond to an uncontrollable outcome such as CHD with anger. This anger is associated with the perceived inability to control goal achievement (Matthews, 1982). The maladaptive nature of this process is compounded by the fact that, in the face of failure, Type A's employ goal achievement strategies that are impossible to fulfil (Matthews, 1982). Thus, Type A's have a persistent perception that they are continuously failing to fulfil increasingly higher goals and that this failure is the exclusive product of insufficient ability (Matthews, 1982). Type A's do in fact, achieve less of their personal goals than Type B's (Ward & Eisler, 1987). As Type A's self-esteem is contingent on the achievement of goals, the perceived failure to do so, will generate frustration and self-directed anger (Matthews, 1982). Whether this anger is suppressed or expressed, it is likely to generate an increase in anxiety.

Anger-in and Change in Anxiety

Among certain Type A individuals, expression of anger compromises the need to be regarded publicly as being in control (Price, 1982). This situation is compounded

within the context of cardiac rehabilitation where CHD patients often experience a sense of physical vulnerability (Erdman, 1990). CHD patients' vulnerability may generate a fear that others will perceive them as weak and out of control and may result in a tendency to suppress anger (Erdman, 1990). This rigid control of emotion has been observed among CHD patients (Dongier, 1974). The extreme energy needed to suppress anger, and so maintain a public image of strength and control, contributes to a state of anxiety (Cochrane, 1973).

From the results of past research (e.g., Schwartz, Burish, O'Rourke, & Holmes, 1986; Ward & Eisler, 1987), it is evident that the high probability of failure resulting from Type A's unrealistic goal setting is associated with anger and anxiety. Further, the act of suppressing anger is associated with elevated blood pressure (Baer et al., 1979; Manuck et al., 1985), which in turn, is a marker for anxiety (Budnick, 1991; Groenman et al., 1990). Indeed, Spielberger et al. (1985) report significant correlations between anger-in and anxiety. Thus, there is theoretical and empirical support for the hypothesis that CHD patients' suppression of anger will limit their reduction in anxiety.

Anger-out and Change in Anxiety

Extreme manifestations of anger are maladaptive regardless of whether they are suppressed or expressed (Taylor & Cooper, 1988). Type A's compulsion to achieve more in less time also results in expressing anger at anyone or anything which threatens to hinder their excessive striving (Hassmén et al., 1993). This excessive striving takes the form of a constant struggle to control the environment and the people in it (Glass & Carver, 1980). A life devoted to exerting control over others generates anxiety (Ray & Simons, 1982). The perpetual struggle and conflict resulting from this behaviour provides a conceptual link between anger-out and anxiety. The limitations placed on the ability to control goal achievement during the

period of recovery often generates anger among Type A CHD patients. Thus, it is possible that the anger expressed by the CHD patients in the present study will impose limitations on their reduction in anxiety. Empirical support for this possibility is provided by Edwards and Baglioni (1991), who report that the anger / temper component of the JAS, which is conceptually similar to the anger-out subscale used here, predicted anxiety. Spielberger et al. (1985) also report significant correlations between anger-out and anxiety.

From the results of past research (e.g., Brown & Munford, 1984; De Geus et al., 1993) it is possible to assume that the present sample's feelings of anxiety will diminish in the months of recovery. It is hypothesised that impatience irritability, hostility, anger-in and anger-out will limit this reduction in anxiety. It is further hypothesised that achievement striving will have no such effect on anxiety.

The Frequency and Duration of Exercise and Change in Anxiety

A relationship between the frequency with which CHD patients attend the exercise programme and a change in anxiety is proposed in the present model. A relationship between the duration of patients' exercise and change in anxiety is also proposed. Time spent at the rehabilitation centre provides a respite from the anxiety resulting from CHD induced stress (Brown, 1991; Wood, 1977). By distracting patients from the stress in their lives, attendance of an exercise programme facilitates effective coping (Brown, 1991). An exercise programme also provides a valuable source of social support and reinforcement which may moderate anxiety levels (De Geus et al., 1993). It has been argued that it is the psychosocial components of an exercise programme which reduce the incidence of anxiety among CHD patients (Hughes, 1984).

Patients' attendance of the programme provides tangible evidence that they are able

to perform regular exercise of some duration without cardiac symptoms, and consequently, that they have survived relatively intact. The realisation that they are able to exercise for some duration allays anxiety concerning the potential for reinfarction (Soloff, 1978b). The new found sense of mastery and competence derived from increasing the duration of exercise also enables patients to perceive stressors as challenges rather than threats (De Geus et al., 1993). The somatic symptoms of exercise and anxiety are similar (i.e., palpitations, breathlessness and fatigue) (Ledwidge, 1980). As regular exercise increases CHD patients' tolerance of the exercise symptoms, it also increases their tolerance of the symptoms associated with anxiety (Ledwidge, 1980). Further, the ability to exercise to the point of breathlessness without experiencing dangerous cardiac symptoms desensitises patients to the fear of breathlessness associated with CHD and consequently, reduces anxiety (Weg, 1985). The hypothesised positive association between the frequency and duration of exercise compliance and reduced anxiety is supported by a number of studies (e.g., Erdman et al., 1986; Folkins, 1976; Shephard et al., 1985; Stern et al., 1983).

Change in Depression

The combination of Type A's underlying sense of inadequacy (Evans, 1990), the fragility of their self opinion, the corresponding dependency on achievement, and the initial limitations imposed by compromised health, may result in CHD being perceived as a personal failure and a threat to self-esteem. As Type A's are less able to cope with diminished esteem than Type B's (Suls & Wan, 1989), it is possible that the perception that they are failing to meet self-imposed goals undermines Type A's sense of self-worth and results in depression.

Indeed, depression is less related to the actual severity of disease and more to a combination of distorted fears and beliefs and a personality vulnerable to stress

(Wise & Rosenthal, 1982). Numerous studies have demonstrated a heightened susceptibility to stress among Type A's (e.g., Brief et al., 1983; Caplan, 1971; Forgays, 1992; Suls & Wan, 1989). There is also evidence to suggest that Type A behaviour is associated with stress induced increases in depression (*cf.* Francis, 1981). Further, Thoreson and Powell (1992) identify impatience, hostility and anger as three of the manifestations of stress associated with increased depression. It is not surprising then, that, in comparison to Type B's, Type A's are more prone to depression (Howard et al., 1976).

The prevalence of depression is compounded within the context of cardiac rehabilitation. The incidence of depression among medically ill samples is double that which is found in healthy populations (Meakin, 1992). CHD patients in particular have demonstrated significantly more depression than healthy control groups (Martin & Lee, 1992). The increased incidence of depression has been attributed to the reduced self-esteem generated by the temporary invalidism experienced by CHD patients (Brown & Munford, 1984). As past research has revealed a heightened susceptibility to depression among both Type A's (Francis, 1981; Howard et al., 1976) and CHD patients (Martin & Lee, 1992), it is possible that Type A CHD patients are at particular risk of suffering depression.

Achievement Striving, Impatience Irritability and Change in Depression

Initial investigations into the multidimensional nature of Type A behaviour revealed a bi-dimensional construct comprising achievement striving and impatience irritability (e.g., Helmreich et al., 1988; Öhman et al., 1989; Spence et al., 1987). Price (1982) describes the achievement striving component as being dynamic and characterised by initiative, energy, swift decision making and persistence in the area of goal attainment. These features of achievement striving are the antithesis of the apathy and passivity which characterise depression (Finman & Berkowitz, 1989).

The absence of a relationship between achievement striving and depression has been demonstrated by past research (e.g., Bluen et al., 1990; Edwards & Baglioni, 1991). However, the impatience and irritability arising from the failure to achieve is associated with depression (Edwards & Baglioni, 1991).

Impatience irritability has been identified as one of the most representative components of Type A behaviour (Tett at al., 1992). Its relationship to depression is possibly due to both a negative self-perception and the negative perception that others have of the individual high in impatience irritability. Impatience irritability is a product of the need to achieve more in less time as a means of maintaining self-esteem (Price, 1982). As the number of accomplishments achieved varies, so too does esteem (Price, 1982). Real or imagined invalidism among CHD patients hinders achievement which in turn, diminishes esteem (Brown & Munford, 1984). This syndrome, together with the time-urgency related to impatience irritability, is associated with negative self-concept (Furnham, 1990b). Thus, the perpetual need to fulfil unattainable goals generates misdirected behaviour and time pressure which causes depression (Edwards & Baglioni, 1991).

Furnham's (1990b) research shows that, in comparison to individuals low in speed and impatience, individuals who achieve high scores in speed and impatience have a lower perception of self. Similarly, Burke (1984) reports that individuals high in impatience have a greater tendency toward believing that self-worth is a product of accomplishments, that worthwhile resources are in short supply, that there is no justice or universal moral principle and that they are worthless. Together, these beliefs culminate in a perpetual battle against a sense of inadequacy (Furnham, 1990b), all of which lead to a debilitating state of desperation and depression (Kernis et al., 1989; Yuen & Kuiper, 1992). The loss of confidence, sense of uselessness and insecurity and perceived invalidism felt by many CHD patients (Cay et al., 1985) render these individuals particularly vulnerable to feelings of

inadequacy and ultimately, depression.

It is also possible that the negative regard that others have of individuals who demonstrate impatience irritability generates depression. Research on Type A children shows that children high in aggression and impatience enjoy less popularity with their peers (Steinkamp, 1990). As Type A behaviour is a relatively stable construct (*cf.* Bergman & Magnusson, 1986) it is possible that this finding extends to adults. In adults, this syndrome is likely to translate to a fear of an inadequate supply of recognition and appreciation which in turn, results in a fear of being unworthy of esteem and ultimately, unlovable (Price, 1982). Insufficient positive reinforcement from social networks is associated with depression (Brown & Munford, 1984).

Empirical support for the present hypothesis is provided by Chesney et al., (1981) who found that speed and impatience correlated with depression. Bluen et al. (1990) also found that impatience irritability was positively related to depression, whereas achievement striving was not. Perhaps the strongest empirical support is provided by Edwards and Baglioni's (1991) study which examined the differential power of the JAS, Bortner, and Framingham scales in predicting mental and physical symptoms. This study revealed that, in all three scales, the component measuring impatience irritability, or equivalents thereof, predicted depression. In Edwards and Baglioni's (1991) study the components associated with achievement striving did not predict depression. The theory and research reviewed above supports the present hypothesis that impatience irritability among CHD patients will retard the anticipated reduction in depression while achievement striving will not.

Hostility and Change in Depression

It is further proposed that hostility will predict a change in depression. The frenetic,

aggressive lifestyle associated with Type A behaviour influences social behaviour (Barling, Bluen & Moss, 1990). If Type A individuals are high in hostility, this influence is likely to be negative. Hostile individuals are typically antagonistic, rude, callous and uncooperative (Dembroski & Costa, 1987), and consequently, elicit anger, aggression and annoyance from others (Steinkamp, 1990). It is perhaps because of this that hostile individuals report fewer intimate relationships and experience more conflict and mistreatment and less support in their relationships with their partners and family members than less hostile individuals (Christensen & Smith, 1993; Friedman & Rosenman, 1974). As a result, hostile Type A's may experience more social isolation and withdrawal (Chesney, 1993). The sense of social isolation is likely to be more profound among those CHD patients who have had to change their employment status and consequently, already have reduced contact with colleagues and friends (Ruberman et al., 1984). CHD patients who exhibit hostility may alienate those around them who are willing to provide vital support (Budnick, 1991). Barefoot et al. (1983) maintain that individuals high in hostility do report a low quality of social support. With little affirmative feedback from those around them, Type A CHD patients are likely to internalise a sense of inadequacy, which ultimately leads to depression (Brown & Munford, 1984).

Hostility may also be counter-productive to achieving goals (Taylor & Cooper, 1988). If Type A's fail to achieve their goals, they resort to a sense of helplessness (Matthews, 1982). Helplessness is associated with depression (Hildebrand-Saints & Weary, 1989). The reduced and unstable esteem resulting from the perceived failure to achieve is similarly related to depression (Kernis et al., 1989). Feelings of helplessness and loss of esteem are typical responses to CHD (Erdman, 1990). Rosenman (1986) maintains that the maladaptive effects generated by hostility are directly related to depression. Moreover, Biaggio and Godwin (1987) report a significant association between hostility and depression. Therefore, there exists both theoretical and empirical support for the proposal that hostility will limit the present

sample's reduction in depression.

Anger and Change in Depression

The present model also includes anger-in and anger-out as predictors of depression. Type A's are inclined to set personal goals which far exceed their performance potential with the result that they frequently experience a sense of failure (Ward & Eisler, 1987). As the failure to achieve exceedingly high goals is attributed to a lack of ability, Type A's lose faith in their capacity to impose the necessary control on their environment (Matthews, 1982). Among Type A's, failure to achieve excessively high self-imposed goals generates anger (Powell et al., 1993; Schwartz et al., 1986). According to Wright (1992), CHD patients demonstrate significantly more anger-in and anger-out than patients hospitalised for other diseases. Thus, it is possible that Type A CHD patients are particularly prone to anger.

CHD imposes considerable limitations on daily functioning (Erdman, 1990). As a result, CHD patients experience difficulty in maintaining their social and physical activities at levels consistent with their behaviour prior to CHD diagnosis (Denolin, 1985). The reduction in activity imposed in the period of recovery limits Type A CHD patients' opportunity for high achievement and consequently, their confidence and sense of esteem (Brown & Munford, 1984; Johnson & Morse, 1990). The loss of an achievement oriented lifestyle and the corresponding self-perception, together with a new found sense of physical vulnerability, generates anger among Type A CHD patients (Berkowitz, 1983; Budnick, 1991). Depression is likely to occur if CHD patients are unable to resolve this anger (Hackett, 1985). A relationship between CHD patients' anger and depression has been reported in past research (e.g., Dimsdale, Hackett, Block & Hutter, 1978). Therefore, it is feasible that CHD patients' anger will retard their recovery from depression.

Anger-in and Change in Depression

If anger is suppressed and unresolved, it leaves the affected individuals with the impression that they are unable to manage stress (Budnick, 1991). This leads to low self-esteem and ultimately, depression (Novaco, 1976; Powell et al., 1993; Schwartz et al., 1986; Yuen & Kuiper, 1992). Among CHD patients, anger suppression is a typical response to the inability to transform the anger generated by a change in status into socially acceptable forms of behaviour (Hackett, 1985). Similarly, CHD patients typically experience a loss of self-esteem in the period of recovery (Budnick, 1991; Wilson-Barnett, 1979). CHD patients who suppress their anger and experience the concomitant loss of esteem, enter into a state of depression wherein they become listless and lose initiative and interest in life (Erdman, 1990). While significant relationships between anger-in and depression have been demonstrated in research using healthy samples (e.g., Biaggio & Godwin, 1987; Spielberger et al., 1988), no research could be found which has examined this relationship among CHD samples. Thus, an aim of the present study is to determine whether anger-in inhibits the reduction in depression among CHD patients.

Anger-out and Change in Depression

The expression of anger may also result in depression. Frequent anger expression creates destructive and problematic situations wherein the potential for conflict resolution is limited (Harburg et al., 1991). This behaviour is likely to damage interpersonal relationships and result in social isolation (Abbott & Peters, 1988). Impaired social functioning and the consequent loneliness, characterise depression (Siegal & Alloy, 1990; Weisse, 1992). Frequent and destructive outbursts of anger on the part of the CHD patient may alienate the family and friends able to provide the support needed to moderate depression (Budnick, 1991). Thus, there exists conceptual support for the relationship between anger-out and depression.

Empirical support is provided by Biaggio and Godwin (1987) who report a significant correlation between anger-out and depression, albeit in healthy samples.

The theory and research reviewed above forms the basis for the present hypothesis that achievement striving will not predict a change in depression but that impatience irritability, hostility, anger-in and anger-out will limit the expected reduction in CHD patients' depression.

The Frequency and Duration of Exercise and Change in Depression

A concurrent aim of the present study is to examine the extent to which participation in an exercise based cardiac rehabilitation programme predicts a change in depression. The relationship between exercise and CHD patients' depression has been discussed at length in previous chapters. Therefore, only a brief synopsis follows.

