

**"TYPE A BEHAVIOUR PATTERN AND COMPETITION IN
ADULTS AND ADOLESCENTS".**

by

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"If one binds one's heart firmly and imprisons it
one can allow one's spirit many liberties: I have
said that before. But no one believes it if he
does not already know it..."

Friedrich Nietzsche

ABSTRACT

A set of behaviour patterns characterized by excessive hostility, competitiveness, and impatience have been identified in patients with coronary heart disease (CHD) and labelled "Type A". Since several studies have shown that the Type A behaviour pattern is an independent psychological risk factor for heart disease, attempts have been made to modify the behaviour pattern in both healthy subjects and patients suffering from CHD by altering physiological, cognitive and behavioural characteristics.

The present research offers an alternative approach to the modification of physiological responses in healthy Type A adults and adolescents. It is proposed that the core elements of Type A such as competition could be exploited in order to induce physiological relaxation rather than excitement. Four experiments were carried out. The first study identified behavioural and physiological characteristics of Type A subjects such as increased heart rate while competing. The second experiment used contingent and false heart rate biofeedback training to challenge Type A adults to decrease heart rate in a competitive situation. Biofeedback was found to be an effective method of training Type A subjects to relax. Furthermore, it was

found that Type As decreased heart rate significantly more than other subjects when challenged to do so by biofeedback.

As a first step in attempting a similar approach with adolescents and to see if an identifiable pattern of beliefs and fears associated with Type A exists in the younger population, a questionnaire was designed to identify these beliefs and fears. Type A adolescents were found to hold a similar pattern of beliefs and fears to that shown in adult subjects in previous studies. Furthermore, measures of beliefs and fears correlated significantly but modestly with measures of Type A.

Using heart rate biofeedback, competition did not seem to have any specific effect on the performance of Type A adolescents. Nevertheless, Type As were found to be more responsive to biofeedback training than Type Bs and results for subjective reports of relaxation were encouraging.

The results are discussed in terms of using challenging incentives to reduce physiological responses in Type A adults and adolescents. It is argued that programmes aimed at modifying Type A in healthy individuals have produced mixed results and have repeatedly failed to alter physiological responses. This is due to many

problems including the fact that the negative consequences of the behaviour pattern are not readily recognized and therefore healthy Type As may be resistant to change. Furthermore, there are ethical issues involved in actually altering behaviour in an apparently healthy population. It is proposed that in view of these problems, challenging incentives such as competition could play a significant role in the reduction of physiological responses in healthy Type A individuals particularly the younger population.

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CHAPTER ONE

GENERAL INTRODUCTION

The purpose of this chapter is to outline and discuss the literature on the Type A behaviour pattern. It will be pointed out that despite some recent negative data, the Type A behaviour pattern has received a great deal of attention as an independent psychological risk factor for coronary heart disease (CHD). Since the mid 1970's, researchers have tried to modify the behaviour pattern in both patients suffering from heart disease (Friedman et al 1984) and healthy Type A individuals (Roskies et al 1987). The present chapter will emphasize the fact that although research has shown that the Type A behaviour pattern is indeed modifiable, results are far from convincing in showing that physiological reactivity can be modified using traditional methods such as stress management techniques. This is particularly the case for healthy Type A subjects. The present chapter will also critically discuss theories of Type A and will outline research on behavioural and physiological characteristics of the Type A behaviour pattern with particular reference to competition. It will be concluded that the cognitive social learning model of Type A seems to be the most promising approach to the understanding of the concept but research is needed to confirm some of its assumptions. It will be pointed out that research is also

needed to confirm some of the behavioural and physiological differences between Type A and B individuals and to examine some of the implications these differences might have.

1.1. CORONARY HEART DISEASE (CHD) AND TRADITIONAL RISK FACTORS

More individuals in western societies die of cardiovascular diseases than of any other cause (Dembroski et al, 1983). In the majority of cases, disease of the coronary arteries is the result of atherosclerosis, the narrowing of coronary arteries by gradual accumulation of plaque or scaling on their walls, although other etiological factors can contribute to the clinical presentation of coronary artery disease such as coronary artery spasm and congenital abnormalities (Feuerstein et al 1986). Coronary heart disease (CHD) takes two principal forms: angina pectoris and myocardial infarction or heart attack. The symptoms of angina pectoris are periodic chest pains and the principal cause is an insufficient supply of oxygen to the heart as a result of atherosclerosis. Myocardial infarction (MI) is a much more serious disorder and is one of the leading causes of death in industrialized countries. The oxygen deficiency, more extreme than in angina pectoris, causes parts of the heart muscles to die (Friedberg, 1966).

The American Heart Association has listed several factors related to increased risk of CHD. They include age, sex (males are at a greater risk), tobacco smoking, consumption of alcohol, elevated serum cholesterol, an increase in the size of the left ventricle of the heart as revealed by an electrocardiogram, and diabetes (Obrien et al 1974). The risk for CHD generally increases with the number and severity of these factors. However, some researchers (for example, Jenkins, 1976; Friedman and Rosenman, 1959) have argued that these traditional risk factors leave at least half of the etiology of CHD unexplained. The search for other causes of CHD has focused on psychological factors such as stress and personality.

1.2. STRESS AND THE ETIOLOGY OF CORONARY HEART DISEASE

The role of stress related factors in the etiology of coronary heart disease (CHD) has been acknowledged for many years. For example, Osler (1892) attributed CHD to "the high pressure at which men live and the habit of working the machine to its capacity". According to Osler, the coronary patient was not "the delicate neurotic person but the robust, the vigorous in mind and body, the keen and ambitious man, the indicator of whose engine is always at full speed ahead". A number of researchers in the first half of the present century have generally agreed with Osler's points. With the dramatic increase in the incidence of

coronary heart disease during the twentieth century, the attention of researchers was diverted more towards links between coronary heart disease and personality factors. For example, Dunbar (1943) claimed that the hard-driving, goal-directed behaviour of CHD patients constituted a coronary personality. Kemple (1945) confirming Dunbar's assertions, suggested, as a motive for this overdependence on external achievement, that it might be a compensation for deficiencies in "introversive experiences of creative thought".

Most researchers, however, remained pessimistic about the role of behaviour in the epidemiology and treatment of CHD until the late 1950's when the relationship between personality factors and coronary heart disease was reconceptualized in a coherent way and the clinical portraits presented by early researchers became known as the Type A behaviour pattern.

1.3. THE TYPE A BEHAVIOUR PATTERN: DEVELOPMENT OF THE CONCEPT

The most promising evidence linking CHD to psychological variables comes from investigations initiated in the mid 1950's by two cardiologists Meyer Friedman and Ray Rosenman. In a review of the literature on the risk factors involved in CHD, Friedman and Rosenman argued that the traditional

risk factors were inadequate to account for the high incidence of heart disease and for the differential rate of the disease between men and women. They speculated that the infrequent exposure of women to the stressful socio-economic milieu in which men spend forty or more hours per week might account for the discrepancy, since neither dietary nor hormonal differences did.

Based on clinical observations on a large group of heart patients, Friedman and Rosenman found that these patients did exhibit a characteristic behaviour pattern which they labelled "Type A" or the "coronary prone behaviour pattern". Glass (1977) describes the Type A individual as having an intense and competitive drive for achievement and advancement; an exaggerated sense of the urgency of passing time, of the need to hurry; and considerable aggressiveness and hostility towards others. Type A persons are overcommitted to their work, often carry on two activities at once and believe that to get something done well, they must do it themselves. They are fast thinking, fast talking and abrupt in gesture. Type As are impatient and highly competitive. They play every game to win even when their opponents are children. The Type B individual on the other hand, is relaxed and relatively free from such pressures.

In order to explore the predictive validity of the Type A behaviour pattern, Friedman, Rosenman and their associates

undertook the Western Collaborative Group Study (WCGS) in 1960. This was a longitudinal study of 3154 apparently healthy men aged 39 to 59 years. Based on a structured interview developed to measure Type A behaviour, subjects were labelled as either possessing the Type A behaviour pattern (n= 1589) or the more relaxed, easy going pattern of behaviour referred to as Type B (n= 1565). Type A was conceptualized as consisting of the following components: an intense desire to achieve, a profound eagerness to compete, a persistent drive for recognition, continuous involvement in activities that require deadlines, a tendency to accelerate all mental and physical activities and mental and physical alertness (Friedman and Rosenman, 1959). The study took eight and a half years to complete and the results showed Type A individuals were more than twice as likely to develop CHD as Type B men. In addition, Type A men with CHD were five times as likely to have a second myocardial infarction as other individuals with CHD. These findings from the WCGS have lent support for the notion that Type A behaviour pattern contributes significantly to the development of CHD.

Although the results of the WCGS were dramatic, replications were necessary, preferably with different populations. The WCGS spawned a number of prospective studies to confirm the ability of the Type A behaviour pattern to predict the incidence of heart disease.

There are a number of studies which have confirmed the results of the WPCS. For example, the Framingham Heart Study, using an American sample but unlike the WPCS including women, blue-collar workers and the unemployed in addition to white-collar men confirmed the ability of the Type A behaviour pattern to predict heart disease over an eight year period. Type A men aged 45 to 64 were twice at risk of angina, myocardial infarction and CHD in general as compared with Type B men (Haynes et al 1980). Furthermore, the increased coronary risk associated with the presence of the Type A pattern was even stronger in women. Type A women had two times the rate of CHD and three times the rate of angina, compared to Type B women (Haynes and Feinleib, 1980).

A second prospective study investigated male white-collar workers in Belgium and France. Results suggest that men who were assessed to be least Type A had an annual incidence of CHD of 5.4 per 1,000, compared with an incidence of 9.2 per 1,000 for the highest Type A scorers (Belgian-French Pooling Project, 1984).

Despite all these results some other prospective studies reported no relationship between Type A behaviour pattern and increased risk of heart disease. The two studies have used samples of men already manifesting CHD or classified as

high risk. The Multiple Risk Factor Intervention Trial (MRFIT), using a sample of men already manifesting at least two of three specified CHD risk factors (smoking, high cholesterol and high blood pressure) reported no relationship between the Type A behaviour pattern and a seven year incidence of CHD (Shekelle et al 1983). Data from the Aspirin Myocardial Infarction Study (AMIS) , using a sample of survivors of a first myocardial infarction, showed that Type A men were at no greater risk of a second infarction or coronary death than Type B individuals (Ruberman, Weinblatt, Goldberg and Chaudbury, 1984). More recently, Gallacher, Yarnell and Butland (1988), in the Caerphilly Heart Study, using 1,956 employed men, have reported mixed results. Type A was found to be linked with the incidence of myocardial infarction but independent of angina pectoris.

Despite the contradiction in findings concerning the predictive validity of the Type A behaviour pattern, Roskies (1987) has criticized the studies reporting negative results. None of the negative findings, except one study, was reported in a sample of the apparently well. And the one study which has used healthy subjects (Leon et al 1988) has been criticized for failing to use reliable assessment techniques in measuring Type A. It is possible that in individuals with high risk status, or clinical signs of

heart disease, the effects of the Type A behaviour pattern be masked.

An influential recent article by Ragland and Brand (1988) has added to the fervor of this debate. The authors studied 257 male patients with CHD from the initial phase of the WCGS to see whether behaviour type assessed before CHD event was related to CHD mortality. It was found that subsequent coronary mortality in patients who had suffered a first coronary event was unexpectedly lower among Type A than Type B patients. Although the authors contend that their results need confirmation, they conclude that patients with CHD and a Type A behaviour pattern are not at increased risk for subsequent CHD mortality.

Based on some of these recent negative findings, Dimsdale (1988) argues that, although early on there was impressive evidence that the Type A behaviour pattern was an independent risk factor for CHD, it must be acknowledged that this evidence has been tarnished by recent findings. Dimsdale agrees that the relationship between personality and heart disease can not be denied. However, the nature of this influence seems to be far more complex than is conveyed by the simple assertion that the Type A behaviour pattern is a risk factor for CHD.

The debate still goes on. The American National Heart, Lung, and Blood Institute following a major review of the possible link between the Type A behaviour pattern and heart disease concluded that at least for employed middle-age men, the Type A behaviour pattern can be categorized as a major independent risk factor for coronary heart disease. The behaviour pattern was thought to be of the same magnitude as smoking, elevated cholesterol, and hypertension (Quoted in Roskies, 1987). Furthermore, an independent review panel in 1981 recognized the Type A behaviour pattern as a high risk factor for coronary artery disease. Recent evidence would seem to refute these conclusions. But despite these negative findings, there are several lines of evidence which seem to support the possible relationship between the Type A behaviour pattern and CHD. Some researchers have argued that there may be finer areas of the Type A behaviour pattern which may be associated to CHD rather than the behaviour pattern as a whole. Some evidence seems to suggest that anger and hostility, for example, may be the Type A elements associated directly with heart disease (Williams, Barefoot and Shekelle, 1985). It has been suggested by some researchers (eg Price, 1983) that salient aspects of Type A such as competition (as in the present research) and hostility should be concentrated upon and that it seems useful to study the Type A behaviour pattern as a multidimensional concept comprising of several behaviours rather than as a independent behaviour pattern.

Some researchers have viewed the physiological data as further evidence for the claim that Type A behaviour pattern is a major risk factor for the incidence of CHD. Evidence from physiological studies on the Type A behaviour pattern seem to suggest that there are distinct physiological characteristics which separate Type As from Type Bs. For example, it has been suggested that the Type A behaviour pattern may be linked to CHD via two related neuroendocrine pathways, namely, the pituitary-adrenal-cortical and more importantly, the sympathetic-adrenal-medullary systems (Williams et al 1978). The sympathetic-adrenal-medullary system, responsible for the fight-flight response, is activated when a person perceives an event to be a stressor. When this system is activated, various neurohormones, especially the catecholamines adrenaline and noradrenaline are released. Excess noradrenaline seems to be excreted when the individual perceives the situation as threatening but is striving to keep control over it (Friedman et al 1975). Empirical evidence suggests that Type A individuals have higher levels of circulating noradrenaline (Friedman et al 1975). Excess secretion of noradrenaline is thought to affect the heart by promoting myocardial and arterial lesions and also by raising systolic and diastolic blood pressure, elevating cholesterol levels and reducing blood clotting time (Vasina, 1983). It is suggested that rapid alteration between the sympathetic-

adrenal-medullary and the pituitary-adrenal-cortical systems may contribute to cardiovascular disease (Engel, 1970). Further physiological evidence will be outlined in future sections.