In the present model a relationship between the frequency of exercise (i.e., the number of times patients attend the exercise programme) and change in depression is proposed. By attending exercise sessions, patients realise that they not only had the strength to survive CHD, but that they also have the potential strength to engage in physical exertion (Erdman, 1990). The resulting improvement in physical self-efficacy diminishes depression (De Geus et al., 1993). Attending an exercise programme enhances CHD patients' autonomy and sense of control over physical symptoms (Weg, 1985). The increased sense of autonomy and control derived from attending the exercise programme enhances esteem, which in turn reduces depression (Brown & Munford, 1984). Attendance also provides the 'time out' from daily routine and stress necessary to reduce depression (De Geus et al., 1993). The individual attention shown by the medical personnel of the rehabilitation centre when patients attend the programme further reduces the incidence of depression

(Weg, 1985). Thus, it is possible that the frequency with which the present sample of CHD patients attend the exercise programme will have a positive effect on their level of depression.

A relationship between the duration of exercise and change in depression is also proposed in the present model. Depression is often a result of insufficient positive reinforcement to adaptive behaviours (Brown & Munford, 1984). By increasing their level of endurance to exercise, CHD patients are able to provide themselves with the positive reinforcement needed to counteract depression. Being able to exercise for increasing lengths of time enhances self-image (Rovario, Holmes & Holmsten, 1984) by increasing patients' confidence in their physical capacity (Rejeski et al., 1985) and consequently, by altering negative perceptions of health (Brown & Munford, 1984). All of these changes are directly related to a reduction in depression (Brown & Munford, 1984). The development of endurance also counteracts some of the symptoms of depression, namely, decreased motivation, sense of control and energy levels (Tang & Critelli, 1990). Specifically, increased mastery achieved through exercising for longer periods of time enhances motivation and CHD patients' perception that they are able to control the process of recovery (Cay et al., 1985). The increased fitness derived from exercising for longer periods at a time also increases energy levels (Hammond, 1985). The literature reviewed above supports the present contention that the duration of exercise will facilitate the reduction in depression.

While a number of studies (e.g., Erdman et al., 1986; Folkins, 1976; Kavanagh et al., 1977; Schomer & Noakes, 1983; Shephard et al., 1985; Stern et al., 1983; Valliant & Asu, 1985) have demonstrated a significant relationship between the frequency of exercise and reduced depression, none could be found that has examined the relationship between the duration of exercise and depression. Indeed, insufficient attempts have been made in the past to measure the predictive role of

the frequency and duration components of exercise on CHD patients' depression (Fletcher et al., 1992). Therefore, the present study will extend the findings of past research by examining the relationship between the frequency and duration of exercise and reduced depression.

A Physiological Outcome of Type A and Exercise Components

Change in Maximal Oxygen Uptake(VO_2max)

The Type A Components and Change in Maximal Oxygen Uptake

A further assertion of the present study is that impatience irritability, hostility, anger-in and anger-out will predict a change in maximal oxygen uptake, while achievement striving will have no such effect. Chapter 4 provides a detailed theoretical and empirical review of the physiological consequences of achievement striving, impatience irritability, hostility, anger-in and anger-out. Therefore, only a brief summary will be provided here.

Achievement striving is not associated with heightened cardiovascular reactivity (Öhman et al., 1989). Conversely, impatience irritability, anger-in, anger-out and hostility are associated with excessive cardiovascular reactivity (Lassner et al., 1994; Taylor & Cooper, 1989). This reactivity generates a substantial discharge of epinephrine and norepinephrine (Ganster et al., 1982). The frequent and protracted discharge of these catecholamines increases vulnerability to cholesterol infiltration (Janisse & Dyck, 1989) and platelet aggregation (Glass, 1977b) and results in the chronic activation of the sympathetic nervous system (Ganster et al., 1982). Over time, these factors damage the integrity of the cardiovascular system and increase susceptibility to atherosclerosis and CHD (Ganster et al., 1991; Melamed et al., 1993). Maximal oxygen uptake provides an index of the severity of atherosclerotic

build-up in the arteries, and consequently, an indication of physiological strain (Bruce, 1971).

Significant relationships between cardiovascular reactivity and impatience irritability, hostility, anger-in and anger-out have been reported in a number of studies (Christensen & Smith, 1993; Harburg et al., 1991; Jennings, 1981; Manuck et al, 1985; Öhman et al., 1989; Suarez & Williams, 1990; Svebak et al., 1992). Direct relationships between these variables and primary and secondary CHD have also been established (e.g., Barefoot et al., 1983; MacDougall et al., 1985; Matthews et al., 1977; Rimé et al., 1989; Tennant & Langeluddecke, 1985; Wright, 1988). Conversely, research has revealed that achievement striving is unassociated with cardiovascular reactivity (Öhman et al., 1989), CHD, and disease in general (Rimé et al., 1989). Thus, there is considerable theoretical and empirical support for the present hypothesis that impatience irritability, hostility, anger-in and anger-out will predict physiological strain as measured by a decrease in maximal oxygen uptake whereas achievement striving will not be associated with physiological strain.

The Frequency and Duration of Exercise and Change in Maximal Oxygen Uptake

The proposed model also makes provision for a positive relationship between exercise compliance and maximal oxygen uptake. The relationship between exercise compliance and enhanced maximal oxygen uptake has been reviewed in Chapter 3. In summary of this review, regular exercise of sufficient duration increases the cardiovascular system's ability to utilise oxygen as measured by maximal oxygen uptake (Jamieson & Lavoie, 1987). A significant relationship between exercise compliance and increased VO_2max among CHD patients has been demonstrated in a number of studies (e.g., Bethell & Mullee, 1990; Grodzinski et al., 1987). Consequently, a hypothesis of the present study is that the frequency and duration

of exercise will be positively related to increased maximal oxygen uptake (see Figure 8.2).

Method

Sample and Setting

The sample comprised 181 men and women who had participated in the Johannesburg City Health Department's Cardiac Rehabilitation Centre (CRC) programme for a minimum of six months. All patients who had completed the physiological and psychological assessment administered on intake and following six months of participation in the exercise programme and who were not part of the samples used in Studies 1 and 2 were included in the present sample. The demographic characteristics are detailed in Table 8.1. Of the sample, 142 participants had experienced myocardial infarctions, 73 coronary artery bypass grafts, 24 percutaneous transluminal coronary angioplasty and 115 coronary artery angiography. A further 12 participants had had surgery to correct valvular or congenital heart disorders (see Table 8.1). Therefore, many of the patients included in the sample had multiple symptoms.

Procedure

The data used in the present study derive from the archives of the CRC. Specifically, the data comprise demographic information (sex, race, age, marital status, education and occupation), the results of the psychometric and exercise tests conducted on admission and following six months of participation in the programme. Also included is a detailed account of the frequency and duration of exercise performed by each participant over the six month period. The means by which this information was collected is outlined in the Measuring Instruments

section. Permission to use this data was granted by the CRC and the ethics committee of the Johannesburg City Health Department. Use of the data was also approved by the ethics committee of the University of the Witwatersrand. The confidentiality and anonymity guaranteed to CRC members was strictly adhered to.

Table 8.1

Summary of the Demographic Characteristics and the Medical History of the Sample(N=181)

Variable	Number	Percentage
Sex		
Male	163	90 %
Female	18	10 %
Race		
White	172	95 %
Non-white	9	5 %
Marital Status		
Married	149	82 %
Unmarried	32	18 %
Employment Status		
Professional	60	33 %
Skilled	57	32 %
Self-employed	45	25 %
Retired	19	10 %
Education		
Ten Years (Std 8)	25	14 %
Twelve Years (Matric)	60	33 %
Technical Qualification	23	13 %
Professional Qualification	32	18 %
Undergraduate/Postgraduate Degrees	41	22 %
Medical History*		
Myocardial Infarction	142	79%
Coronary Artery Bypass Graft	73	40%
Percutaneous Transluminal Coronary Angioplasty	24	13%
Coronary Artery Angiography	115	64%
Congenital/Valvular Heart Surgery	12	7%

* The majority of the patients included in the sample had experienced more than one of the cardiac events or associated surgical procedures listed above.

Design

The present study is based on an experimental, longitudinal design (Christensen,

1985). Included in the design are seven predictor variables, namely, achievement striving, impatience irritability, acting-out hostility, anger-in and anger-out and measures of the frequency and duration of exercise. The three outcome variables included in the design are changes in anxiety, depression and maximal oxygen uptake. The outcome variables were created by means of change score analyses, whereby the Time 1 scores were subtracted from the Time 2 scores. Provision was also made for the inclusion of covariates. Potential covariates were considered to be sex, race, age, marital status, occupation, and education. Given that stress operates over time (Leventhal & Tomarken, 1987), the longitudinal nature of the design constitutes the appropriate means of assessing stress variables. Further, a longitudinal design which comprises both clinical and experimental tests constitutes the most suitable means of identifying risk factors for CHD (Harburg et al., 1991)

Measuring Instruments

The Type A Component Questionnaire, developed, validated and discussed in Chapter 7, was used to measure the five components of Type A behaviour, namely, achievement striving, impatience irritability, acting-out hostility, anger-in and anger-out (see Appendix C). The Illinois Personality Assessment Test Anxiety Scale (Cattell & Scheier, 1976) and the Beck Depression Inventory (Beck et al., 1961) (see Appendix a), discussed in Chapter 6, were used to measure anxiety and depression, respectively, and a discussion of the scales' psychometric properties and applicability will not be repeated here. The descriptive statistics of all measuring instruments are listed in Table 8.2.

Table 8.2

Descriptive Statistics of All Measuring Instruments

Variable	N. of items	M	SD	Mini-mum	Maxi-mum	Internal reliability	Six month Test-retest
Achievement Striving	4	8.74	2.50	4	16	.67+	.76**
Impatience Irritability	3	4.92	1.43	3	8	.69+	.66**
Anger-in	6	11.20	2.98	6	21	.73+	.66**
Anger-out	8	7.09	3.89	8	19	.78+	.66**
Acting-out Hostility	4	0.08	2.53	4	8	.64+	.49**
Exercise Frequency		53.44	11.15	7	76		
Exercise Duration (minutes)		1882.40	551.10	187	3492		
Anxiety (Time 1)	40	30.08	11.34	5	62	.83++	.76**
Anxiety (Time 2)	40	28.97	11.67	5	62	83++	
Depression (Time 1)	21	7.59	5.02	0	31	.81++	.56**
Depression (Time 2)	21	6.40	4.68	0	31	.81++	
VO2max (Time 1)		26.09	6.67	11 60	47.80		.87**
VO2max (Time 2)		29.91	7.82	14.60	58.10		

** $p < .01$

\+ Reliability assessed by means of Kristof's formula

++ Reliability assessed by means of Cronbach's alpha

Note. Exercise attendance is measured in number of sessions attended. Exercise duration is measured in number of minutes of supervised exercise for the six month period.

Compliance

Exercise compliance was measured in terms of the sum of the number of minutes (i.e., exercise duration) each participant spent exercising and the sum of the sessions attended (i.e., exercise frequency) over a period of six months. The required

frequency and duration of exercise compliance was determined by the exercise prescription compiled by a physiotherapist on the basis of the results of each participants' stress test. All compliance figures were recorded by the participants after each session. The compliance records were closely monitored by the staff at the CRC.

Each session was preceded by a warm-up session involving a series of stretching exercises and movement progressing from a slow, stationary walk to jogging. The goal of the warm up session was to increase the heart rate gradually to the training heart rate indicated by each individual's exercise prescription. A cool-down period, comprising slow walking, followed each session and served to reduce the heart rate to approximately ten beats above the baseline measure.

Exercise Frequency

The prescription dictated that participants exercise three times a week. In the present study, the frequency measure was taken as the total number of sessions attended by each participant over the six month period. In accordance with the prescription, each participant should have attended approximately 72 sessions over the period of study. From the literature, it is evident that the majority of studies have determined the frequency of exercise by calculating the total number of sessions attended (e.g., Bengtsson, 1983; Blumenthal et al., 1982; Blumenthal et al., 1988; Mayou, 1983; Oldridge & Jones, 1983; Roman, 1985; Shaw, 1981). Therefore, the method of calculating the frequency of exercise in the present study, is consistent with past research.

Exercise Duration

At each session, participants were required to exercise for 30 to 45 minutes at their

prescribed intensity. The duration measure reflected the total number of minutes spent exercising at the CRC over six months. Measuring the duration of exercise in this manner is consistent with the method used by Lakka et al. (1994) in their recent study of the relationship between exercise, cardiorespiratory fitness and myocardial infarction.

The inclusion of both measures of exercise compliance was motivated by the current belief that improvements in cardiorespiratory fitness are contingent on the frequency and duration of exercise (Hickson et al., 1981; Froelicher, Myers, Follansbee, & Labovitz, 1993; Pollock et al., 1975; Rejeski et al., 1984; Seals et al., 1984). The need to include both measures of compliance is also supported by empirical research. Several studies (e.g., Pollock et al., 1975; Seals et al., 1984) have established that enhanced maximal oxygen uptake is determined not only by the frequency, but also by the duration, of exercise. Clearly, the optimal benefit of a rehabilitation programme is achieved only if the individual's effort complies with the frequency and duration defined by the exercise prescription set out by medical authorities (Sackett & Haynes, 1976). Thus, statements regarding high compliance and consequent improvements in the intake, transportation and utilisation of oxygen require the measurement of both the frequency and duration of exercise. Hence the inclusion of both in the present study.

Maximal Oxygen Uptake

Maximal oxygen uptake (VO_2max) was used in the present study to measure physiological condition. VO_2max reflects maximal cardiorespiratory function during physical exertion and therefore, provides an objective, but indirect measure of CHD severity (Cohn, Kamm, Feteih, Brand, & Goldschlager, 1979). It is a reliable predictor of prognosis for recovery or reinfarction (Cooper, Meyer, Blide, Pollock, & Gibbons, 1991) and is used widely as a means of prescribing aerobic exercise

among CHD patients (Milesis, 1987).

The present study's use of VO_2max as a measure of physiological strain is supported by past research. Studies (e.g., Cohn et al., 1979; Uhl, Hopkirk, Hickman, Fischer, & Medina, 1984) in which measures of VO_2max and angiographic results have been compared have found that VO_2max is able to predict the extent of disease in the coronary arteries. Measures of VO_2max have also been used effectively to monitor the response to rehabilitative procedures. For example, in a controlled study, Bethell and Mullee (1990) used VO_2max to assess myocardial infarction patients' progress following a three month exercise programme. The improvement in the VO_2max of patients who participated in the programme was significantly greater than the VO_2max of patients who did not. The differences in VO_2max were reflected by a 10% decrease in clinical angina pectoris among the exercising group and a 60% increase in angina pectoris in the non-exercising group (Bethell & Mullee, 1990). Thus, VO_2max is sensitive to changes in the cardiovascular system and the presence of underlying atherosclerosis.

In sum, VO_2max provides a measure of the cardiovascular system's ability to utilise oxygen (Desharnais et al., 1993; Jamieson & Lavoie, 1987). Consequently, it is used as a measure of cardiorespiratory fitness (Desharnais et al., 1993; Pierce, Weltman, Seip, & Snead, 1990; Weltman et al., 1990) and an indication of the severity of CHD (Bruce, 1971). VO_2max is also used to predict prognosis for recovery from a primary coronary event (Crean & Fox, 1987; Sami, Kraemer, & DeBusk, 1979). Therefore, it is considered an appropriate objective measure of physiological strain in the present study.

VO_2max can be determined by assessing the maximal aerobic power achieved on a treadmill or bicycle ergometer test (De Geus et al., 1990). Both methods were used in the present study to determine VO_2max. Specifically, each participant was subjected to the symptom limited multi-stage stress test described below. Currently, stress tests constitute the most widely used method of determining the existence and severity of CHD (Kligfield & Okin, 1994). The majority of participants' tests were based on the Chung protocol (Chung, 1983). Participants who demonstrated a high level of fitness followed the more demanding Bruce protocol (Bruce, 1971). A bicycle ergometer test based on the World Health Organization protocol (WHO Standard Program, 1982) was reserved for participants who, due to physical disability, were unable to walk on the treadmill. As few participants' tests were based on the Bruce and World Health Organization protocols, only the Chung protocol is outlined below.