Finally, there have been several attempts to actually modify the Type A behaviour pattern in both patients suffering from CHD and normal subjects (Friedman et al 1982; Powell et al 1984; Roskies, 1987). The results of these modification programmes seem to suggest that not only the Type A behaviour pattern can be successfully altered in subjects but Type As can also be helped to remain healthy, for example, by preventing a second heart attack. Despite the equivocal nature of the evidence concerning the relationship between Type A and CHD the role of the Type A behaviour pattern as a risk factor for CHD, is confirmed by the fact that patients have been less prone to CHD after undergoing these modification programmes. A full discussion of these programmes will be offered in future sections.

1.4. THE TYPE A BEHAVIOUR PATTERN: COMPONENT ANALYSIS

There have been many attempts to isolate various dimensions of the Type A behaviour pattern and analyze different characteristics which typify Type A individuals. The first description of the characteristics exhibited by Type As came from clinical impressions from Friedman and Rosenman. These

clinical impressions were based on extensive work with CHD patients. Friedman and Rosenman (1974) pointed out that the major components of the Type A behaviour pattern were hostility and time urgency. Although their clinical impressions provided a most promising lead, empirical research to establish the central role of these characteristics has been surprisingly meagre until relatively recently (Price, 1983).

Various correlational studies have aimed to establish an association between certain aspects of the Type A behavior pattern and certain physiological mechanisms (for example, Friedman et al 1964). More recently Williams et al (1985) examined the relationship between hostility and coronary artery disease (CAD). A significant correlation between hostility and incidence of atherosclerosis, a primary precursor of symptomatic CHD was reported.

A major criticism aimed at correlational studies has been that a significant correlation does not necessarily mean a causal relationship. That is, association does not imply causation. In order to establish causality other lines of scientific research should be considered.

The same criticism applies to a variety of methodological approaches which try to identify the major components of Type A behaviour by either using factor analytic or multiple

regression techniques. For example, Zyzanski and Jenkins (1970) factor analyzed the Jenkins Activity Survey, a questionnaire measuring Type A, and identified two major independent factors. These were speed and impatience, competition and ambition. Factor analysis of the structured interview developed to identify Type A individuals in the WCGS, revealed five major factors: competitive drive, past achievement, impatience, non job related achievements, and speed.

Using a multiple regression technique, Schucker and Jacobs (1977) found that a substantial portion of the variance in the total Type A score, as measured by the structured interview, was accounted for by the volume and speed of subjects' speech. Type A individuals tended to speak loudly and quickly. Therefore, a very important part of the behaviour pattern is speech and motor characteristics. These statistical methods, however, are based on analyzing self report measures which will be criticized in this chapter. It is necessary to observe subjects' behaviour in controlled situations in order to test the assumptions made by the above methods.

1.4.1 Laboratory research: Behavioural and physiological characteristics of Type A individuals

The most promising method of identifying various components of the behaviour pattern is experimental laboratory research which attempts to test the conclusions from studies using methods such as factor analysis and multiple regression to isolate particular aspects of the Type A behaviour pattern. Laboratory studies have not only confirmed a great deal of these conclusions but have also found distinct behavioural and physiological characteristics in Type As. For example, the achievement striving component of Type A individuals has been tested in a number of experiments. In one experiment the performance of Type As and Type Bs were compared in deadline versus non deadline conditions (Burnham et al 1975). The task was to solve 240 arithmetical problems. It was expected that Type As and Bs would perform similarly under the deadline condition, but Type As would attempt to solve more problems than Type Bs under the non deadline condition, since Type As are thought to work at near maximum capacity irrespective of task demands. Both experimental hypotheses were confirmed by the results.

In another experiment, examining the hypothesis that Type A individuals are more aggressive than Type Bs, male subjects were exposed to a situation in which half of them were insulted by a confederate while trying to perform a complex perceptual task (Carver and Glass, 1978). Subjects were then given a chance to administer electric shocks to the confederate. It was found that Type As delivered higher

levels of shock than Type Bs. It was concluded that given a challenging or threatening situation, Type A individuals exhibit significantly more aggression than Type B individuals.

Similarly, Yarnold and Grimm (1988) have found that Type A subjects exhibit pro-self and anti-others behaviour when involved in an argumentative discussion. Type As were found to strive for dominance and show more hostility and aggression towards others.

The role of sex differences has also been examined by some researchers but with mixed results. Van Egeren (1979) found that females had larger heart rate responses than males when playing a game under cooperative strategy. Other studies however, show that females respond to demanding situations with less psychological and physiological arousal than males (Manuck et al 1978; Matthews and Haynes, 1986). However, a number of studies have failed to report any significant differences between males and females (Price, 1983).

Nevertheless, there seems to be a trend in the literature on competition suggesting that males particularly male Type As may exhibit more behavioural and physiological reactivity than females.

Recently, several studies have investigated attitudes and beliefs of Type A vs Type B individuals when involved in a

variety of situations. For example, Furnham et al (1985) presented subjects with controllable and uncontrollable situations. It was predicted that Type B subjects would show clear differences in their reactions to controllable and uncontrollable situations, while Type A individuals would show less differentiation. Results showed that Type As and Bs differed mainly in their reaction to uncontrollable situations. Type As perceived more causal and more moral responsibility and reported more anger with themselves.

Tramill et al (1985) found that Type As had a much higher level of death anxiety and lower self esteem than Type B individuals. It was concluded that Type As may exhibit coronary prone behaviour pattern because of social insecurity. Fontane et al (1986), however, have reported the Type A behaviour pattern to independent of social insecurity.

Schwartz et al (1986) conducted an experiment to compare the task performance of Type A and B persons following failure on a task in which no one succeeded (universal failure) versus failure on a task in which others had succeeded (personal failure). Post failure performance was measured in terms of speed of completion of anagrams. Initial analysis indicated that the failure manipulation was effective in influencing subjects' perceived cause of their failure and that subjects were more anxious and depressed following

personal failure rather than universal failure. Furthermore, it was found that Type A individuals performed better following personal failure rather than universal failure, whereas, type of failure had no significant effect on the performance of Type B subjects. The authors suggest that contrary to what is thought, Type A individuals do not struggle for success indiscriminately.

Furnham et al (1986) examined how Type A and B subjects perceive themselves by analyzing self ratings of personality in Type As and Bs and differences in recall of positive and negative personality information. It was suggested that Type A behaviour is associated with a tendency to process information about the self in such a way as to bolster self esteem. Furthermore, Type A individuals tended to rate themselves much more negatively than Type Bs. It is concluded that Type As may have unrealistically high internal standards and may hold unrealistically high expectations of success, and it follows that they may suffer more disappointments and self doubt than Type Bs who assess their capabilities more accurately.

These studies clearly show distinct behavioural differences between Type A and B subjects. These differences exist not only in terms of what kind of behaviours are exhibited such as aggression or competitiveness but also in terms of feelings and attitudes to certain situational outcomes such

as attitudes towards self when failure occurs. The majority of these studies suggest that the Type A behaviour pattern may be strongly associated with social insecurity and fear of failure and that Type A characteristics are exhibited because of certain fears and anxieties.

Apart from behavioural differences, evidence suggests that Type A individuals differ significantly from Type B subjects in physiological responses. Some of these differences have already been outlined in this chapter. The evidence suggesting cardiovascular differences between Type A and B individuals will be outlined here.

A vast body of research exists concerning physiological differences between Type A and B individuals. These experiments mainly consist of studies on healthy subjects. Some studies have shown clear differences in cardiovascular reactivity between Type As and Bs (eg Ward etal 1986) while others have shown differences in some physiological aspects and not others (eg Van Schijndel etal 1984). Some studies have shown no significant differences in physiological reactivity between Type A and B individuals (for example, Lawler etal 1984). Generally, most of these studies support the notion that Type A individuals vary significantly from Type Bs in cardiovascular reactivity. In particular Type As have been found to exhibit excess cardiovascular and endocrine reactivity while working and during laboratory

induced stress (Lundberg et al 1987; Albright, Andreassi and Steines, 1988). Elevated cardiovascular activity has also been observed in Type A individuals following disappointment while performing a task in the laboratory (Perkins et al 1984). Similarly, excess heart rate and blood pressure increases have been reported in Type A adults, as compared with Type Bs, while placed in an unpredictable and uncontrollable situation (Evans and Moran, 1987). This pattern of heightened physiological arousal has even been reported in Type A children as young as six years old (Jennings and Matthews, 1984).

Since the main theme of the present research is concerned with competition, the role of competition in the Type A behaviour pattern will be dealt with here in some detail.

1.4.1.2 Competition and the Type A behaviour pattern

Competition or competitiveness has been identified as a major component of Type A behaviour pattern and most questionnaires measuring Type A include a competition sub scale (Glass, 1977).

Price (1983) has defined competition as a need to "seek or strive for something for which others are also contending. To vie with another or others for or as if for a prize". To compete may also indicate simply the fact of struggle to win

out over others or to continue to exist despite the strength and effort of others. This functional definition of competition indicates that there may be at least two fundamental incentives for competing. The first is to obtain some coveted prize and the second is recognition by others and the maintenance and reinforcement of self-pride. Price (1983) argues that in a highly industrialized society which values material gain, competitiveness above all else is strongly emphasized. A person who does not compete, on the other hand, is thought to be afraid of competition and is regarded by the society as being insecure about his ability to win. For this reason some researchers (for example, Price, 1983; Gasturf, Suls, and Saunders, 1980) have argued that low self regard may be a particularly salient aspect in that it may be antecedent to a person's hypervigilance (being constantly aware in evaluating others' performance on an important dimension). Similarly, Burke and Weir (1980) have found that Type A individuals Typically report continuing through out their working lives to "strive more and more for higher and higher positions even if it means leaving a position they enjoy or moving into a position for which they are not as well qualified." according to Burke and Weir, This obsession to compete and strive for higher positions in the work place may be tied to the possibility that Type A individuals may fear that they may be negatively evaluated and their self worth reduced if they do not try to achieve the highest possible merits.

Gasturf et al (1980) carried out an experiment with the hypothesis that Type As are more concerned with positive evaluation by others than Type B subjects. It has been shown in the social facilitation literature that performance is generally enhanced when performing simple tasks and impaired when performing complex tasks by the presence of others (Zajonc, 1965). Subjects in the Gasturf's experiment were asked to perform simple and complex tasks in the presence of co-actors. It was found that the presence of co-actors did not distract Type B subjects but Type A subjects were distracted on both simple and complex tasks in the presence of others. However, the presence of co-actors did in fact facilitate the performance of Type As on the simple task and impair their performance on the complex task. Type A individuals also reported significantly higher feelings of competition in the presence of both similar and superior co-actors than did Type B subjects.

There is further evidence that in competitive situations, individuals classified as Type A exhibit characteristic behavioural patterns which are distinctly different from those exhibited by Type B subjects. For example, Van Egeren (1979) conducted a series of controlled laboratory studies to investigate the notion that Type As are more competitive than Type Bs. In one study, Type A and B subjects took part in a computerized performance-based test of competitiveness.

Subjects interacted in pairs and had the opportunity to cooperate or compete during these interactions. Also they had the option of rewarding or punishing each others' actions. It was found that subjects classified as Type A were noticeably more punitive and competitive than Type B subjects. Interestingly when Type As interacted with Type Bs, the behavioural differences between the two groups largely disappeared.

Apart from behavioural evidence that Type A individuals are more competitive than Type Bs, there is ample evidence suggesting that there are distinct physiological differences between Type A and B individuals. These findings are generally similar to other studies done on the physiological differences between Type As and Bs. Despite some discrepancy concerning which physiological responses are elevated, most of this evidence shows that Type As exhibit elevated cardiovascular reactivity as compared to Type Bs (Glass and Karkoff, 1980; Jones 1985; Spiga, 1986; Lawler and Schmied, 1986). Further research is needed to highlight behavioural and physiological characteristics of Type As.

Some researchers have argued that Type A characteristics such as competition may become habitual behaviour patterns. Therefore, Type A persons may exhibit competitive behaviour even in the absence of challenges or appropriate stimuli. There is some evidence suggesting that people who adopt a

generally competitive orientation tend to view the motives of others as more homogeneously competitive than they really are (Kuhlman and Wimberley, 1976). Price (1983) argues that if competitiveness has become a person's habitual mode of approaching a task, the presence of an external competitor may become unimportant. Therefore, even without a challenge from his external environment, the Type A individual may begin to show competitive behaviour, providing positive or punitive consequences for his performance based on how well he does compared to some self established criterion. The notion that the Type A behaviour pattern may be a habitual characteristic has not been specifically examined and needs to be addressed in future research.

On the basis of the physiological and behavioural differences between Type A and B individuals, the Type A behaviour pattern has been the target of numerous treatment outcome studies which have attempted to actually alter the behaviour pattern both in healthy individuals and in patients suffering from CHD. These studies will be discussed since a major part of the present research is concerned with offering a new approach for the modification of physiological responses in healthy Type As.

1.5. MODIFYING THE TYPE A BEHAVIOUR

The major questions asked in attempting to modify the Type A behaviour pattern have been whether the behaviour pattern is modifiable and, if so, whether modification will reduce the incidence of CHD. Friedman, Thoresen and Gill (1981) hypothesized that the Type A behaviour pattern can not be altered in healthy individuals since they would not be sufficiently motivated. Therefore, a great deal of attention was originally directed towards patients suffering from CHD. Although as early as 1974 modification programmes have been used with Type A patients (Suinn, 1974; Thompson, 1976), the most ambitious attempt to alter Type A behaviour pattern among heart patients has been the Recurrent Coronary Prevention Project (RCPP) initiated by Friedman and his colleagues in 1982. Type A subjects who had had an MI at least six months before the beginning of the programme were recruited. All subjects were under 65 years of age, had been non-smokers for at least six months, and had never shown signs of diabetes. Subjects were divided into three sections: Section I (n=270) subjects were given detailed medical advice about CHD and strict control of traditional risk factors such as diet and smoking for a period of three years. Patients met regularly to discuss anxieties with a psychologist. Section II (n=592) patients were taught how to modify the Type A behaviour pattern in addition to medical advice. Patients in Section II were a) taught how to control physiological variables that could increase the risk of reinfarction, b) made aware of the kind of behaviour they

were exhibiting and alternative strategies for handling situations that elicit Type A behaviour were developed, c) were taught how to change their environment. For example, patients were taught how to seek help from their children or spouses to bring about changes in the environment, and d) treatment concentrated on cognitive or philosophical factors which encourage and sustain the Type A behaviour pattern. Patients in Section III (n=151) were given no treatment at all.