Prior to conducting the symptom limited multi-stage stress test, participants were thoroughly informed of the purpose of the test and the procedures involved. They were given standard instructions to exercise to the limit of their ability but were given no further encouragement during the test. After being informed of all matters concerning the stress test, participants were required to sign a form of consent and indemnity. Thereafter, participants were given a physical examination by a medical doctor and asked to provide details of their medical history. Anthropometric tests were then performed and comprised measures of height, weight and percentage body fat. A standard supine 12 lead electrocardiogram[32] (ECG) was conducted to establish a baseline measure of the electrical patterns of the heart.

The stress test comprised a continuous ECG, respiratory gas analysis and blood pressure monitoring. A metabolic cart (Jaeger Sprint) was used to determine oxygen

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[32] *An electrocardiogram provides a graphic representation of the electrical activity of the heart, and consequently, facilitates the diagnosis of heart disease and detection of cardiac abnormalities (Oxford Reference Concise Medical Dictionary, III Edition, 1990).*

uptake, carbon dioxide exhalation and minute volume[33]. Participants were given an opportunity to practice on the treadmill for a few minutes prior to the test. They were then asked to sit down while the oxygen and carbon dioxide analysers and the volume measuring device (i.e., the pneumotach analyser[34]) were calibrated. This information was entered on computer. A mouth piece was then placed in participants' mouths and attached to the pneumotach analyser for the purpose of collecting expired gases. The treadmill was then turned on and participants were asked to start walking in as erect a position as possible. The test proceeded in stages of three minutes. In the last minute of each stage the automatic blood pressure monitor fixed to participants' arms inflated and the reading was recorded on the computer. Heart rate and oxygen consumption were monitored continuously and an average recorded for every 30 seconds. In the second three minute stage, the speed of the treadmill was increased from 2.72 kilometres per hour to 4.8 kilometres per hour in accordance with the Chung protocol. Participants were informed of the speed increase prior to its occurrence. At each subsequent stage, the speed remained consistent and the elevation was increased by four percent every three minutes (Chung, 1983). Participants were asked how they felt at frequent intervals. VO_2max was established as being the amount of oxygen consumption which occurred at the peak of exercise performance.

Termination of the stress test was determined by exhaustion, severe angina, a S-T segment depression exceeding 3mm[35], persistent ventricular tachyarrhythmias[36] and

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33 *The minute volume reflects the total volume of gas expired per minute and is expressed in litres (Jaeger, 1990).*

34 *The pneumotach analyser measures the flow of exhaled gas and integrates it mathematically so as to calculate the minute volume (Jaeger, 1990)*

35 *The S-T segment reflects the interval between the depolarisation of the ventricles and the onset of rapid repolarisation. A S-T segment depression of 1 mm or more indicates that the heart has an inadequate supply of oxygenated blood and predicts between 20% and 50% of cardiac events (Constant, 1986).*

/ or a drop in systolic blood pressure in excess of 10 mm Hg. Participants' ECG and blood pressure readings were also monitored in the recovery period.

Data Analysis

The aim of the present study is to determine the extent to which the five components of Type A behaviour and the two components of exercise compliance predict changes in anxiety, depression and maximal oxygen uptake among a sample of CHD patients. Multiple regression determines the best combination of predictors of a particular outcome variable, and consequently, fulfils the aim of the present study (Mason & Perreault, 1991). Multiple regression is also able to accommodate the complex nature of the variables. Because the five components of Type A behaviour are interrelated (see Table 8.3), the modification of one component may alter the presence and intensity of the other components (Evans, 1990; Price, 1982). The frequency and duration of exercise performance are similarly interrelated. Multiple regression is able to determine both the independent and interactional effects of the predictor variables on the dependent variables (Yuen & Kuiper, 1992). Therefore, multiple regression is particularly suited to the analysis of variables as complex and interrelated as the ones included in the present study. This method of analysis has also been used extensively in research concerning the relationship between psychological and behavioural factors and CHD (e.g., Follick et al., 1990; Harburg et al., 1991; Martin & Lee, 1992).

Using the principles of correlation and regression, multiple regression determines the effect and magnitude of a number of independent variables on a dependent variable (Kerlinger, 1986). The goal of multiple regression is to fit a straight line to a

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[36] *Tachyarrhythmias are increases in the heart rate and abnormalities in rhythm which are often exercise induced (Oxford Reference Concise Medical Dictionary, III Edition, 1990).*

number of points wherein each point represents an individual observation (Kerlinger, 1986). The more the plotted points cluster along the regression line, the greater will be the correlation (Kerlinger, 1986). This procedure is sometimes referred to as linear least squares estimation as the line is computed in such a way that the squared deviations of the various points from that line is kept to a minimum (Groebner & Shannon, 1985). The estimate of the linear equation between the independent and dependent variables takes the following form:

$$Y^1 = \beta + B_1 X_1 + B_2 X_2 + ... B_k X_k$$

(Lewis-Beck, 1980)

In this formula, $\underline{Y}^1$ is the predicted value of the dependent variable $\underline{Y}$. The parameter β is the constant or intercept of the regression line and $\underline{B}$, a slope or regression coefficient which represents the unique contribution of each independent variable to the prediction of the dependent variable (Lewis-Beck, 1980). The scores of the independent variables are represented by $\underline{X}_1$ and the final independent variable in the equation is represented by $\underline{K}$ (Kerlinger, 1986).

The regression line provides the best prediction of the dependent variable when accounting for the independent variables (Lewis-Beck, 1980). There is usually considerable variation of the different points around the fitted regression line. This deviation of a point from its predicted value (i.e., the regression line) is termed the residual value. As the variability of the residual values around the regression line, relative to the overall variability, decreases, so the precision of prediction increases (Lewis-Beck, 1980). A perfect relationship between $\underline{X}$ and $\underline{Y}$ would mean that the ratio between the residual variability of the $\underline{Y}$ variable and the original variance is .0 (Dillon & Goldstein, 1984). Where no relationship exists between the $\underline{X}$ and $\underline{Y}$ variables, the ratio of the variance would equal 1.0 (Dillon & Goldstein, 1984). The ratio typically occurs between the two extremes.

Given that the process of using several variables to predict an outcome variable can result in errors of prediction, it is advisable to subject the data to the principle of least squares wherein the β and $\underline{B_1}$ coefficients are selected (Kerlinger & Pedhazur, 1973). With the least squares principle, the model best able to minimise the distance between the observed and the predicted values is selected (Kleinbaum & Kupper, 1978). By invoking this principle, the sum of the squared residuals is kept to a minimum, and consequently, so too are the squared errors of prediction (Kerlinger & Pedhazur, 1973).

The multiple correlation coefficient $\underline{R}$ provides an index of the size of the relationship between the sum of least squares composite of the independent variables and the dependent variable (Kerlinger, 1986). $\underline{R^2}$ is the square of the multiple correlation coefficient $\underline{R}$, and represents the overall effect of the independent variables on the dependent variable (Kerlinger, 1986). Thus, from $\underline{R^2}$ it is possible to see what percentage of the original variance is explained by the independent variables. As such, $\underline{R^2}$ provides an indication of how well the model fits the data (Lewis-Beck, 1980). The remaining percentage comprises residual variance. With the goal being to explain as much of the original variance as possible, the ideal residual variance should be minimal (Lewis-Beck, 1980). Thus, an $\underline{R^2}$ which approaches 1.0 suggests that the variables included in the model account for the bulk of the variance (Kerlinger, 1986).

For a variable to be declared predictive of the outcome (i.e., it accounts for a substantial amount of the variation in the dependent variable) any changes in $\underline{R^2}$ of that variable must also be significant (Lewis-Beck, 1980). The significance criterion is specified at the outset of the analysis and is determined by a comparison between the actual tabled $\underline{F}$ values and the predicted $\underline{F}$ values (Belsey, 1991; Pedhazur, 1980). In the present study the accepted .05 level of significance is used as the cut-

off point for inclusion of predictor variables in the model (Cohen & Cohen, 1983).

Multiple regression supersedes bivariate regression by allowing more than one independent variable to be included in an equation using the underlying principles of correlation and regression (Kerlinger, 1986; Lewis-Beck, 1980). With bivariate regression, the single independent variable accounts for some of the variation in the dependent variable. Unless a perfect correlation exists between the two, the error term will be present. That is, there is a difference between the regression line and the real value of Y (Groebner & Shannon, 1985). The error term has a mean value of zero and a standard deviation termed the standard error of the estimate (Groebner & Shannon, 1985). A large standard error indicates that the regression model has limited power of prediction (Groebner & Shannon, 1985). With each new independent variable included in a multiple regression model, more variance is explained and consequently, the standard error of the estimate is reduced. Hence, multiple regression offers an increased power of prediction.

In allowing the study of the multiple influences of a number of independent variables on one or more dependent variables, multiple regression provides a statistical representation of the complexity of behavioural reality (Kerlinger, 1986). According to Lewis-Beck (1980), multiple regression presents a more comprehensive explanation of the dependent variable as most phenomena are the result of multiple causes. By eliminating the possibility of other independent variables imposing a distorting influence, multiple regression also brings greater certainty to the effect of the independent variable under study (Lewis-Beck, 1980). Multiple regression constitutes a fairly precise and very powerful means of controlling variance (Kerlinger, 1986).

Methods of Multiple Regression

There are two primary components to the multiple regression routine. In the first, a correlation matrix is calculated, and in the second, the multiple regression analysis is performed (Lewis-Beck, 1980). This can take a number of different forms, each of which enters the independent variables into the equation in a different manner. A primary goal is to choose the method capable of achieving the best combination of variables (Montgomery & Peck, 1982). The choice of method is contingent on whether the decision is based on conceptual or empirical grounds (Cohen & Cohen, 1983). If sufficient information exists concerning the conceptual order or relevance of the predictor variables, then hierarchical regression is the preferred method (Cohen & Cohen, 1983). Where there is insufficient, or inconclusive information regarding the conceptual order of variables, stepwise regression offers a sound alternative (Cohen & Cohen, 1983). The two forms of multiple regression are discussed below.

Hierarchical regression.

Hierarchical regression is based on the premise that there is sufficient information concerning the relative predictive power of each independent variable to define, at the outset, the order in which they are included in the regression model (Cohen & Cohen, 1983). With hierarchical regression, the R^2 and partial coefficients pertaining to each variable are determined at the point of entry into the equation (Cohen & Cohen, 1983). The significance of each partial coefficient is also assessed at this point. Thereafter, the independent variables are included in the predetermined order, and the R^2 calculated following the addition of each conceptually plausible variable (Cohen & Cohen, 1983).

Stepwise regression.

In stepwise regression, the independent variables are added or deleted from the model on an individual basis until such time as the 'optimum' regression model is achieved (Dillon & Goldstein, 1984). Operationally, variables are included in the model in successive steps with the variable possessing the greatest coefficient of partial determination being included at each step (Montgomery & Peck, 1982). While the coefficient of determination is a measure of the proportion of variance explained by all independent variables included in the model, the coefficient of partial determination is the proportion of variance explained by a particular independent variable, given that other independent variables have been included in the model (Groebner & Shannon, 1985).

The process of stepwise regression begins with the selection of the independent variable which has the highest correlation with the dependent variable (Groebner & Shannon, 1985). The second step selects the independent variable which, when combined with the first variable selected, explains the highest proportion of the remaining unexplained variance in the dependent variable (Groebner & Shannon, 1985). This process continues until the optimum predictor equation is achieved (Montgomery & Peck, 1982). Alternatively, it is terminated once either all independent variables have been included or the remaining independent variables fail to make a significant contribution to R^2 (Groebner & Shannon, 1985). A useful feature of stepwise regression is its ability to determine whether a variable selected in an earlier step overlaps with a subsequent addition to the extent that the former is no longer significant (Groebner & Shannon, 1985). In these instances, stepwise regression eliminates the non significant variable from the model (Groebner & Shannon, 1985; Montgomery & Peck, 1982). Thus, by eliminating trivial variables, stepwise regression is able to yield the most satisfactory predictor equation (Montgomery & Peck, 1982; Rawlings, 1988).

The multidimensional model proposed in Figures 8.1 and 8.2 has not been examined by previous research. As a consequence, there is not enough information on the relative predictive power of each independent variable to define an *a priori* hierarchy of predictor variables for inclusion in the model (Cohen & Cohen, 1983). Thus, hierarchical regression is inappropriate for the purposes of the present study. Stepwise regression, on the other hand, is empirically based and does not require that the sequence of predictor variables be defined (Cohen & Cohen, 1983; Montgomery & Peck, 1982). In stepwise regression, each model variation is considered until the most satisfactory equation is found (Dillon & Goldstein, 1984). As the aim of the present study is to test empirically the multidimensional model proposed in Figures 8.1 and 8.2, forward stepwise regression is believed to be the more appropriate method of analysis for the present study.

Covariates

Spurious results are often the product of confounding demographic variables (Neale & Liebert, 1980). The impact of confounding demographics can be controlled by including such variables as covariates (Neale & Liebert, 1980). Covariates can be determined by assessing the relationship between demographic variables and independent variables. Where demographic variables are significantly related to the independent variable they are likely to generate spurious results (Neale & Liebert, 1980). To limit this possibility, the demographic variables which are significantly related to the independent variables are included in the multiple regression model as covariates. In the present study, six variables will be considered as potential covariates, namely, age, sex, race, marital status, occupation, and education. As outlined below, past research has revealed relationships between these variables and prognosis for recovery from CHD.

Age plays a role in the perception of the stressfullness of CHD and the response

thereto (Rejeski et al., 1985). Age has also been found to predict reinfarction in the three years following an initial event (Norris et al., 1970). With men showing a greater risk than women for an initial coronary event (Boogaard, 1984), CHD is designated a sex linked phenomenon (Kavanagh et al., 1977). However, the role played by sex in the aetiology of subsequent events is reversed and female CHD patients are at greater risk of reinfarction and experience poorer psychological and physiological outcomes than their male counterparts (Guiry et al., 1987; Wenger et al, 1993). In America, the incidence of CHD is higher, and the survival rates are lower, among blacks than whites (Becker et al., 1993). The relationship between race and CHD would appear to be reversed in South Africa, where the lowest incidence of CHD is seen in the black population (Wyndham, 1979). The incidence of reinfarction is greater among unmarried CHD patients (Stern et al., 1976). Higher rates of CHD related mortality and morbidity occur among blue collar CHD patients than among white collar CHD patients (Hartley et al., 1987), thus indicating that occupation is associated with prognosis. High education levels have also been found to predict long term survival among CHD patients and low education levels to predict a poor prognosis for recovery from CHD (Hlatky et al., 1980; Weinblatt et al., 1982). It is apparent that age, sex, race, marital status, occupation, and education have the potential to exert some influence on the projected outcome of the present study. Therefore, it is necessary to determine which, if any, of these variables are significantly associated with the independent variables.

The relationship between age and the independent variables will be assessed by an examination of the correlation matrix. Should the correlation be significant, age will be incorporated as a covariate. The relationship between the dichotomous demographic variables (i.e., sex, race and marital status), and the independent variables will be assessed by means of t tests. A one way analysis of variance will reveal any relationships between the independent variables and the multi-level demographic variables, occupation and education. Those demographic variables

which yield significant relationships with the independent variables will be included in the multiple regression analyses as covariates.

Dummy Variables

Multiple regression is based on quantitative or interval variables, namely, variables which have precise numerical values (Lewis-Beck, 1980). Qualitative variables have no inherent numerical value. Therefore, qualitative variables cannot be included in multiple regression in their original form. This problem is overcome by creating dummy variables for each of the qualitative variables (Groebner & Shannon, 1985). These are variables which adopt a finite number of values, each of which reflects a different category of the qualitative variable (Kleinbaum & Kupper, 1978). The creation of dummy variables involves the assignment of arbitrary numerical values to the different outcomes, wherein a value of '1' will indicate membership in a particular category and '0' non-membership to that category (Rawlings, 1988). The rule for determining the number of dummy variables is to subtract one from the number of categories of the qualitative (noninterval) variable (Lewis-Beck, 1980). Consequently, the last dummy variable is excluded and serves as a comparative reference point for the dummy variables included in the model (Kerlinger & Pedhazur, 1973). Examples of qualitative variables include sex and marital status (Groebner & Shannon, 1985). The use of dummy variables extends the range of multiple regression to produce more distinct information (Kleinbaum & Kupper, 1978). By assigning finite values to the categories contained in qualitative variables, it is possible to determine the effect these have on the outcome variables (Montgomery & Peck, 1982).

Assumptions Underlying Multiple Regression

Prior to running multiple regression, it is necessary to determine whether the data

meet the assumptions underlying multiple regression. The primary assumptions of multiple regression include minimal measurement error, the absence of multicollinearity and outliers, linearity and normality (Groebner & Shannon, 1985; Lewis-Beck, 1980).

Measurement error.

An underlying assumption of multiple regression is the absence of measurement error. This assumption is based on the maxim that inaccuracies in the method of measurement will be reflected in inaccurate estimates (Lewis-Beck, 1980). To establish whether the variables in the model have been measured accurately, and hence, that there is no measurement error, it is necessary to subject the variables to reliability tests and to confirmatory factor analysis. In the present study, the internal reliability of the Type A variables will be assessed by means of Kristof's formula (see Chapter 7) and the dependent variables, anxiety and depression, by means of Cronbach's alpha formula. The six month test-retest reliability of all independent and dependent variables will be calculated by means of a correlation matrix. Following the recommendation of Nunnally (1967), a cut-off point of .60 will be used to determine reliability.

Confirmatory factor analysis will also be used to cross validate the Type A Component Questionnaire. Cross-validation provides a powerful indication of factor stability. Successful cross-validation demonstrates an absence of measurement error (Cudeck & Brown, 1983), while the failure thereof indicates bias in the coefficient estimates and increases in random error and in the variance of the error term (Dillon & Goldstein, 1984). Thus replication across samples demonstrates that the factor structure is robust and meaningful (Welch et al., 1990) and meets the

measurement error assumption of multiple regression.

To determine the stability and generalisability of the five factor solution across samples, the data derived from the present sample's response to the 26 items will be analysed using the same principle components analyses with varimax rotation employed in Study 2. The same five methods of factor extraction will be used, namely, Kaiser's eigenvalue greater than one criterion (Kaiser, 1960), Kaiser's MSA (Kaiser, 1970), the communality estimate, component saturation, and Cattell's scree test (Cattell, 1966).

In order to assess the extent of factor stability and consequently, the absence of measurement error, two comparison statistics will be used, namely, the root mean square formula (RMS; Harman, 1976) and the coefficient of congruence (CC; Wrigley & Neuhaus, 1955). RMS reflects the degree of agreement between the factor weights of a set of fixed variables (i.e., items) over different samples (Harman, 1967). To determine RMS, the square root of the average deviations of two independent samples' factor loadings is calculated (Levine, 1977). Perfect agreement is indicated by a RMS deviation of zero, while a perfect reflection between factors yields a maximum measure of two (Levine, 1977). The CC provides an alternate means of determining the extent of factorial similarity (Harman, 1967). CC reflects the proportion of similarity between the factor loadings generated by two independent samples (Levine, 1977). The CC is sensitive to differences in magnitude and pattern between the factor structures of two samples and in contrast to the RMS, indicates perfect agreement when a measure of one is achieved (Wrigley & Neuhaus, 1955; Guadagnoli & Velicer, 1991).

Where many of the factor loadings of the two samples share the same algebraic signs, the CC will yield a high coefficient (Harman, 1967). With its ability to locate small differences in the magnitude and pattern of item loadings, the RMS is regarded as a more conservative measure (Levine, 1977). However, the CC is considered to be the more consistent of the two procedures (Levine, 1977). Both procedures require that the identical set of variables be used across samples (Harman, 1967). In the present study, a RMS which approaches zero and a CC which approaches one will indicate factor stability across samples.

While measurement error can never be eliminated in its entirety (Anastasi, 1968), the internal and test-retest reliability estimates, results of the principle component analysis, RMS and CC statistics will indicate the extent to which measurement error is present in the data.

Multicollinearity.

An absence of multicollinearity constitutes an assumption of multiple regression. High correlations (i.e., $r > .80$) between independent variables imply multicollinearity (Lewis-Beck, 1980). This condition translates to one predictor variable being almost completely redundant when included with the other predictors (Lewis-Beck, 1980; Montgomery & Peck, 1982). High correlations influence the magnitude of the regression coefficients contained in the equation and correspondingly, limit the generalisability and applicability of the model (Belsey, 1991). Specifically, where perfect multicollinearity exists, it will not be possible to achieve the desired 'best linear unbiased estimates' (Lewis-Beck, 1980).

While perfect multicollinearity is rare, high multicollinearity is common and can compromise the reliability of the least squares parameter estimates (Lewis-Beck, 1980). Compromised reliability is a result of the typically large sizes in the variances

in the slope estimates and in the standard errors which are synonymous with muticollinearity (Lewis-Beck, 1980). In the extreme, multicollinearity can render estimated regression coefficients so unstable as to be non significant in instances where the variables are, in reality, associated (Lewis-Beck, 1980). The existence of multicollinearity can be determined by an examination of the correlation matrix of independent variables. This method is useful for determining linear dependency between two independent variables (Montgomery & Peck, 1982). Multicollinearity is said to exist if the correlations between the independent variables exceed .80 (Lewis-Beck, 1980). In the present study, multicollinearity will be assessed by means of this method.

When the standard least square method cannot achieve stable estimates of the regression coefficients due to high intercorrelations between independent variables, ridge regression can be used to eliminate the problem of multicollinearity (Montgomery & Peck, 1982). This procedure entails the addition of a constant (lambda) to the diagonal of the correlation matrix and the subsequent re-standardisation which returns the diagonal elements to 1.0 and divides the off-diagonal elements by the constant. In essence, ridge regression allows the more stable estimates or beta coefficients to be computed by decreasing the correlation coefficients. A simpler alternative involves the deletion of the relevant variable. As the offending variable is essentially a combination of other variables, little information is lost by exercising this option (Tabachnick & Fidell, 1983). Should correlations between independent variables exceed the .80 cut off in the present study, the relevant variable will be eliminated from further analysis.

Outliers.

An assumption underlying multiple regression is that there is an absence of outliers, namely, cases which do not conform to the overall pattern of the data (Belsey, Kuh,

& Welsh, 1980). The presence of outliers suggests that the relationship between the independent and dependent variables is non-linear and / or there is an absence of constancy concerning the variance (Chatterjee & Price, 1977). Outliers can compromise the results by altering the direction of the regression line and consequently, generating biased regression coefficients. Cook's D statistic (Rawlings, 1988) is a test of discordancy of outliers which offers an acceptable means of detecting the presence of outlying cases and will be used in the present study. Cases will be considered outliers where the Cook's D value generated for the residual of each case exceeds the value determined by the following equation:

$$\text{Cook's } D = 4 / n$$

where n = sample size

(Rawlings, 1988, p. 269)

Outlier cases influence both their own residuals and the residuals of other cases (Barnett & Lewis, 1978). This has a carry over effect whereby the more extreme outliers disguise the less extreme outliers. As a consequence, elimination of the initial set of outliers on the basis of a test of discordancy will reveal previously concealed outlying observations and this process may continue to the point where it is no longer possible to draw inferences about the population under study (Barnett & Lewis, 1978). As there is no prescribed point at which to terminate the elimination of observations which exceed the Cook's D statistic, subjective judgement is required (Chatterjee & Price, 1977; Barnett & Lewis, 1978). In the case of multiple regression models, tests of discordancy of outliers yield the most extreme outlying observations in the initial assessment (Barnett & Lewis, 1978). While less extreme outliers are likely to be revealed by subsequent tests of discordancy, they are unlikely to compromise the general linear model of the data (Barnett & Lewis, 1978). Therefore, in the present study, only the first set of cases classified as outliers according to Cook's D statistic will be eliminated from further analysis (Montgomery & Peck, 1982).

Linearity.

A further assumption of multiple regression is the linearity of data (or absence of specification error) (Lewis-Beck, 1980). It dictates that the theoretical model underlying the equation is correct, that the relationship between independent and dependent variables is depicted by a straight line, and that the relevant variables have been included, and the irrelevant excluded (Lewis-Beck, 1980; Tabachnick & Fidell, 1983). While it is almost impossible to confirm linearity, it can be assessed by computing residual plots of the independent and dependent variables (Chatterjee & Price, 1977). Residuals constitute the difference between the predicted and the actual value of the data and consequently, the variation not accounted for by the regression model (Montgomery & Peck, 1982). Visual examination of the residual plots will reveal any deviations from linearity (Pedhazur, 1980). If the data are linear, the plots will reveal a straight, and not a curved line. Minor deviations from linearity do not compromise multiple regression (Barnett & Lewis, 1978). Linearity will be determined in the present study by a visual examination of the residual plots (Pedhazur, 1980). The data will be considered linear where the plots show an absence of curvature (Pedhazur, 1980).

Normality.

An assumption underlying multiple regression is that errors follow a normal, and not a skewed or flat tailed, distribution (Lewis-Beck, 1980). That is, that the errors between the predicted and actual values are distributed normally with 95% of the observations falling within approximately two standard deviations of a mean of zero (Groebner & Shannon, 1985; Lewis-Beck, 1980). Normality can be determined by a visual inspection of the normal probability plot of the predicted values of the ordered observations and of the actual ordered residual values (Tabachnick & Fidell, 1983). For the assumption of normality to be satisfied, the scatterplot should be manifested

by a relatively straight line which passes through zero (Rawlings, 1988). The angle of the line's slope is established by the standard deviation of the residuals (Rawlings, 1988). Violation of the normality assumption is evidenced by an irregular distribution (e.g., an S-shaped curve) and requires the transformation of the data (Tabachnick & Fidell, 1983). However, where the data derives from a large sample, violation of the normality assumption will not compromise the results to a significant degree (Lewis-Beck, 1980). Normal probability plots will be generated in the present study to assess the normality of the data.

Results

Outlined below is the process used to identify which demographic variables should be included in the multiple regression analysis as covariates. This is followed by a discussion of the assumption test results and the results of the multiple regression analysis.

Covariates

The six demographic variables, namely, age, occupation, marital status, sex, race and education, were considered as potential covariates. Various tests were conducted to identify those demographic variables which are significantly related to the independent variables and consequently, are likely to generate spurious results if not included as covariates in the regression analysis. Specifically, the potential significance of age was assessed by means of a correlation matrix, sex, race, and marital status by means of t-tests and occupation and education by a one way analysis of variance.

Results of the correlation can be seen in Table 8.3. Age correlated significantly with impatience irritability, acting-out hostility, anger-in and anger-out and the

frequency and duration measures of exercise compliance. Consequently, age was determined to be a covariate. The t tests revealed no significant differences between the dichotomous variables, race, sex and marital status on the seven independent variables (see Table 8.4). The one way analysis of variance showed occupation to be significantly related to acting-out hostility and the frequency and duration of exercise compliance (see Table 8.5) and was included in the multiple regression analysis as a covariate. Education was not found to relate significantly to any of the independent variables. Thus, age and occupation were included in the multiple regression analysis as covariates, following the creation of dummy variables for occupation.

Table 8.3

Correlation Matrix of Age and the Independent and Dependent Variables (N=181)

		1	2	3	4	5	6	7	8	9	10	11
1	Age											
2	Achievement Striving	.4										
3	Impatience Irritability	.18*	.26**									
4	Anger-in	-.16*	-.07	-.20**								
5	Anger-out	-.19*	-.33**	-.27**	-.11							
6	Acting-out Hostility+	.28**	.23**	.21**	-.26**	-.20**						
7	Attendance	.29**	.15*	.11	-.13	-.17*	.20**					
8	Duration	.26**	.10	.03	-.13	-.12	.13	.37**				
9	Change in Anxiety	.04	.08	.15	-.03	-.07	.10	.09	-.03			
10	Change in Depression	.12	.14	.16*	-.14	-.18*	.15	.21**	.13	.35**		
11	Change in VO_2max	-.32**	.01	-.08	-.03	.04	-.03	-.03	.05	.08	.03	

* $p < .05$
** $p < .01$
\+ The Acting-out Hostility scale is negatively worded.

Table 8.4

The t Tests of the Demographic Variables, Race, Sex, Marital Status and the Independent Variables

Independent Variables	Levels	Achievement Striving			Impatience Irritability			Anger-in			Anger-out		
		n	M	t	n	M	t	n	M	t	n	M	t
Sex	Male	161	8.72	0.80	161	4.88	1.12	161	11.29	0.76	160	7.11	0.40
	Female	18	9.22		18	5.28		18	10.72		18	6.72	
Race	White	170	8.77	0.14	170	4.89	1.13	170	11.25	0.35	170	6.96	1.43
	Black	9	8.89		9	5.44		9	10.89		8	9.00	
Marital status	Married	147	8.72	0.57	147	4.93	0.20	147	11.18	0.50	148	6.89	1.20
	Unmarried	32	9.00		32	4.88		32	11.47		30	8.00	

Independent Variables	Levels	Acting-out Hostility			Attendance			Duration		
		n	M	t	n	M	t	n	M	t
Sex	Male	161	7.63	1.27	163	53.40	0.35	163	1887.82	0.50
	Female	18	7.78		18	54.39		18	1957.33	
Race	White	170	7.64	0.55	172	53.70	1.08	172	1890.17	0.48
	Black	9	7.78		9	49.56		9	1982.00	
Marital status	Married	147	7.64	0.11	149	53.65	0.40	149	1910.76	0.83
	Unmarried	32	7.66		32	52.78		32	1820.13	

** $p < .01$

Table 8.5

Summary of the Analyses of Variance for the Demographic Variables Education and Occupation and the Independent Variables

	Achievement Striving		Impatience Irritability		Anger-in		Anger-out	
Demographic variables	F	df	F	df	F	df	F	df
Education	0.88	4/178	0.29	4/178	1.24	4/178	1.05	4/177
Occupation	1.17	3/178	1.74	3/178	2.47	3/178	0.21	3/177

	Acting-out Hostility		Attendance		Duration	
Demographic variables	F	df	F	df	F	df
Education	1.40	4/178	1.39	4/180	0.39	4/180
Occupation	2.62*	3/178	5.04**	3/180	5.07**	3/180

** $p < .01$
* $p < .05$

Assessment of the Assumptions Underlying Multiple Regression

Measurement error.

As a first step towards establishing an absence of measurement error, the internal reliability of the Type A subscales was assessed using Kristof's reliability formula, and the anxiety and depression scales using the standardised Cronbach's alpha formula (Cronbach, 1951). As can be seen from Table 8.2, all scales yielded coefficients in excess of the .60 cut-off point suggested by Nunnally (1967). Test - retest reliability was calculated for a six month period. All test-retest coefficients were significant ($p < .01$; see Table 8.2) thus suggesting satisfactory test-retest reliability. As the measure of VO_2max represents scores unique to each subject, the internal reliability of this measure could not be assessed. The present study focuses on the first six months of each patient's exercise programme. Therefore, the Time 1 measures of exercise frequency and duration are zero while the Time 2 measures reflect the incremental sum of exercise performance which occurred in the preceding six months. Existing tests for internal reliability do not accommodate measures which are incremental in design (Anastasi, 1968). Moreover, assessment of test-retest reliability requires the repeated administration of an identical test (Anastasi, 1968). Given the nature of the exercise frequency and duration measures, it is not possible to assess their internal and test-retest reliability.

The second step toward establishing an absence of measurement error involved the cross-validation of the Type A Component Questionnaire by means of principle components analysis with varimax rotation. The five methods of factor extraction employed in Study 2 were applied to the present data. These included Kaiser's eigenvalue greater than one criterion, Kaiser's (1970) MSA criterion, the communality estimate, component saturation, and Cattell's scree test. Results of the analysis showed that all five factors met Kaiser's eigenvalue greater than one

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criterion (see Table 8.6). Further, all items met the minimal .50 acceptability level of Kaiser's (1970) MSA criterion. One item (i.e., "I get angry easily and then get over it soon") failed to generate a communality estimate in excess of .20 and was eliminated from the questionnaire in the present study. A further item (i.e., "Sometimes I enjoy hurting persons I love") was eliminated for failing to load on any one of the five factors. Thus, subjecting the data to confirmatory factor analysis refined the Type A Component Questionnaire further and resulted in final factor solution comprising 24 items.