The results of the RCPP show that among subjects who completed the treatment, a new self report measure showed significantly greater reductions of the Type A behaviour pattern in Section II treatment group (from an average of 2.74 on a 1 to 5 scale to 2.14, more than one SD) than by the Section I group (2.69 to 2.40) (Friedman et al 1984). Furthermore, reports by spouses and co-workers and continuous assessment using a videotaped structured interview (Friedman and Powell, 1984) largely confirmed the results. More importantly, the three year cumulative rate of a recurrent cardiac event was lower among Section II patients (7.2%) than among Section I (13.2%) and Section III (14.0%) participants (Powell et al 1984).

The results of the RCPP are impressive. However, it is not known whether the changes effected during treatment will be maintained after the termination of the treatment programme

(Haaga, 1987). Furthermore, there is no indication of what aspects of the treatment programme were responsible for the effects observed. Further research should attempt to answer these questions.

The results of the RCPP have prompted many researchers to initiate modification programmes for healthy Type A individuals. Various techniques such as meditation (Muskatell et al 1984), Rational-Emotive therapy (Thurman, 1983; Woods, 1987), and Anxiety Management Training (Hart 1984) have generally reported limited success in reducing aspects of the Type A behaviour pattern. For example, hostility and anger have been successfully reduced in some Type A populations. Recent attempts at modification in healthy subjects has been reported by Ethel Roskies and her associates who have proposed a stress management programme for the modification of the Type A behaviour pattern in healthy individuals. This programme is comprised of twenty sessions during which subjects are taught deep muscle relaxation and are encouraged to keep records of variations in physical tension. Subjects are given "home work" assignments and are taught coping skills (Roskies, 1987). The use of this programme was reported in the Montreal Type A Prevention Project (Roskies et al 1986) where significant reductions in overall Type A scores were found. Subjects also reported greater life satisfaction at the end of the programme. However, there were no significant changes in

physiological responsivity. In fact, unlike studies conducted with Type A heart patients, research on healthy Type A individuals has been unsuccessful in reducing physiological activity, thought to be a major precursor to CHD (Haaga, 1987).

The hypothesis put forward by Friedman, Thoresen, and Gill (1981) suggesting that the Type A behaviour pattern can not be modified in healthy subjects is therefore rejected by the above studies. However, on the basis of some of the studies reporting failure to modify physiological responses, it may be argued that Type A is much more difficult to change in the healthy population than in patients already suffering from the adverse effects of Type A. This, however, does not mean that altering the Type A behaviour pattern in a clinical population is free of problems. As the researchers in the RCPP point out (Thoresen et al 1985; Friedman et al 1984) a major problem in any modification programme is the fact that subjects are reluctant or unable to change. This may be due to the fact that by the time a person has reached middle age, the behaviour pattern is established to such an extent that change becomes extremely difficult. Price (1983) has therefore suggested that modification programmes should aim at a much lower age group and attempt to educate children and adolescents about the negative consequences of the Type A behaviour pattern.

The modification programmes directed at healthy Type As have only been successful in terms of reducing self reported Type A behaviour and increasing life satisfaction in subjects. The danger of relying on self report measures as the only index of Type A will be discussed in future sections. Nevertheless, there are some problems with these studies. Firstly, there have been no follow up studies to indicate a) whether the effects of Type A change are sustained long term after the termination of the programme, and more importantly b) whether these modification programmes have any clinical utility in terms of reducing the incidence of CHD. Furthermore, most of these studies have failed to show reductions in actual physiological reactivity in subjects.

Since it has been argued that the Type A behaviour pattern is maintained in part by lack of awareness of its long term negative consequences (Price, 1983; Rosenman, 1978), repeatedly drawing subjects' attention to the physiological processes that may be associated with the behaviour pattern might go far to help motivate people to reduce their Type A behaviour. The most successful studies which have reported significant reductions in physiological activity of healthy type As have used biofeedback training as part of the modification programme (Prior et al 1983; Stern and Elder, 1982). It must therefore be noted here that any modification programme designed for healthy Type As should consider the following points: a) there should be follow up studies to

clarify the effects of altering Type A in terms of subsequent incidence of CHD, b) emphasis should be placed on physiological as well as cognitive factors, and c) because healthy Type As are unwilling to alter their behaviour pattern and may even be unaware of the consequences of their behaviour, new challenges and incentives should be provided to motivate them, and d) programmes should be designed to alter the type A in the younger population before the behaviour pattern becomes fully established.

1.6. TOWARDS A THEORETICAL MODEL OF TYPE A

Despite the fact that Friedman and Rosenman, in their original conceptualization of Type A behaviour pattern, seriously attempted not to portray Type A as an underlying trait, many medical investigators and other scientists have viewed or labelled Type A, directly or indirectly, as comprising several underlying traits. Trait refers to relatively stable, and highly consistent inborn personality dispositions (Hilgard and Bower, 1975). Traits are considered "intrapsychic causes of behavioural consistency" across situations, and are inferred from the behaviour they are thought to determine (Mischel, 1973). For example striving to beat a rival in contest is thought to be caused by the person's underlying trait of competitiveness, rather than being seen as perhaps a function of situational and other influences. Mischel (1968) argues that trait theories

do not provide information about the conditions that may affect the acquisition of certain behaviours, nor are they concerned with factors that encourage or discourage the continued expression of the behaviours in question. Yet it is precisely these considerations which must be taken into account if we hope to develop a clinically useful theory of the Type A behaviour pattern.

Despite such severe criticism trait theory has played a prevalent role in theoretical models of Type A behaviour until very recently. Some of the more prominent models will be discussed.

1.6.1 The Mechanistic Interactional Approach

The predominant approach to this area describes overt Type A behaviour as a characteristic style of responding to certain classes of stimuli, for example, challenges, demands, threats to control. The expression of Type A behaviour is in turn associated with enhanced sympathetic nervous system activation. Hemodynamic stresses, such as increased blood pressure (BP), and circulatory catecholamines associated with sympathetic arousal are believed to produce initial injuries to the linings of coronary arteries, creating the site for subsequent development of atherosclerosis or the blockage of arteries by plaque and fatty deposits. Thus, physiological reactivity may create the site and provide the

"raw materials" for coronary artery disease (CAD).

Furthermore, physiological reactivity may precipitate acute clinical manifestations of CHD, such as angina and myocardial infarction (Williams, 1975). Thus, behaviours and physiological responses are seen as co-effects of the same cause- a situationally activated, constitutionally based predisposition towards autonomic nervous system (ANS) reactivity.

Kahn et al (1980) and Krantz et al (1982) have demonstrated that Type A individuals undergoing coronary artery bypass surgery, in comparison with their Type B counterparts, show elevated blood pressure during but not prior to surgery (that is, under general anesthesia). These findings have suggested the possibility that Type A persons' reactivity does not require conscious mediation. Instead it may reflect an underlying constitutional factor.

Krantz et al (1982) argue that a biological model suggests that Type A behaviour pattern itself in part reflects excessive sympathetic response to environmental stressors. Perhaps, excessive or continuous elicitation of sympathetic responses over the course of a life span, may both enhance the expression of Type A behaviour and predispose the individual to clinical coronary disease as well.