As can be seen from Table 8.6, the reduced questionnaire yielded a satisfactory overall MSA of .76 and individual MSA's in excess of the .50 acceptability level (Kaiser & Rice, 1974). Thus, support was found for the appropriateness of the common factor model. All 24 items generated communality estimates in excess of the necessary .20 cut-off (Cureton & D'Agistino, 1983) and factor loadings greater than the prescribed .40 level of component saturation (Guadagnoli & Velicer, 1988). Multiple loadings were generated for one item (i.e., "I boil inside, but I don't show it"). Given that this item loaded negatively on the anger-out factor and positively on the anger-in factor, the multiple loading is conceptually consistent. This item was included in both the anger-in and anger-out factors. However, in constructing the anger-out variable, this item was included as a negative item, while in the construction of the anger-in variable, it was included as a positive item. A further item (i.e., "I keep things in") loaded negatively on the anger-out factor and was similarly included as a negative item in the construction of the anger-out variable. Cattell's' scree test, the fifth and final method of factor extraction, generated a scree plot which was supportive of the five factor solution.

Table 8.6

Varimax Rotated Factor Loadings on Five Factors. (N=181)

	Scale Items	Factor Loadings 1	2	3	4	5	h^2	MSA
1	I express my anger.	.77	.01	.03	-.20	-.11	.55	.83
2	I argue with others.	.58	.14	-.09	-.11	-.17	.40	.87
3	I strike out at whatever infuriates me.	.41	.06	-.14	.09	-.14	.21	.69
4	I say nasty things.	.56	.32	-.13	.04	-.04	.44	.79
5	I lose my temper.	.66	.25	-.27	-.12	-.07	.58	.85
6	If someone annoys me, I am apt to tell him or her how I feel.	.63	-.08	-.02	-.04	-.01	.41	.83
7	I keep things in.	-.65	.31	-.19	.10	.09	.56	.76
8	I boil inside, but I don't show it.	-.59	.41	-.15	.01	-.07	.54	.75
9	I pout or sulk.	.29	.63	-.11	.12	.06	.51	.80
10	I withdraw from people.	.04	.60	.09	-.24	.01	.43	.71
11	I tend to harbor grudges that I don't tell anyone about.	.06	.64	-.14	.11	-.06	.45	.74
12	I am angrier than I am willing to admit.	-.07	.75	-.01	-.12	-.12	.59	.80
13	I am irritated a great deal more than people are aware of.	.02	.70	-.10	-.05	-.17	.53	.79
14	I can easily make other people afraid of me, and sometimes do for the fun of it.	-.07	.01	.70	.17	.08	.52	.71
15	At times I have a strong urge to do something harmful or shocking.	-.03	-.14	.67	-.05	.03	.48	.69
16	At times I feel like smashing things.	-.16	-.34	.50	-.03	.07	.40	.78
17	At times I feel like picking a fist fight with someone.	.01	.06	.76	.14	.01	.59	.63
18	When you were younger, did most people consider you to be (Definitely hard-driving and competitive? ... Definitely relaxed and easy going?)	-.03	.10	.03	.83	.01	.70	.55
19	How would your spouse (or closest friend rate you currently? (Definitely hard-driving and competitive? ... Definitely relaxed and easy going?)	-.16	-.04	.10	.72	.23	.61	.73
20	How was your temper when you were younger?	-.36	-.18	.03	.51	-.09	.43	.73
21	I approach life in general (Much more seriously ... Much less seriously).	-.06	-.06	.07	.47	.07	.24	.72
22	When you listen to someone talking, and this person takes too long to come to the point, how often do you feel like hurrying the person along?	-.02	-.06	.03	.10	.84	.72	.68
23	How often do you actually "put words in the person's mouth" in order to speed things up?	-.15	-.09	-.03	.07	.75	.59	.76
24	I easily become impatient with people.	-.27	-.15	.24	.08	.64	.56	.73
	Eigenvalues	4.5	3.0	1.7	1.5	1.4		
	Percentage of total variance accounted for	19%	12%	7%	7%	6%		
	Over-all MSA							.76

Note. Underlined figures represent items which loaded in excess of .40. The column in which items are underlined reflects the factor on which each item loaded.

The various methods of factor extraction confirmed the five factor solution and indeed, yielded a similar item composition to that which was obtained for the

original sample. To assess the extent of this similarity, the factor structures derived from the two samples were subjected to the RMS and CC procedures. As both methods require that the two factor structures contain an identical number of variables, the items eliminated in the second sample were also eliminated in the first sample (Harman, 1967). Therefore, the 24 item Type A Component Questionnaire was used in the assessment of RMS and CC and all subsequent statistical analysis.

Table 8.7 shows that the levels of fit between the two samples of cardiac rehabilitation patients were acceptable for both the RMS and CC measures (RMS: M rating = .12, range = .07 - .22) (CC: M rating = .97, range = .91 - .99). Based on the results of these comparison statistics, the five factor solution can be deemed consistent across samples and thus, to possess external validity. Further, when taken together with the confirmatory factor analysis, these results demonstrate that the present data satisfies the assumption regarding an absence of measurement error.

Table 8.7
Factor Comparison for the Two Samples of Cardiac Rehabilitation Patients

	Comparison Statistic	
Factor	Coefficient of Congruence	Root Mean Square
Anger - In	.99	.11
Anger - Out	.99	.10
Acting-Out Hostility	.91	.22
Impatience Irritability	.99	.07
Achievement Striving	.99	.09

Multicollinearity.

A correlation matrix (see Table 8.3) was used to determine whether the data met the assumption of an absence of multicollinearity. An inspection of the correlation

matrix revealed that all correlations between independent variables were below the .80 cut-off level prescribed by Lewis-Beck (1980). Therefore, the assumption of multicollinearity was met.

Outliers.

Cook's D statistic (Rawlings, 1988) was used to detect the presence of outliers. Calculation of the Cook's D equation (i.e., 4/181) identified the cut off point for outliers to be .02. A number of cases generated Cook's D values in excess of the .02 cut off and were eliminated from subsequent analysis. Based on the argument that the less extreme outliers revealed by further application of the Cook's D statistic would not compromise the general linear model of the data (Barnett & Lewis, 1978), the process of outlier elimination was terminated after one iteration. As discussed below, the reduced data set revealed linear relationships between the independent and dependent variables. Thus, it is apparent that elimination of outliers on the basis of the Cook's D equation increased the linear fit of the model to the point where the assumption of an absence of outliers was met.

Linearity and normality.

Residual plots were generated to assess the multiple regression assumption of linearity (see Appendix D). It should be noted that, because the acting-out hostility subscale comprised items of different response lengths, the subscale was standardised. To standardise the subscale the distribution of the scores was set to have a mean of zero and a standard deviation of one. The standardised subscale generated a skewed distribution. The distribution of a scale does not effect the measure of its linearity (Cook & Weisberg, 1982). Indeed, a visual examination of the residual plots revealed linear relationships between the independent and

dependent variables. The normal probability plots (see Appendix E) revealed relatively straight lines for all variables (Lewis-Beck, 1980). Thus, the assumption of normality was met.

Results of the Stepwise Regression Analyses

Change in Anxiety

During the six month period under review, the present sample experienced a reduction in anxiety (see Table 8.2). After controlling for the covariates age and occupation (R^2 = .01), stepwise multiple regression was used to determine how much of the variance in change in anxiety was accounted for by the joint effects of achievement striving, impatience irritability, acting-out hostility, anger-in and anger-out and the frequency and duration of exercise. Results of the stepwise regression are shown in Table 8.8.

Anger-out was significantly and negatively associated with change in anxiety ($F(1/128) = 6.27$, $p < .05$; Beta = -.387), accounting for 4% of the variance. A further 3% of the variance in change in anxiety was accounted for by the duration of exercise ($F(1/128) = 4.23$, $p < .05$; Beta = -.002). The relationship between duration of exercise and change in anxiety was also negative. No support was found for the prediction that impatience irritability, acting-out hostility, anger-in and the frequency of exercise would contribute significantly to change in anxiety. However, as predicted, achievement striving did not yield a significant contribution to the variance in change in anxiety.

Table 8.8
Stepwise Regression for Change in Anxiety (N= 134)[a]

Variable entering equation	Partial R^2	Model R^2	Beta	F- Statistic
Covariates:				
Age			-0.007	0.01
Occupation 1[b]			-1.905	1.10
Occupation 2			-0.614	0.12
Occupation 3	0.01	0.01	-1.048	0.30
Independent variables:				
Anger-out	0.04	0.05	-0.387	6.27**
Exercise Duration	0.03	0.08	-0.002	4.23*

** $p < .01$
* $p < .05$

a The removal of outliers, together with a listwise deletion of cases resulted in the reduction of the sample size from 181 to 134.

b Dummy variables were created for the categorical variable occupation.

Note. The independent variables achievement striving, impatience irritability, acting-out hostility, anger-in and exercise frequency did not contribute significantly to the variance in change in anxiety.

Change in Depression

During the six month period, the depression levels of the present sample decreased (see Table 8.2). Controlling for age and occupation, change in depression was regressed on achievement striving, impatience irritability, acting-out hostility, anger-in, anger-out and the frequency and duration of exercise (see Table 8.9). The covariates accounted for 10% of the variance. Anger-out was negatively related to change in depression ($F(1/131) = 8.64$, $p < .01$; Beta = -.185) and accounted for 4% of the variance. Anger-in was also negatively related to change in depression ($F(1/131) = 7.05$, $p < .01$; Beta = -.209) and accounted for a further 4% of the variance. Contrary to expectations, impatience irritability, acting-out hostility and exercise frequency and duration did not contribute significantly to change in

depression. As expected, achievement striving did not contribute to the variance in change in depression.

Table 8.9
Stepwise Regression for Change in Depression (N= 138)[a]

Variable entering equation	Partial R^2	Model R^2	Beta	F- Statistic
Covariates:				
Age			0.036	1.96
Occupation 1[b]			-0.912	1.35
Occupation 2			-0.603	0.59
Occupation 3	0.10	0.10	-0.172	0.04
Independent variables:				
Anger-out	0.04	0.14	-0.185	8.64**
Anger-in	0.04	0.18	-0.209	7.05**

** $p < .01$

a The removal of outliers, together with a listwise deletion of cases resulted in the reduction of the sample size from 181 to 138.

b Dummy variables were created for the categorical variable occupation.

Note. The independent variables achievement striving, impatience irritability, acting-out hostility and exercise frequency and duration did not contribute significantly to the variance in change in depression.

Change in VO_2max

As can be seen from Table 8.2, the present sample's VO_2max levels increased during the six month period. A regression was performed between the increase in VO_2max and achievement striving, impatience irritability, acting-out hostility, anger-in, anger-out, and the frequency and duration of exercise, after controlling for age and occupation (see Table 8.10). The covariates accounted for 5% of the variance. Contrary to predictions, none of the five Type A components contributed significantly to the variance in change in VO_2max. As predicted, exercise duration was significantly and positively associated with change in VO_2max ($F(1/130)$ =

4.70, $p < .05$; Beta = 8.7×10^{-4}), and accounted for 3% of the variance. Exercise frequency did not contribute significantly to the variance in change in VO_2max.

Table 8.10
Stepwise Regression for Change in VO_2max (N= 136)[a]

Variable entering equation	Partial R^2	Model R^2	Beta	F- Statistic
Covariates:				
Age			- 0.055	5.56*
Occupation 1[b]			1.221	2.42
Occupation 2			1.445	3.27
Occupation 3	0.05	0.05	1.038	1.52
Independent variables:				
Exercise duration	0.03	0.08	8.7×10^{-4}	4.70*

* $p < .05$

a The removal of outliers, together with a listwise deletion of cases resulted in the reduction of the sample size from 181 to 136.

b Dummy variables were created for the categorical variable occupation. The number of dummy variables is determined by subtracting one from the original number of categories defining the categorical variable.

Note. The independent variables achievement striving, impatience irritability, anger-in, anger-out, acting-out hostility and exercise duration did not contribute significantly to the variance in change in VO_2max.

Discussion

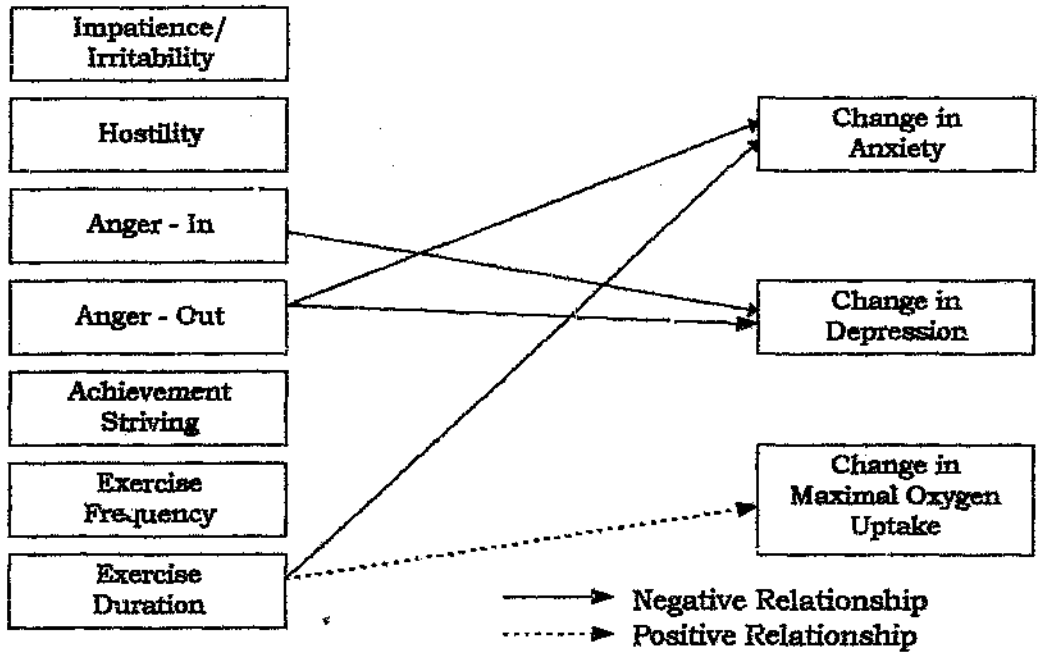
The aim of the present study was to develop and test a multidimensional model of Type A and exercise predictors of CHD patients' psychological and physiological health. To this end, a theoretically based model of CHD patients' Type A behaviour was proposed in Chapter 4. The development and empirical assessment of the theoretical model in Chapter 7 yielded a measure appropriate for use on CHD patients, namely, the Type A Component Questionnaire. Using a further sample of CHD patients, the present study assessed the relationship between the Type A Component Questionnaire and the measures of exercise compliance argued for in

Chapter 3 on the one hand and changes in anxiety, depression and maximal oxygen uptake on the other (see Chapter 2).

In the period under review, the present sample experienced the expected reduction in anxiety and depression and increase in maximal oxygen uptake (see Table 8.2), indicated by past research (e.g., Brown & Munford, 1984; De Geus et al., 1993; Erb et al., 1979; Kavanagh et al., 1977; Roth, 1990). Thus, the relationships between the Type A and exercise components and changes in the outcome variables should be interpreted in terms of the direction of the changes which occurred in anxiety, depression and maximal oxygen uptake. The results of the present study are discussed in relation to extant theory and past research. Following the interpretation of results is a discussion of the theoretical and practical implications of these results and the limitations of the study.

From the summary of results depicted in Figure 8.3 it is evident that Type A and exercise compliance components are differentially associated with CHD patients' changes in anxiety, depression and maximal oxygen uptake. Specifically, high levels of anger-out and a longer duration of exercise predict a smaller reduction in anxiety. Conversely, achievement striving, impatience irritability, hostility, anger-out and the frequency of exercise do not predict changes in anxiety. An increase in anger-in and anger-out predicts a decrease in the reduction of depression but achievement striving, impatience irritability, hostility and the frequency and duration of exercise are unassociated with a change in depression. While none of the five components of Type A behaviour is associated with maximal oxygen uptake, increases in the duration, but not the frequency, of exercise predict improved maximal oxygen uptake. These results highlight the inadequacy of using global measures of Type A behaviour and unidimensional measures of exercise compliance to predict health outcomes of CHD patients. The results of the present study are discussed in more detail below.

Figure 8.3
Summary of Findings of the Relationships Between Type A and Exercise Compliance Components and Changes in Psychological and Physiological Health.



Change in Anxiety

The results of the present study support the hypothesis that Type A components are differentially related to psychological health. Specifically, they show that high levels of anger-out are negatively associated with a reduction in anxiety. This implies that high levels of anger-out predict a smaller reduction in anxiety. The present results also support the hypothesis that achievement striving is unrelated to anxiety. However, the present results failed to support the hypothesis that impatience irritability, hostility and anger-in would predict smaller reductions in anxiety. In the present study, the duration of exercise was also negatively associated with a reduction in anxiety, while the frequency of exercise was unassociated with anxiety.

It was hypothesised that increases in anger-out would predict a change in anxiety. A spontaneous reduction in anxiety is expected to occur in the majority of CHD patients in the period of recovery (Brown & Munford, 1984; Roth et al., 1990).

Relationships between anger-out and anxiety have been reported in past research (e.g., Edwards & Baglioni, 1991; Spielberger et al., 1985). Therefore, it was expected that anger-out would lessen the anticipated reduction in anxiety. The present sample did, indeed, experience a reduction in anxiety over time. The negative relationship between anger-out and reduction in anxiety found in the present study suggests that high levels of anger-out limited this reduction in anxiety. Thus, the hypothesis linking anger-out to a change in anxiety was supported.

The expression of anger functions as a means of exerting control over factors associated with goal achievement (Glass & Carver, 1980). The present result may then be attributed to Type A's propensity to express anger at anyone or anything which threatens to impede the desired high rate of achievement (Hassmén et al., 1993). The perpetual struggle to maintain control increases Type A's anxiety (Ray & Simons, 1982). The period of recovery from CHD is characterised by numerous physical and psychological impediments to achievement (Erdman, 1990). Type A CHD patients are known to express considerable anger in response to these impediments (Budnick, 1991). While CHD patients can expect to feel less anxiety as time passes (Brown & Munford, 1984), the persistent expression of anger is likely to limit this reduction in anxiety.

A review of the literature failed to yield any research which has examined the impact of anger-out on CHD patients' reduction in anxiety. However, partial empirical support for the present result is provided by Edwards and Baglioni (1991) and Spielberger et al. (1985) who report that anger-out predicted anxiety in samples free of CHD. Thus, from the results of the present and past research it is possible to conclude that anger-out exerts a negative effect on the anxiety levels of both healthy individuals and individuals with CHD.

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The present result may be explained further by the relationship between poor self-esteem and anger-out and increased anxiety (Kernis et al., 1989; Price, 1982). Anger expression is often a response to th[illegible] to self-esteem (Kernis et al., 1989). The sense of physical vulnerability, coupled with the inability to regain the level of achievement that they were able to maintain prior to the disease challenges CHD patients' self-esteem (Budnick, 1991). Those who have an unstable self-esteem typically attempt to protect themselves from such challenges by expressing anger and denying the legitimacy of assaults on their self-esteem (Fishman Alschuler & Alschuler, 1984; Kernis et al., 1989). Despite such attempts, these individuals are often left with the perception that they are unable to manage self-esteem threats which results in high levels of anxiety (Bandura, 1991; Martocchio, 1994). It may be that the present sample's perceived failure to stabilise esteem in the months of recovery generated high levels of anger-out, which in turn limited their reduction in anxiety. However, as self-esteem was not assessed in the present study, it remains for future research to explore the potential relationship between self-esteem, anger-out and anxiety.

Contrary to expectations, exercise duration was negatively associated with change in anxiety in the present study. This suggests that the more time CHD patients spend exercising, the less will be their reduction in anxiety. The ability to increase the duration of their exercise is thought to enhance CHD patients' sense of mastery (De Geus et al., 1993), their tolerance of the somatic symptoms of anxiety (Hughes, 1984), and by association, the symptoms of CHD (Weg, 1985). It is also thought to reduce the fear of reinfarction (Soloff, 1978b). The benefits derived from increased exercise duration are likely to contribute to the reduction in anxiety typically seen in the months following CHD diagnosis (Brown & Munford, 1984).

Past research into the relationship between exercise compliance and anxiety is equivocal. A reduction in CHD patients' anxiety following participation in an exercise

programme has been demonstrated in a number of studies (e.g., Erdman et al., 1986; Folkins, 1976; Shepard et al., 1985; Stern et al., 1983). Other studies (e.g., De Geus et al., 1993; Stern & Cleary, 1981; Van Dixhoorn et al., 1983) have revealed an increase in anxiety following a period of exercise compliance. One explanation which has been offered for the increase in anxiety in these studies is that exercise training increases both psychological and physical mobilisation which results in increased tension levels among anxious individuals (Stern & Cleary, 1981). Despite these equivocal findings, a number of researchers (e.g., Brown & Munford, 1984; Kavanagh et al., 1977; Roth et al., 1990; Stern et al., 1976) argue that CHD patients experience a spontaneous recovery from feelings of anxiety within the six months following a cardiac event. The negative relationship between the duration of exercise and change in anxiety found in the present study implies that exercise duration limits this reduction in anxiety. An explanation for the present result lies in CHD patients' persistent fear of death.

Like exercise, anxiety is accompanied by somatic symptoms such as palpitations, breathlessness and fatigue (Ledwidge, 1980). These symptoms are also signs of cardiovascular distress (Weg, 1985). Learning to associate these symptoms with exercise duration rather than cardiovascular distress should reduce patients' fear of suffering a heart attack, and consequently, their anxiety. However, patients' fear may be so unwavering that they interpret the symptoms produced by exercise as signs of cardiovascular distress. Perceived physical danger is associated with anxiety (Mathews, 1990). This situation will be exacerbated if patients perceive their disease to be more severe than their medical advisors have determined it to be (Rejeski et al., 1985). If patients mistrust the accuracy of their diagnoses, then compliance with the exercise prescriptions based on these diagnoses is likely to generate a fear that exercising for the required duration will result in a heart attack. It is possible that the present sample associated the symptoms of increased exercise duration with the symptoms of CHD. Therefore, the fear that the duration

component of their prescriptions was beyond their physical capabilities, and had potential dire consequences, may have retarded the present sample's reduction in anxiety.

Indirect support for this contention is provided by several studies (e.g., Cay et al., 1973; Singh et al., 1970; Stern et al., 1976) which revealed that a substantial number of CHD patients continue to demonstrate anxiety for as long as two years after diagnosis. In each of these studies, CHD patients attributed their anxiety to the fear that physical exercise would either exacerbate their physiological condition or result in a heart attack (Cay et al., 1973; Singh et al., 1970; Stern et al., 1976).

The argument that the duration component of exercise generated fear becomes plausible in the light of a comparison between the prescribed and actual duration of exercise performed by the present sample. The present sample were expected to exercise for 30 to 45 minutes, three times a week depending on the results of their stress tests. This translates to 36 to 54 hours for the six month period. As can be seen from Table 8.2, the average amount of time the present sample spent exercising over the six month period was just over 31 hours. It is possible that patients' fear regarding the consequences of complying with the duration component contributed to their habit of exercising for less time than was prescribed.

The comparison between the prescribed and actual exercise duration provides support for the aforementioned argument that patients' fear associated with exercise duration limited their reduction in anxiety. The comparison also highlights the possibility that the present sample invested insufficient time in the exercise duration component for it to exert a positive effect on anxiety. Without exercising for the prescribed amount of time, the present sample may not have reached the level of fitness associated with a reduction in anxiety (Cupelli et al., 1984; Oberman, 1983).

If the CHD patients in the present sample evaluated their persistent failure to meet the prescribed exercise duration goals negatively, they are likely to have experienced less of a reduction in anxiety. While failure to achieve motivates some individuals to expend greater effort, it results in a syndrome of frequent failure for others (Ward & Eisler, 1987). For these individuals, frequent failure results in a dysfunctional self-evaluation system. Individuals who are prone to evaluate themselves negatively, are quick to devalue their actual performance on the grounds that it failed to meet prescribed standards, regardless of whether such performance was adequate or not (Ward & Eisler, 1987). Frequent failure to achieve performance goals and the devaluation of actual performance results in a global sense of personal incompetence, and ultimately, anxiety (Price, 1982; Schwartz et al., 1986). It is feasible that, because the duration component inspired fear among the present sample, they consistently failed to exercise for the prescribed amount of time. The fear associated with the duration of exercise would have diminished the reduction in anxiety. Correspondingly, the failure to exercise for the amount of time necessary to achieve the fitness level associated with improvements in psychological condition would have further compromised the reduction in anxiety. So too would the sense of persistent failure have limited the reduction in anxiety. Thus, while the negative relationship between exercise duration and change in anxiety is contrary to the present hypothesis, it is conceptually defensible.

As predicted, achievement striving was not associated with change in anxiety. This finding supports the belief that achievement striving is associated with performance and not measures of health. Type A's high in achievement striving demonstrate a preference for tasks which are difficult and require effort (Feather & Volkmer, 1991). If the effort and difficulty inherent to the process of rehabilitation and recovery is compatible with the preferences of individuals high in achievement striving it follows that they are likely to regard the process as a performance challenge rather than a source of anxiety. Should this be the case, then achievement striving would not be

associated with anxiety. The failure for achievement striving to predict change in anxiety in the present study is consistent with past research (e.g., Edwards & Baglioni, 1991). It is also consistent with research demonstrating that achievement striving is unrelated to measures of health (e.g., Barling & Charbonneau, 1992).

The hypothesised relationship between impatience irritability and anxiety was not demonstrated in the present study. This result is inconsistent with past research which has revealed relationships between impatience irritability and anxiety (e.g., Edwards & Baglioni, 1991; Edwards et al., 1990b). This result is also surprising given the conceptual link between impatience irritability and anxiety outlined below. Type A's positive perception of self is governed by their ability to maintain a high level of acquisitions and achievements (Furnham & Linfoot, 1987; Lee, 1992). Consequently, Type A's demonstrate impatience irritability when their attempts to maintain esteem through acquisitions and achievements are frustrated (Furnham, 1990b; Perry et al., 1990; Price, 1982). Type A's impatience irritability can be seen as a response to their fear of losing the achievements which they believe define them as worthwhile individuals. A primary antecedent of anxiety is the fear of losing something which is vital to the maintenance of esteem (Drever, 1958). Moreover, the need for a rapid rate of achievement generates the tension, restlessness and frenetic behaviour which characterise anxiety (Cattel & Scheier, 1976; Schomer, 1985; Yuen & Kuiper, 1992). As recovery from CHD is slow and requires steady effort over an indefinite period of time (Feather & Volkmer, 1991; Fletcher et al., 1992), it was proposed that, by its very nature, the process of rehabilitation would frustrate Type A's need for rapid achievement, and consequently result in impatience irritability. Further, CHD patients' increasing impatience irritability with the inability to maintain self-esteem would predict a change in anxiety.

Becker and Suls (1982) provide a possible explanation for the absence of a relationship between impatience irritability and change in anxiety found in the

present study. They suggest that, in certain situations, the achievement striving component may override the impatience irritability component. In these situations, the Type A devotes more time and energy than usual to a given activity (Becker & Suls, 1982). Type A's seek out personal challenges which carry the potential for achievement (Bryne & Rosenman, 1986; Ganster et al., 1991). However, Type A's high in impatience irritability also demonstrate anxiety when such challenges require slow, but considerable, effort (Feather & Volkmer, 1991). If successful adaptation to CHD is perceived as a worthwhile achievement, and it is recognised that the accomplishment thereof requires steady and time consuming effort, Type A CHD patients may overcome their impatience irritability with the slow nature of the process. In terms of this possibility, the achievement striving component reported to be unassociated with anxiety, rather than the impatience irritability component, will dominate Type A CHD patients' behaviour. Hence, the absence of a relationship between impatience irritability and change in anxiety.

While Becker and Suls' (1982) suggestion provides a possible explanation for the present result, it is highly speculative. As a literature search failed to identify any past research which has examined Type A CHD patients' achievement striving and impatience irritability in relation to a change in anxiety, the relative roles of the two components and the interaction between them will need to be established in future research.

Contrary to predictions, hostility was also not associated with change in anxiety in the present study. Hostility is characterised by hyper-vigilance and insecurity. The belief that other people are set on undermining their control causes hostile individuals to be hyper-vigilant (Hart, 1984) and results in repeated acts of aggression toward objects and other people (Houston & Vavak, 1991). Both hyper-vigilance and a combative mode of response are associated with increased anxiety (Matthews, 1990; Ray & Simons, 1982). Hostile individuals' underlying feelings of

insecurity regarding their capabilities are similarly associated with anxiety (Houston & Vavak, 1991; Yuen & Kuiper, 1992).

CHD threatens feelings of control and security (Budnick, 1991; Rejeski et al., 1985; Wilson-Barnett, 1979). In the development of the present model, it was proposed that the threats posed by CHD would illicit hostility among predisposed individuals. As a relationship between CHD patients' hostility and anxiety has been found in past research (e.g., Cleveland & Johnson, 1962), it was further proposed that this hostility would predict a change in anxiety. However, this relationship did not occur in the present study.

While CHD may impose the feelings of loss of control and insecurity associated with hostility, active participation in a rehabilitation programme serves to restore control and security (Soloff, 1978a; 1978b). In the period of recovery, the constraints of hospital care are removed and the involvement of medical professionals is reduced to the extent that patients are forced to take control of their health (Rejeski et al., 1985). In accordance with this, exercise based cardiac rehabilitation programmes are structured in such a way that control of the process of recovery is placed in the hands of the individual (Cay et al., 1985). The ability to control the process of recovery by actively participating in an exercise programme provides hostile individuals with a constructive means of fulfilling their need for control. As the focus of this control is on individual performance, hostile individuals have less need and opportunity to display the acts of aggression associated with increased anxiety (Ray & Simons, 1982). Further, the ability to exert considerable physical effort reduces inherently hostile patients' feelings of insecurity regarding their life expectancy, and consequently their feelings of anxiety (Soloff, 1978b). Thus, it is possible that the way in which the rehabilitation programme is structured serves to reduce the feelings of loss of control and insecurity and the combative mode of response linking hostility to anxiety.

An explanation for the absence of a significant relationship may also lie in the type of hostility measured in the present study. Like Type A behaviour, hostility is a multidimensional construct (Dembroski & Costa, 1987; Suarez & Williams, 1990). Selection of the acting-out hostility subscale in the present study was based on its conceptual similarity to the type of hostility described in Friedman and Rosenman's (1974) definition of Type A behaviour. In the original definition of Type A behaviour, hostility was described as expressed aggression and represented antagonistic, rather than neurotic, hostility (Engebretson & Matthews, 1992; Matthews & Haynes, 1986; Siegman et al., 1992). The acting-out hostility measured in the present study reflects the expressed aggression associated with antagonistic hostility (Foulds et al., 1960). A further motivation for measuring the acting-out dimension of hostility was that it is a better predictor of CHD than neurotic hostility (Carmody et al., 1989; Dembroski et al., 1989). However, past research (e.g., Carmody et al., 1989; Dembroski & Costa, 1987; Siegman et al., 1987) indicates that neurotic hostility may be more closely associated with anxiety than acting-out hostility. Though acting-out hostility may have been the more appropriate dimension for the overall examination of the Type A behaviour of CHD patients and its effect on physiological health, it may not have been appropriate for an assessment of change in anxiety.

The present study did not yield the hypothesised relationship between anger-in and change in anxiety. Like anger-out, anger-in is a product of Type A CHD patients' feelings of physical vulnerability and their frustration with the limited ability to control the rate of achievement in the period of recovery (Erdman, 1990). Some Type A's may believe that the outward manifestation of these feelings will jeopardise their public image of strength and control (Price, 1982). Hence, the inclination among certain CHD patients to suppress their anger (Erdman, 1990).