This biological approach suffers from potentially serious limitations which will be discussed. Though a biological rather than a psychological trait is given primary importance, stable traits according to this model are activated by relevant situations. Little attention is given to how these situations are themselves influenced by psychological and social correlates of the Type A behaviour pattern. Further, by concentrating mainly on biological factors, psychological factors are largely ignored.

Several variations of this general model of situationally elicited Type A behaviour have been proposed. Under the influence of Seligman's (1975) learned helplessness model, Glass (1977), has suggested that overt Type A behaviour pattern represents an attempt to assert and maintain control over stressful aspects of the environment. Matthews and Spiegel (1983), on the other hand, have suggested that Type A behaviour pattern is a result of a strong value in productivity and achievement combined with ambiguous standards for evaluative achievement. Thus, situations which provide opportunities for achievement but lack explicit standards, are likely to elicit the aggressive striving typical of Type As.

The general model proposed by Glass (1977) and Matthews and Spiegel (1983) suggests that Type A individuals display competitive, hostile, impatient, and achievement oriented

behaviour, as well as autonomic arousal, only when confronted with challenging or demanding situations. However, according to some critics of the mechanistic interactional model, Type A tendencies and the relevant situational characteristics are viewed as independent, static sets of variables (Smith and Anderson, 1986). These implicit assumptions contain an important limitation. It is likely that Type As rather than simply reacting to challenges and demands, actively seek and create them. Furthermore, it is likely that challenging and demanding environments, rather than simply eliciting Type A behaviour from predisposed individuals, also reinforce and maintain it. Thus, "traditional approaches to Type A behaviour pattern and reactivity seem to lack acknowledgement of dynamic, reciprocal relationships between behaviour and relevant situations" (Smith and Anderson, 1986).

1.6.2 The Biopsychosocial Interactional Model

Recently, a new approach based on the cognitive social learning theory has been proposed to explain Type A behaviour pattern (Price, 1983; Smith and Anderson, 1986). The new approach highlights the influence of Type A individuals on their environment as critical for understanding the link between behaviour and disease. Cognitive social learning theory (Bandura, 1977; Mischel, 1973) provides a framework for examining such reciprocal relationships between persons

and situations. Rejection of unidirectional causal models and the explicit statement of reciprocal causality is an integral part of this theoretical orientation. The cognitive social learning theory maintains that behaviour partly determines which of the many potential environmental influences will come into play and what forms they will take. Environmental influences , in turn, partly determine which behavioural repetoires are developed and activated (Bandura, 1977).

Although there seems to be some evidence for a genetic basis for the Type A behaviour pattern mainly based on twin studies (Pedersen etal 1989; Matthews etal 1984), the cognitive social learning model is concerned mainly with the social and environmental development of the Type A behaviour pattern. To explain the development and maintenance of Type A behaviour, Price (1983) has proposed a social learning framework in which cognitions and personal beliefs form the core. These personal beliefs foster a variety of fears and anxieties, and together the beliefs and fears sustain and promote Type A behaviour pattern. Thus, Type A develops to help individuals cope with fears and anxieties associated with particular beliefs they have about the environment. Price suggests that the behaviours and attitudes typically associated with Type A behaviour derive from cognitive factors which are the result of sociocultural values being communicated to individuals through the family, school, and

the mass media. Participation in an achievement oriented, materialistic society develops particular beliefs which in turn generate fears and anxieties that promote and maintain the Type A behaviour pattern. Price's theoretical framework will be discussed in more detail in chapter five.

Smith and Anderson (1986) have gone one step further and proposed the following biopsychosocial model of Type A. Smith and Anderson (1986) hypothesize that through cognitive and overt behaviours, Type A individuals construct a subjective and objective environment rich in those classes of stimuli known to elicit enhanced physiological reactivity. This approach differs from the previous ones because it emphasizes the point that the Type A behaviour pattern represents an ongoing process of challenge and demand engendering behaviour. Hence, Type A individuals do not simply respond to challenges and demands; they actively seek and create them through their cognitions and actions. This constructed environment also elicits and maintains further Type A behaviour.

To date the biopsychosocial interactional theory seems to be the most promising explanation for the development and maintenance of the Type A behaviour pattern. The model seems to be a useful theory about how Type A is elicited and maintained, though research is needed to test various assumptions made by the theory. For example, it is necessary

to validate the beliefs and fears against reliable Type A measures. Burke (1984) developed a pencil and paper measure of these beliefs and fears and validated them against a standardized Type A measure, the JAS. The findings suggest that the beliefs and fears had a generally acceptable level of internal reliability. However, these results need confirmation. Furthermore, the relationship between the beliefs and fears proposed by Price's theory and the Type A behaviour pattern should be investigated with different populations. For example, the development of beliefs and fears has not yet been investigated. This could open new doors to our understanding of the etiology of the behaviour pattern which according to Price begins in childhood or adolescence. A major part of the present research is concerned with this issue.

1.7. ASSESSMENT OF THE TYPE A BEHAVIOUR PATTERN

The first technique to identify and screen Type A individuals was developed by Friedman and Rosenman in the mid 1950's in the form of a structured interview (SI). The interview is designed to reveal the presence of certain attributes that characterize the Type A individual. These include excessive drive for achievement and advancement, aggressiveness, vigor, a chronic sense of time urgency and a habitual tendency to increase the speed of thought and action (Friedman and Rosenman, 1959). The interview consists

of twenty two questions and interviewees are asked about their characteristic ways of responding to a variety of situations which might elicit a Type A response. The interview is videotaped and the interviewer can assess Type A behaviour from the manner in which questions are answered as well as the content of the answers. On the basis of this analysis, each individual is classified into one of five categories: Type A1 if the individual exhibits Type A behaviour pattern in its most developed form, Type A2 if the Type A is exhibited to a lesser degree, Type B4 if Type B behaviour is shown fully and Type B3 if Type B is exhibited to a lesser degree. If an individual is thought to exhibit approximately equal amounts of Type A and B behaviour, a Type X classification is made.

However, researchers have used different scales based on this original form. Therefore, care must be taken when considering the reliability of classifying individuals as Type A or B. The interscorer agreement using different scales varies from 75% to 90% (Dembroski, 1978). Test retest reliabilities are reported at being 80% in the WCGS with a period of 12 to 20 months elapsing between testings and a single A/B dichotomous scaling being used; but the reliability is only 64% for the four point scale (Jenkins etal 1968).

The SI seems to be the best measure of Type A available to researchers and in fact most other measures of Type A are validated against the SI. However, there are some disadvantages. The main problem with the structured interview is that the technique results in a subjective and holistic assessment of Type A behaviour pattern exhibited by the subject during the interview. There are no objective, quantitative criteria for making the assessment (O'Looney, 1984). Furthermore, the interview procedure is time consuming and expensive. The carrying out and interpretation of the interview requires a period of specialized training, making the acquisition of the technique costly and unsuitable for large scale use.

Because of these problems, a variety of self report pencil and paper questionnaires have been developed. These questionnaires attempt to provide a convenient, cost effective measure of the Type A behaviour pattern suitable for use in industrial and epidemiological studies. The measures devised so far include the Jenkins Activity Survey or JAS (Jenkins etal 1965), the Framingham Type A Scale (Heynes etal 1978), the Bortner Scale (Bortner, 1969), and the Eysenck Type A Scale (Eysenck and Fulker, 1983).