The chronic and extreme effort needed to suppress anger increases both anxiety (Cochrane, 1973) and blood pressure (Baer et al., 1979; Budnick, 1991; Manuck et al., 1985). Among CHD patients, physiological factors are more predictive of prognosis than psychological factors (Palmer et al., 1992). It is possible, then, that anger-in has a greater effect on physiological outcomes such as increased blood pressure than it does on psychological outcomes such as anxiety, to the extent that the former overrides the latter (Palmer et al., 1992). The more dominant relationship between anger-in and blood pressure may explain the absence of a relationship between anger-in and change in anxiety found in the present study. However, as blood pressure was not assessed in the present study, the cogency of this explanation will need to be determined by future research.

It was further hypothesised that the frequency of exercise would predict a reduction in anxiety. Attendance of an exercise programme is believed to distract from daily stressors, provide social support and reinforcement and consequently, reduce anxiety (Roth et al., 1990). However, frequency of exercising was not related to reduction in anxiety. The present result is inconsistent with existing theory concerning the beneficial effects of regular attendance of an exercise programme on psychological condition (Erdman et al., 1986; Folkins, 1976; Shephard et al., 1985; Stern et al., 1983). This result is however, consistent with past research which has failed to find any relationship between exercise frequency and reduced anxiety (De Geus et al., 1993; Pauly et al., 1982). It is plausible that, contrary to extant theory, individual differences in aerobic fitness are the result of a healthy psychological predisposition. This would suggest a reversal of cause and effect wherein, exercise programmes attract individuals who are intrinsically psychologically adjusted, confident and congenial and who have the discipline and energy to comply with long term exercise regimens (De Geus et al., 1993). In turn, this would imply that the present results are a product of self-selection bias and that CHD patients suffering acute anxiety elect not to participate in the exercise programme. To determine the

efficacy of this possibility, the present sample's measure of anxiety taken on admission to the exercise programme was correlated with the exercise frequency measure. This analysis yielded a significant ($p < .01$) negative correlation between the Time 1 measure of anxiety and exercise frequency. The inverse relationship suggests that lower levels of anxiety on admission are associated with higher levels of exercise frequency. It is thus feasible that the present result can be attributed to self-selection bias.

An alternate explanation is that individuals who are in the poorest psychological condition are typically those who derive the most benefit from attending an exercise programme (Altchiler & Motta, 1994; Desharnais et al., 1993). From Table 8.2 it is evident that the present sample's average anxiety scores fall well below the 38 cut-off point required for a diagnosis of clinical anxiety (Cattell et al., 1968). As the present sample do not fit into the category of the clinically anxious, it is possible that they are too psychologically normal for exercise to have exerted an effect. Reductions in anxiety following attendance of an exercise programme have been demonstrated in samples of clinically anxious samples, but not in samples characterised by low levels of anxiety (e.g., De Geus et al., 1993; Desharnais et al., 1993). Thus, a possible cause of the absence of a relationship between exercise frequency and a reduction in anxiety may be the low levels of anxiety of the present sample.

The low levels of anxiety seen in the present sample do not discredit the possibility that anxiety is a typical consequence of CHD. The majority of patients will experience a reduction in anxiety in the first six months of recovery (Brown & Munford, 1984). While the intervening period between experiencing a cardiac event or related surgery and admission to the programme is typically shorter than six months, it may have been sufficient for patients to reach a state of relative psychological stability (Giese & Schomer, 1986). It is possible that most of the

reduction in anxiety occurred prior to admission. As there is a point after which a reduction in anxiety no longer occurs (i.e., a floor effect) (Altchiler & Motta, 1994), it is not surprising that the present study failed to yield a relationship between the frequency of exercise and a reduction in anxiety.

The present results point to the complexity of CHD patients' behaviour. While anger-out predicted less reduction in anxiety, achievement striving, impatience irritability, anger-in and hostility did not. These results highlight the need to define and operationalise Type A behaviour as a multidimensional construct and support the current trend of differentiating between the toxic and benign components of Type A behaviour. However, the present results suggest that the benign and toxic categorisation of the five components recommended by past research on healthy samples (e.g., Bluen et al., 1990; Dembroski et al., 1985; Helmreich et al., 1988; Ganster et al., 1991; Spence et al., 1987) does not apply to CHD patients. In other words, research linking impatience irritability, anger-in and hostility to the anxiety levels of healthy individuals renders the toxic classification of these components appropriate for samples free of CHD. The present results indicate that the effect of impatience irritability, anger-in and hostility on CHD patients' levels of anxiety is benign. Consequently, the toxic classification of these components is inappropriate for models of CHD patients' Type A behaviour. These results support the contention that there is a fundamental difference between the Type A behaviour of healthy individuals and individuals with CHD and its effect on the respective groups' levels of anxiety.

The present results also highlight the multidimensional nature of exercise compliance and the differential effects of its components. Exercise duration was negatively associated with reduction in anxiety, while exercise frequency was unassociated with anxiety in the present study. These results demonstrate the inadequacy of using exercise frequency as the single measure of exercise

compliance. On its own, a measure of exercise frequency establishes how often patients attend the exercise programme but says nothing of their actual exercise performance. It is not surprising then, that use of exercise frequency as a measure of compliance fails to yield a relationship between exercise performance and anxiety. Conversely, exercise duration provides a measure of actual exercise performance. Therefore, use of exercise duration as a measure of compliance makes it possible to identify the underlying mechanisms linking exercise to anxiety. In summary, the present assessment of the predictors of a reduction in anxiety emphasises the importance of developing multidimensional models of Type A and exercise behaviour which are specific to CHD patients.

Change in Depression

In the present study, both anger-in and anger-out were negatively related to depression. As predicted, achievement striving was unrelated to depression. Contrary to expectations, impatience irritability, acting-out hostility, exercise frequency and exercise duration were also unrelated to a change in depression.

The present study yielded negative relationships between anger-in and reduction in depression and anger-out and reduction in depression. Both anger-in and anger-out reflect resentful responses which sustain feelings of anger (Harburg et al., 1991). The fact that both modes of anger expression were negatively associated with change in depression supports the contention that anger is maladaptive regardless of whether it is suppressed or expressed (Taylor & Cooper, 1988). The present results suggest that, whether CHD patients elect to suppress or express their anger, the negative impact on their level of depression is the same. In view of the similarity of results, the relationship between anger-in, anger-out and the reduction in depression will be discussed concurrently.

Among CHD patients, depression has the potential of becoming self-perpetuating (Brown & Munford, 1984). The inability to resolve anger, coupled with the subsequent experience of depression, leads CHD patients to believe that they are failing to manage the stress of CHD (Budnick, 1991; Schwartz et al., 1986). Feelings of failure compromise esteem further and result in more depression (Novaco, 1976; Powell et al., 1993; Yuen & Kuiper, 1992). This self-perpetuating cycle is not typical and the majority of CHD patients overcome their initial depression in the months of recovery (Brown & Munford, 1984). In accordance with the typical process of recovery, the present sample experienced a reduction in depression between Time 1 and Time 2 (see Table 8.2). However, the negative relationship between anger and depression found in the present study suggests that high levels of anger-in and anger-out diminished this reduction in depression.

Confidence and esteem is compromised by the limitations on goal achievement imposed by CHD (Brown & Munford, 1984; Johnson & Morse, 1990). The resulting negative perception elicits anger among CHD patients (Berkowitz, 1983; Budnick, 1991). CHD patients who are unable to transform the anger derived from their change in health status, and the corresponding limitations into socially acceptable forms of behaviour, often suppress their anger (Hackett, 1985). Suppression of anger may leave individuals with the feeling that they are unable to cope with stress (Novaco, 1976; Powell et al., 1993). This perception may, in turn, undermine esteem and create the hopelessness and helplessness associated with depression (Beck, 1970; Erdman, 1990).

Externalising conflict enables outwardly angry individuals to divert attention from their feelings of negative esteem to the failings of others. By expressing their anger, individuals are able to present themselves as potent, determined, in charge and ultimately, able to manage stress effectively (Novaco, 1976). However, the expression of anger does not remove the underlying negative esteem (Budnick, 1991). Further,

any gains in superficial esteem are counteracted by the damage to interpersonal relationships and the consequent social alienation and loneliness caused by irrational outbursts of anger (Budnick, 1991). A persistently poor esteem, together with social alienation and loneliness, contributes to depression (Siegal & Alloy, 1990; Weisse, 1992). Thus, from the present results, it is evident that the presence of anger, be it suppressed or expressed, retards the natural process of recovery from depression. Partial support for the present results is provided by Biaggio and Godwin (1987) and Spielberger et al. (1988), who found that anger-in and anger-out had a negative impact on depression.

As predicted, achievement striving was unrelated to depression in the present study. Individuals characterised by high levels of achievement striving are dynamic, energetic, persevering and quick to take initiative (Price, 1982). These characteristics are diametrically opposed to the apathy and passivity which accompany depression (Finman & Berkowitz, 1989). Kavanagh et al. (1975) maintain that CHD patients who are characterised as driving, ambitious and goal oriented do not experience depression. This result conforms to the widespread theory that achievement striving behaviour is associated with performance and not health (*cf.* Furnham, 1990a). This result also conforms to past research which has found that achievement striving is not associated with depression (e.g., Bluen et al., 1990; Chesney et al., 1981; Edwards & Baglioni, 1991).

The hypothesised relationship between impatience irritability and change in depression did not occur in the present study. The hypothesis relating impatience irritability to a change in depression was, again, based on Type A's need to maintain a high level of esteem through acquiring and achieving as much, in as little time, as possible (Furnham, 1990b; Price, 1982). Price (1982) maintains that impatience irritability is a product of Type A's intolerance and disapproval of any incompetence or delays which threaten their rate of achievement, and consequently, their sense of

esteem. In terms of this definition, impatience irritability is a bi-product of Type A's attempt to overcome negative self-esteem through a high level of achievement (Burke, 1984; Furnham, 1990b). The need to fulfil seemingly impossible goals generates the rapid, but misdirected behaviour and unwarranted time pressure associated with depression (Edwards & Baglioni, 1991; Yuen & Kuiper, 1992). Poor self-esteem is further exacerbated by the negative opinion others have of individuals high in impatience irritability (Steinkamp, 1990). Negative feedback from social networks contributes to depression (Brown & Munford, 1984).

Given the relationship between impatience irritability, poor self-esteem and depression established in the literature, it was assumed that the relationship between impatience irritability and depression would be compounded within the context of cardiac rehabilitation. Thus, following development of the present model, it was hypothesised that among individuals high in impatience irritability, the limited ability to achieve in the period of recovery from CHD, and the negative response from others to their behaviour, would result in a loss of esteem. Consequently, the reduction in depression expected to occur in the months of recovery would be diminished. Contrary to this hypothesis, impatience irritability was unassociated with change in depression.

The present result can be attributed to the difference between the Type A behaviour of healthy individuals and CHD patients, and the different effect it has on the two groups' health (Abbott & Peters, 1988; Orth-Gomèr & Undèn, 1990). In view of the risk of recurrent CHD, patients are encouraged by medical practitioners and significant others to reduce the toxic components of their behaviour (Dembroski & Costa, 1987). Knowledge of their increased risk alters Type A CHD patients' methods of coping (Martin & Lee, 1992). In comparison to Type B's, Type A's are more willing to recognise the consequences of the toxic components of their behaviour (Wright, 1988). Once aware of these consequences, Type A CHD patients attempt to increase

their chances of survival by modifying the toxic components of their behaviour (Evans, 1990; Haynes & Matthews, 1988; Palmer et al., 1992). A significant reduction in the toxic components of Type A behaviour following CHD diagnosis has been demonstrated in past research (e.g., Friedman et al., 1986). Type A CHD patients are also more compliant with medical regimens than Type B patients (Rejeski et al., 1985). Type A's positive response to rehabilitation is likely to result in their psychological recovery occurring earlier and more completely than that of Type B's (Rejeski et al., 1985). Type A CHD patients' modification of their behaviour may result in their impatience irritability being rationalised to the extent that it no longer exerts a negative influence on the natural process of recovery from depression.

From the present result, it is evident that CHD patients' depression is not effected by impatience irritability. The fact that this result is contrary to research which has revealed a positive relationship between impatience irritability and depression (e.g., Billings & Moss, 1982; Bluen et al., 1990; Edwards & Baglioni, 1991; Edwards et al., 1990a, 1990b) among healthy samples supports the notion that the Type A behaviour of CHD patients is fundamentally different to that of individuals free of the disease, and consequently exerts a different effect on depression.

Further support for the contention that Type A CHD patients modify the toxic components of their behaviour is provided by the absence of a relationship between hostility and change in depression found in the present study. In developing the present model, it was hypothesised that CHD patients' hostility would have a negative effect on their recovery from depression. This hypothesis was based on the belief that hostile individuals' antagonistic, rude and uncooperative behaviour hinders the goal achievement so important to Type A's (Taylor & Cooper, 1988). Further, Type A's failure to achieve creates the lack of esteem and feeling of helplessness associated with depression (Hildebrand-Saints & Weary, 1989; Kernis et al., 1989; Matthews, 1982).

An additional factor linking hostility to depression in the literature is reduced social support. With their offensive behaviour, hostile individuals can rely on little social support (Barefoot et al., 1983; Christensen & Smith, 1993; Friedman & Rosenman, 1974). The paucity of social support may be exacerbated among those CHD patients who have changed their employment status and consequently, have limited contact with colleagues and friends (Ruberman et al., 1984). Decreased social contact reduces the possibility of affirmative feedback and leaves hostile individuals with the sense of inadequacy associated with depression (Brown & Munford, 1984). Contrary to existing theory and research (e.g., Biaggio & Godwin, 1987), this hypothesis was not supported in the present study.

It is possible that, in the interests of survival, CHD patients target impatience irritability and hostility for modification. Indeed, during the six month period of review, the present sample demonstrated a dramatic reduction in hostility[37]. Moreover, when the present sample was assessed on entry to the programme, hostility was significantly ($p < .001$) related to depression. Six months thereafter, hostility was no longer related to depression. From these results it is possible to deduce that, in the months of recovery, the present sample of CHD patients' modified their hostility to the point that the effect of hostility on their recovery from depression was nullified.

It is evident from the literature, that Type A CHD patients' modification of the toxic components of their behaviour is often so successful that their behaviour classification prior to the disease is radically different to their classification following CHD diagnosis (Haynes & Matthews, 1988; Matthews, 1988; Wright, 1988). While the absence of a relationship between hostility and change in depression failed to

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[37] *The mean standardised score for the acting-out hostility subscale went from .08 at Time 1 to 11.98 at Time 2. As the acting-out hostility subscale is reverse scored and has a five point range, this represents a considerable reduction in hostility between Times 1 and 2.*

support the present hypothesis, it contributes to the argument that attempts to modify behaviour in the months of recovery results in a basic difference between the Type A behaviour of healthy and CHD patient samples, and the effects thereof on depression.

In addition to the Type A components, the present study assessed the relationship between the components of exercise compliance and depression. Contrary to the present hypothesis, neither the frequency nor the duration of exercise predicted a change in depression. Regular exercise of sufficient duration is believed to reduce the deleterious impact of stress on psychological health in general and depression in particular (De Geus et al., 1990; Folkins & Sime, 1981; Kavanagh et al., 1977; Selye, 1974). The means by which stress exerts a negative influence is through heightened sympathetic activity (Verrier, 1987). Aerobic fitness is thought to reduce both the sympathetic response to stress and the recovery time from stress, and consequently, lower levels of strain such as depression (De Geus et al., 1990; McGlynn et al., 1983). However, the present study failed to show that compliance with the frequency and duration components of the exercise prescription had an attenuating effect on the level of depression experienced by the present sample.

It may be that exercise does not, at least in the short term, have the hypothesised attenuating effects on sympathetic activity. If exercise does not have a stabilising effect on sympathetic activity, it is also unlikely to have an effect on depression. Indeed, De Geus et al. (1990) have shown that a seven week exercise programme failed to alter sympathetic responses to stress. A further study by De Geus and colleagues (1993) has shown that the aerobic fitness derived from an eight month exercise programme was not associated with an improvement in a number of psychological variables, including depression. Martinsen (1987) has demonstrated that with 30 to 60 minutes of exercise performed three times a week it is possible to achieve aerobic fitness within six to eight weeks, but that the reduction in

depression associated with exercise occurs in a considerably shorter space of time. From the results of both the present and past research it is possible to conclude that the reduction in depression may not be dependent on increased aerobic fitness (Altchiler & Motta, 1994; De Geus et al., 1993; Fletcher et al., 1992).

The lack of significant results may also be a function of the level of depression experienced by the current sample. It has been argued that exercise only tempers the effects of depression in cases of severe depression (Shephard et al., 1985). Indeed, Kavanagh et al. (1977) have shown that a four year exercise programme reduced the depression of severely depressed patients. The present sample's average BDI depression scores of 7.59 at Time 1 and 6.40 at Time 2 fall below the cut-off point of 9 required for a diagnosis of severe depression (Endler et al., 1992).

In summary, the present results indicate that the various components of Type A behaviour have a differential effect on psychological health. Specifically, anger-in and anger-out predict less reduction in depression, while achievement striving, impatience irritability and hostility are not associated with a reduction in depression. As the present results indicate that not all of the 'toxic' components of Type A behaviour predict negative outcomes, the multidimensional nature of the construct and the differential effect of its components is confirmed. The results support the view that there is a difference between the Type A behaviour of healthy individuals and individuals with CHD. Also indicated is the toxicity of anger in respect to CHD patients' levels of depression and consequently, the need to examine the dynamics of anger in future research concerning CHD patients' psychological health. The relationship between CHD patients' exercise behaviour and depression will also require closer scrutiny in future research. The present study's failure to yield relationships between exercise duration and frequency and the reduction in depression suggests that changes in depression are not contingent on increased aerobic fitness. However, these results may also be a product of the low levels of

depression of the present sample. In view of the pervasiveness of depression in the general CHD patient population (Brown & Munford, 1984), and the potential for exercise to reduce severe depression, future research should attempt to identify the point at which physical fitness exerts an effect on psychological health.

Change in Maximal Oxygen Uptake

Components of the present sample's Type A and exercise compliance behaviour were assessed in relation to a measure of physical health, namely, maximal oxygen uptake (Cohn et al., 1979; Cooper et al., 1991; Desharnais et al., 1993; Uhl et al., 1984). While the hypothesis stating that achievement striving would not be associated with maximal oxygen uptake was supported in the present study, the hypothesis stating that impatience irritability, hostility, anger-in and anger-out would be negatively associated with change in maximal oxygen uptake was not. Support was found for the hypothesis predicting a positive relationship between the duration of exercise and the present samples' increase in maximal oxygen uptake, but not for the predicted relationship between the frequency of exercise and maximal oxygen uptake.

The present study found partial support for the hypothesis that compliance with an exercise programme would predict physiological condition as measured by maximal oxygen uptake. Specifically, the duration of exercise predicted an increase in maximal oxygen uptake while the frequency measure did not. The present results demonstrate that the frequency of exercise does not have an isolated impact on cardiovascular condition. These results indicate, that while patients might attend the exercise programme with the required frequency, little physiological benefit will be achieved unless this occurs for sufficient duration. That is, without exercising for the required one to two hours per week (Oberman, 1983), patients will not activate the relevant systems needed to exert a physiological effect (Cupelli et al., 1984).

The results of the present study are similar to those reported by Lakka et al. (1994), where the duration of exercise correlated directly with maximal oxygen uptake after adjustment for age and year of examination. Further, both exercise duration of a minimum of two hours per week and maximal oxygen uptake were independently and inversely related to the risk of myocardial infarction (Lakka et al., 1994).

Much research has argued for the strong protective effect of exercise (Willich et al., 1993). The present findings question those studies which have measured compliance exclusively in terms of attendance (e.g., Blumenthal et al., 1982; Oldridge & Jones, 1983). These findings also provide a possible explanation for some of the non-significant relationships between exercise frequency and improved physiological condition reported by past research (e.g., Fontana, Kerns, Rosenberg, Marcus, & Colonese, 1986). These results also question the practice in exercise rehabilitation of using attendance as a marker for compliance (Oldridge, 1988). The present results showing a significant association between the duration of exercise and maximal oxygen uptake support the argument for a relationship between exercise and enhanced physiological condition.

As predicted, achievement striving was unrelated to maximal oxygen uptake in the present study. This result is consistent with the theory and research which states that achievement striving behaviour is unassociated with measures of health (e.g., Barling & Charbonneau, 1992; Öhman et al., 1989; Rimé et al., 1989; Spence et al., 1987). However, contrary to predictions, none of the toxic components of Type A behaviour predicted a decrease in maximal oxygen uptake. These results are also contrary to past research on healthy samples in which relationships between the toxic components and indices of physiological health have been reported (e.g., Christensen & Smith, 1993; Harburg et al., 1991; Jennings, 1981; Manuck et al, 1985; Öhman et al., 1989; Suarez & Williams, 1990; Svebak et al., 1992).

The absence of a relationship between the toxic components and maximal oxygen uptake found in the present study does not dispel the importance of studying Type A components, but instead, adds to the increasingly widespread belief that the processes underlying the initial development of CHD are not the same as those influencing the incidence of recurrent CHD (Barefoot et al., 1989). The psychological impact of CHD may vary according to behavioural type (Evans, 1990). It is believed that CHD generates a re-evaluation of lifestyle and modification of behaviour among Type A's with the result that the constellation and intensity of Type A behaviours alters in the first few months following CHD diagnosis (Miller et al., 1991). This may translate to better compliance with medical and exercise regimens among Type A's (Suls & Marco, 1990).

Medical and exercise regimens, alone, have been found to alter Type A behaviour. For example, the beta-blocking drugs prescribed for many CHD patients reduces the intensity of Type A behaviour (Abbott & Peters, 1988; Powell, 1987). Aerobic exercise is also thought to moderate the toxic components of Type A behaviour to the extent that they no longer exert a negative effect. Research by Blumenthal, Williams, Williams and Wallace (1980) does show that participation in an exercise programme reduces Type A behaviour.

A further influencing factor is that Type A behaviour advances the occurrence of cardiac events with the result that Type A's are diagnosed with CHD at an earlier age than Type B's (Miller et al., 1991). The resilience associated with their relative youth is likely to enhance Type A's ability to make the behaviour changes necessary to reduce the risk of recurrent CHD (Miller et al., 1991). Together, these factors may result in a decreased risk of recurrent CHD (Evans, 1990; Palmer et al., 1992).

The present results contradict the results of some studies of Type A components (e.g., Edwards & Baglioni, 1991) and the earlier findings of the WCGS. However, the

present results are consistent with the growing body of research which suggests that Type A components do not predict recurrent CHD (Palmer et al., 1992). For example, Miller et al.'s (1991) meta-analysis shows that studies incorporating samples of CHD patients account for the majority of null findings in Type A-CHD research while many of the studies using healthy samples generated positive associations between Type A behaviour and CHD. The absence of significant relationships between the toxic components of Type A behaviour and indices of CHD found in the present and past research can be attributed to the differential influence of Type A behaviour on primary and secondary CHD. Therefore, it would be incorrect to reject the overall predictive power of Type A components on CHD based on the data derived from studies concerning survival following primary CHD (Palmer et al., 1992).

In summary, the maximal oxygen uptake of the present sample increased during the six months of review. The Type A predictors achievement striving, impatience irritability, anger-in, anger-out and hostility were not associated with this increase. The absence of a relationship between achievement striving and maximal oxygen uptake is consistent with research which has demonstrated that the component is unassociated with measures of health. However, the results concerning impatience irritability, anger-in, anger-out and hostility are surprising given the relationship between these components and physical health which have been reported in past research concerning healthy individuals. There is increasing consensus in the literature that the toxic components of Type A behaviour predict primary, but not secondary, CHD. The present results indicate that Type A behaviour has a different effect on the physical health of individuals who have CHD and individuals who are free of the disease. Therefore, the aforementioned consensus is added to by the present study's failure to yield any relationships between the five components of Type A behaviour and a measure reflecting CHD patients' prognosis for recovery or reinfarction, namely, maximal oxygen uptake (Cooper et al., 1991).

Of the two measures of exercise compliance which were assessed, only the duration component of exercise was associated with an increase in maximal oxygen uptake. Again, these results support the argument that exercise frequency is an inadequate measure of exercise compliance. In order to increase understanding of the means by which exercise effects health, future research should focus on the duration, and not the frequency, of exercise performance. A discussion of the theoretical and practical implications of the present findings follows.

Theoretical Implications of the Present Study

The global conceptualisation of Type A behaviour has been subjected to considerable criticism in recent years (Dembroski et al., 1989; Grossarth-Maticek & Eysenck, 1990; Lee, 1992). Use of the global construct is thought to have concealed important relationships between Type A components and outcome variables (Forgays, 1992; Lee, 1992, Steinkamp, 1990) and to have been responsible for the ambiguous and equivocal findings of past research (Carver, 1989; Ganster et al., 1991). The global Type A construct has also demonstrated limited predictive power (Evans, 1990). Thus, the broad and often vague conceptualisation of Type A behaviour can be said to have undermined past research (Ganster et al., 1991). The current trend of conceptualising Type A behaviour as a multidimensional construct has met with greater success. In those studies which have operationalised Type A behaviour as a multidimensional construct (e.g., Bluen et al., 1990; Burns, 1992; Weidner et al., 1989), it has been demonstrated that Type A behaviour comprises a number of components each of which exert differential effects.

Recent component research has eliminated much of the ambiguity and vagueness surrounding Type A behaviour and has contributed greatly to current understanding of the mechanisms linking Type A behaviour to health, albeit in low-risk samples. This research has also confirmed that Type A behaviour is a multidimensional

construct (Edwards et al., 1990a; Tett et al., 1992). The present application of the multidimensional conceptualisation of Type A behaviour within the context of CHD has further advanced extant knowledge of the construct. Ganster et al. (1991) criticise the popular trend of stating that Type A behaviour is a complex construct comprising a number of yet to be revealed components. The present research addresses this criticism by providing insight into the nature of CHD patients' Type A behaviour and increases the precision of its definition by revealing five of its components. The results of the present study support the contention that Type A behaviour is a multidimensional, and not a global, construct. By demonstrating the differential effects of these components on CHD patients' health the present study has also identified those components which impede recovery and those which have no such effect. Identification of the differential effects of the Type A components would not have been possible with a model based on the global construct. Therefore, the results of the present study support the independent conceptualisation and operationalisation of the Type A components.

Models of Type A behaviour developed in the past have also been criticised for being insufficiently comprehensive (Russek et al., 1990). Specifically, they have been criticised for failing to consider anger and hostility (Evans, 1990; Forgays, 1992; Lee, 1992). Numerous researchers contend that anger and hostility are major determinants of CHD and psychological ill health (e.g., Booth-Kewley & Friedman, 1987; Edwards et al., 1990a; Evans, 1990; Julius et al., 1986; Lee, 1992; Matthews, 1982; Meininger et al., 1991). Past research which reveals a significant relationship between anger, hostility and indices of CHD supports this contention (e.g., Barefoot et al., 1983; Dembroski et al., 1985; Gilbert & McArthur, 1988; MacDougall et al., 1985; Tennant & Langeluddecke, 1985; Wright, 1988). So too does the research which has revealed significant relationships between anger, hostility and anxiety (e.g., Cleveland & Johnson, 1962; Edwards & Baglioni, 1991; Spielberger et al., 1985) and anger, hostility and depression (e.g., Biaggio & Godwin,

1987; Dimsdale et al., 1978; Spielberger et al., 1988). Despite the aforementioned evidence, anger and hostility are not included in the majority of extant measures of Type A behaviour. This omission has limited the possibility of identifying the effects of two of the most toxic correlates of Type A behaviour in past research.

In the present research, this limitation was addressed by incorporating measures of anger-in, anger-out and hostility in the Type A Component Questionnaire. No significant relationships between anger-in, anger-out and maximal oxygen uptake were found in the present study. However, anger-out was negatively related to decreased anxiety and both anger-in and anger-out, were negatively related to decreased depression. This implies that, as a consequence of their anger, Type A CHD patients are at particular risk of suffering persistent anxiety and depression. The negative effects of anger-in and anger-out on the present sample's psychological health suggests that these two components are primary predictors of psychological ill heath among CHD patients. Consequently, anger-in and anger-out should be included in models of CHD patients' Type A behaviour.

While anger-in and anger-out were associated with psychological outcome in the present study, neither were associated with the physiological outcome variable. These findings are contrary to research on healthy samples wherein anger-in and anger-out were significantly related to indices of CHD (e.g. Baer et al., 1979; Engebretson et al., 1989; Falkner et al., 1981; Gentry et al., 1982; Harburg et al., 1991; Haynes et al., 1980; Johnson & Broman, 1987; Manuck et al., 1985; Tennant & Langeluddecke, 1985). The absence of relationships between hostility, impatience irritability and anxiety, depression and physiological condition (as measured by VO_2max) is also contrary to past research on healthy samples (e.g., Barefoot et al., 1983; Biaggio & Godwin, 1987; Bluen et al., 1990; Edwards & Baglioni, 1991; Harburg et al., 1991; Johnson & Broman, 1987; Matthews et al., 1977; Maes et al., 1987; Shekelle et al., 1983; Tennant & Langeluddecke, 1985). It appears then, that

the components which predict physiological ill health among healthy individuals do not necessarily predict ill health among CHD patients.

From the results of the present and past research, it is evident that Type A behaviour, and the components thereof, predict psychological and physiological health in healthy samples but not in samples of CHD patients (Matthews, 1988). This discrepancy has led a number of researchers to argue that there is a fundamental difference in both the nature and effect of the Type A behaviour demonstrated by healthy individuals and individuals with CHD. The difference between the present results and those of past research based on healthy samples suggests that the Type A behaviour of CHD patients is not as toxic as the Type A behaviour of healthy individuals. The difference supports Wright's (1988) contention that, unlike healthy Type A's who deny the ramifications of their behaviour, CHD patients recognise and address the negative consequences of their behaviour to the extent that it no longer exerts an impact on their health. The non-significant relationships between the present sample's hostility, impatience irritability and their psychological and physiological health attests to the success of their attempts to modify some of the more toxic components of their behaviour. These findings also raise a question as to the relevance of including measures of hostility and impatience irritability in research concerning CHD patients.

Given the difference between Type A's who are healthy and Type A's who have CHD, multidimensional models developed for healthy samples have limited utility in research concerning samples of CHD patients. Thus, existing component research was extended in the present thesis by the development of a multidimensional model of Type A behaviour which is specific to CHD patients. From the present study it is possible to conclude that in developing models of Type A behaviour for CHD patients, anger-in and anger-out should be emphasised, and hostility and impatience irritability de-emphasised.

Measures of the frequency and duration of exercise compliance were also included in the present multidimensional model of CHD patients' behaviour. While much is known about exercise, there is a limited amount of actual data on the effects of exercise on the cardiovascular system and on long term survival (Fletcher et al., 1992). The present study provides data on the effects of the duration of exercise on maximal oxygen uptake. From the results of the present study, it is evident that the duration of exercise facilitates an increase in maximal oxygen uptake, while the frequency of exercise has no such effect. It is possible to conclude from these results that cardiovascular fitness is a product of the duration of exercise compliance.

The bulk of past research has utilised the unidimensional measure of attendance (i.e., the frequency of exercise) to assess the effects of exercise (e.g., Bethell & Mullee, 1990; Cay et al., 1985; Grodzinski et al., 1987; McGlynn et al., 1983; Pisa et al., 19[illegible] Shaw, 1981; Stern et al., 1983). A number of these studies have revealed significant relationships between the frequency of CHD patients' exercise and improvements in their physiological condition and prognosis for survival (e.g., Bethell & Mullee, 1990; Cay et al., 1985; Grodzinski et al., 1987; McGlynn et al., 1983; Shaw, 1981). In other studies, frequency of exercise was not associated with changes in CHD patients' physiological condition (e.g., Pisa et al., 1985; Stern et al., 1983). Given the present results, it is possible that the equivocal nature of past research can be attributed to the use of attendance as the single measure of compliance. The fact that the duration, and not the frequency, of exercise predicted improvements in physiological health in the present study has important implications for future research. From the present findings it appears that the frequency with which patients exercise is only relevant if each period of exercise is long enough to activate the systems associated with improved physiological condition (Cupelli et al., 1984). In order to determine the precise effects of exercise on cardiovascular health, future research will need to focus on the duration of exercise compliance.