The degree of agreement between the JAS and the SI is reported to be around 65% (Jenkins etal 1965). The JAS has also been found to be a good discriminating instrument

between healthy subjects and groups of patients with CHD (Jenkins et al 1971). However, the JAS is reported to misclassify a large proportion of subjects classified as Type A or Type B by the SI and agrees with the classifications made by the SI in about 60 to 70 % of the cases (Jenkins, 1978). As Matthews (1978) has pointed out, the agreement rates by chance should be 50%, since 50% of the WCGS sample were classified as Type A1 or A2 by the interview. Therefore, the JAS agrees with the SI classification of middle aged, middle class men at 10 to 20% above chance level.

A large number of editions of the JAS have been developed and care should be taken to establish which forms have been used and how they differ before comparisons between studies are made. The JAS has been validated using a population of employed, white collar, middle aged American men. To date, several studies have reported on the validity of the questionnaire when a student sample are used (Yarnold et al 1988) and although the cross cultural validity and generalizability of the JAS has been established by several researchers (for example, Rustin et al 1976 in Belgium; Appels et al 1982 in Holland), O'Looney (1984) concludes that the validity of the JAS remains questionable when the questionnaire is used with men and women of different ages, socio economic classes and ethnicity.

The Framingham Type A Scale comprises of ten questions and has been validated against two other predictive techniques in measuring Type A behaviour pattern. MacDougall et al (1979) used three subscales of the Framingham ten item questionnaire and found a correlation of 0.32 (0.39 for females and 0.25 for males) with the JAS. An even lower correlation was found when the Framingham scale was compared with the SI.

Haynes et al (1980) found higher correlations when comparing the Framingham scale with the JAS Type A subscales (0.53 with the competitive, hard driving subscale, and 0.64 with the speed and impatience subscale). When compared with the ratings made by the SI assessment method, 60% agreement was found.

Although the Framingham Scale has all the advantages of a questionnaire in terms of convenience and price and has been prospectively related to CHD in a wider population than the JAS or the SI, it suffers from the problem that it is possibly too short to adequately discriminate Type A from Type B individuals, and the response alternatives to the questions are extremely limited (O'Looney, 1984).

The Bortner Scale consists of 14 rating scales (Bortner, 1969). It was originally conceived of as a crude index of pattern A behaviour. Indeed, in validating the scale in

1969, Bortner decided to use only 77 heart patients and factor analysis was based on data collected from a very limited sample. Often, seven out of the original 14 items are used as a short alternative form of the scale. The 14 item scale and the seven item scale are found to display 64% and 65% agreement with the SI classification respectively (O'Looney, 1984).

A comparison of the Bortner scale and the SI in Belgium (Rustin et al 1976) found that there was agreement in 75% of the cases. Correlations of 0.68 and 0.71 were also found between the assessments made on two different occasions by the JAS and the Bortner Scale in Britain (Johnston and Shaper, 1983).

Although the Bortner Scale has been shown to discriminate successfully between Type A and B individuals as defined by the SI (Robinson and Heller, 1980), its predictive value as far as CHD is concerned is questionable. For example, Heller (1979) found that the Bortner Scale could significantly discriminate between healthy men and men who had suffered from CHD. However, Koskenvou et al (1983) reported the Bortner Scale to have no predictive validity for mortality from CHD.

The Eysenck Type A scale was developed by Eysenck and Fulker (1983). The authors argued that the proponents of Type A

behaviour pattern have neglected the problem of the possible relationship between Type A and established dimensions of personality. It is suggested the description of the Type A behaviour pattern seems to relate it closely to both the extraversion (E) and neuroticism (N) dimensions as measured by the Eysenck Personality Questionnaire (EPQ) designed by Eysenck and Eysenck (1975).

Eysenck and Fulker drew up a 34 item questionnaire based on these hypotheses. The questionnaire was administered to 1082 volunteers. Four factors were extracted which were similar for males and females and labelled as tenseness, ambition and competition, activity, and unrepressed. The reliabilities for the first three factors were significant (0.74, 0.61, 0.50 respectively) and inadequate for the fourth factor (0.41). As expected, tenseness correlated significantly with neuroticism and ambition and competition with extraversion. The Eysenck Type A Questionnaire has been found to correlate significantly with one other Type A measure, namely the Bortner Scale. for example, Furnham et al (1985, 1986) have reported correlations of 0.49 and 0.66 with the Bortner Scale.

The Eysenck Scale is a convenient questionnaire to use with large populations since it is short and easy to score. However, the scale has not been used for assessing Type A as much as other established Type A measures such as the SI.

Furthermore, the validity of the scale in terms of predicting CHD is unknown.

The limitations faced by self report measures of Type A pose a serious threat to an objective measurement of the behaviour pattern. More recent research has shown conflicting evidence as to the validity of these self report measures in predicting CHD. Furthermore, validity and reliability figures for some of these measures are unavailable.

Herman et al (1986) found a discrepancy between questionnaire scores and interview scores of Type A behaviour pattern in 281 patients referred for diagnostic coronary angiography. Two distinct groups were identified by the authors: Type A subjects whose Type A self ratings were consistent with the SI classification (n=43), and subjects who obtained low self rating scores but were classified as Type A by the SI (n=33). The authors maintain that the results emphasize the dangers of subjective assessment of the behaviour pattern. Condon (1988) developed a 46 item spouse questionnaire which aimed at measuring Type A behaviour pattern by asking questions about the individual from his or her spouse. A modest correlation between the spouse questionnaire and self report measures of Type A was found but the author concludes that individuals' self reports were not as accurate as those made by their spouse and it seems that Type A individuals

tend to under estimate the extent to which they exhibit Type A behaviour pattern.

Price (1983) argues that in view of these problems future research should concentrate on designing objective yet cost effective measures which would ultimately enhance and facilitate the assessment of the Type A behaviour pattern. This is particularly important since some researchers (eg Rosenman, 1978; Byrne et al 1986) have argued that Type A individuals have a lack of insight into their behaviour and many believe that they lack the Type A qualities, which they excessively exhibit. If this is the case, then self report may not be an ideal means of assessment for the Type A behaviour pattern. The task for future researchers is the search for an objective method for the assessment of Type A. The validity of any objective measure of Type A in terms of its ability to predict CHD must be established.

Despite the above criticisms, some of the present assessment devices seem to offer certain advantages which make their use particularly desirable. Three questionnaires offer particular advantages; therefore, they will be used to assess Type A in the present research. These are the Bortner Scale, the Eysenck Type A scale and the Jenkins Activity Survey (student version). All of these questionnaires are easy and convenient to administer and score, particularly with large populations. Although the Eysenck, and to a

lesser extent the Bortner have not been used very often in Type A research, their use has been recommended by some critiques (eg O'looney, 1984) because the scales are short and simple to administer.

1.8. CONCLUSIONS

The present chapter was concerned with outlining a general overview of the development of the concept of the Type A behaviour pattern. It was pointed out that despite the ongoing debate concerning the actual relationship between the Type A behaviour pattern and CHD, a vast amount of research has emerged during the last two decades which has investigated various dimensions of Type A. For example, several theories have been proposed and tools for the assessment of the behaviour pattern have been devised. Furthermore, distinct behavioural and physiological differences have been reported between Type A and B individuals. Attempts at modifying the Type A behaviour pattern were outlined and directions for further research were discussed with reference to modification programmes for healthy Type As. This is the ultimate aim of the present research. Subsequent chapters will describe experimental work which will outline a method for modifying physiological reactivity in healthy Type As using heart rate biofeedback.

It was pointed out in the present chapter that research is needed to clarify several issues as far as behavioural and physiological differences between Type As and Bs are concerned. For example, the notion that the Type A behaviour pattern may be a habitual characteristic, that is the question of whether or not Type As would exhibit Type A behaviour without the presence of external cues has not been specifically examined. Thirdly, the differences reported between Type As and Bs may have several important practical implications. These implications seem to have been largely ignored by researchers. The purpose of the next chapter will be to highlight behavioural and physiological characteristics of Type As during competition. These characteristics will be discussed in terms of their implications for the assessment of Type A, predicting driver performance and the modification of the behaviour pattern.

CHAPTER TWO

TYPE A BEHAVIOUR PATTERN: THE EFFECTS OF COMPETITION ON HEART RATE AND "DRIVING STYLE".**2.1. INTRODUCTION**

Despite the fact that the Type A behaviour pattern has been defined differently by different authors, some elements seem to represent the core of most definitions. As pointed out in the previous chapter, the characteristics most commonly associated with the Type A behaviour pattern are competitiveness, time-urgency, aggressiveness, and drive and achievement striving.

There is considerable evidence that distinct behavioural and physiological differences exist between Type A and B individuals when involved in competition (Ward, 1986; Jones, 1985; Lawler and Schmied, 1986). Furthermore, some researchers have argued that Type A characteristics such as competition may become habitual so that the Type A individual may need no external or environmental cues for exhibiting behaviours such as competition (Kuhlman and Wimberley, 1976). The aim of this experiment is to demonstrate these differences using three different measures of competitiveness: driving style, heart rate and self report. In doing this, it may be possible to illuminate the

role played by the Type A behaviour pattern in driving style and the possible implications of Type A assessment in predicting driver performance.

Some recent research in the field of road safety shows that behaviours similar to the Type A behaviour pattern are found to be exhibited by car drivers who have a worse driving record and tend to cause more accidents and commit more errors on the road. For example, it has been found that sensation seeking, aggression, risk taking and high self esteem are salient characteristics in drunk drivers in the United States (Mookherjee, 1986) and non-compliant drivers in Austria (Hofner, 1982). For example, Rioux and Wapner (1986) in a study of drivers who did not use seat-belts and committed seat-belt users and Jona (1986) in a study of driving violation conclude that drivers who are more likely to take risks and who think of themselves as being in control tend to cause or be involved in more accidents.

Some hypotheses have been put forward by researchers in the field to account for aggressive driving by some individuals and by Type As in particular. For example, Marsh and Collett (1987) have discussed the automobile as a means of expressing anger, aggression and frustration. The authors describe the notion of the car as a special kind of territory and outline forms of territorial defense and territorial invasion. From this point of view, they suggest

that individuals make most use of the car as a weapon especially when it is their only personally owned territory.

Perry (1986) studied driving style in Type A individuals. Perry argues that Type As are slow discriminators because they have greater response latencies in both simple and complex choice reaction times. Perry aimed to examine the relationship between the Type A behaviour pattern and the incidence of automobile accident and violation. It was found that Type A subjects as a whole, and Type As with a chronic sense of time urgency, in particular were involved in more accidents and received more tickets for driving violation. Perry concludes that it seems that the patience required to operate a motor vehicle increases the tension in the Type A person and this results in slower discrimination.

Panek and Wagner (1986) have found that some subscales of certain personality tests such as aggression and directive behaviour and some personality traits such as extraversion and neuroticism can be used successfully to predict driving performance such as number of moving violations. To date, one study has directly examined the role of the Type A behaviour pattern in relation to driving performance. Hence, there seems to be some evidence that at least some aspects of the Type A behaviour pattern may be related to driving violation.

2.1.1 Hypotheses of the present study

The present study concentrates on the importance of competition in Type A behaviour pattern. Type A and B subjects will be asked to drive a scalextric model car. Subjects' driving style will be measured during a practice trial where competition is not mentioned and during a competition trial where subjects will be competing for a prize. Furthermore, heart rate changes will be monitored during a rest period, the practice trial and the competition phase. Subjects' ratings on their desire to win the competition will also be collected as subjective reports.

The following hypotheses will be examined:

1. Type A subjects will show a more aggressive driving style, will drive faster and will be involved in more crashes than Type B subjects.
2. An even greater difference in aggressiveness and erroneous driving style may be predicted for Type A subjects when competing than when not competing.
3. Type A subjects will show a higher heart rate than Type Bs.
4. In the presence of an objectively competitive situation, Type As will show an even greater increase in heart rate.

5. It is predicted that Type As will express a greater desire to win the competition than their Type B counterparts.

6. Finally, the role of sex differences will be investigated in the present study. Unfortunately, research on sex differences in Type A behaviour has been mixed (see chapter one). However, research seems to indicate that males may be more competitive than females. In the present experiment it is predicted that male subjects will show a greater increase in heart rate and a more aggressive driving style when involved in competition than females.

2.2. METHOD

2.2.1 Subjects

The study was carried out using forty undergraduate students at London University (mean age was 20.7 years, age range was 18 to 29 years). Subjects reported to having had little or no experience with scalextric. The majority of subjects were informed about the experiment by letter and were asked to participate in a "psychology experiment". Some subjects were approached in person and were asked to take part in the experiment. Each subject completed one questionnaire measuring the Type A behaviour pattern, the Eysenck Type A

Questionnaire (see chapter one and appendix 1). Subjects scoring 23 and above out of a maximum of 34 on the Eysenck were labelled as Type A and 17 and below were labelled as Type B. This was roughly one standard deviation above and below the mean. There were 13 Type A (6 males 7 females mean age 21.2) and 13 Type B (7 males and 6 females mean age 20.5) subjects. Each subject was paid £2. Data for all forty subjects and for the 26 extreme scorers were analyzed separately.

2.2.2 Design

For "driving style" measurements a 2X2X2 factorial design was employed which included two between subjects factors: behaviour pattern (Type A and Type B) and sex (male and female) and one within subjects factor: competition (practice and competition). The dependent variables were erraticness, time and error (see below). For heart rate measurement a 2X2 factorial design was used with behaviour pattern and sex as between subjects factors and changes in heart rate as the dependent variable.

2.2.3 Apparatus

2.2.3.1 Heart rate analysis

This section provides a brief outline of various methods of heart rate analysis so that the reader is informed about the methods of measurement used in the present research.

Analysis of heart rate varies considerably and how data are analyzed depends on the needs of particular experiments (Schneiderman, Dauth and Van Dercar, 1974). Furthermore, heart rate analysis in biofeedback studies has largely depended upon how heart rate has been recorded. Various ways of measuring and recording heart rate include counting heart beats in a specified time with the use of a transistor driven electro magnetic counter (Brener, 1967); heart rate averaging where a current proportional to the rate of input pulses is produced by converting interbeat intervals (IBI's) into heart beats so that continuous graphic records of average heart rate is provided (Earnshaw, 1956); and beat to beat records where immediate information about short term changes in heart rate is provided using a digital elapsed time meter (Tove and Czekajewski, 1964).

The last two methods of recording have been most popular in recent years. Most researchers analyze beats per minute (BPM) and, particularly in biofeedback experiments, elapsed time meters are used where BPM's across time are analyzed (Stern and Elder, 1982; Stern, etal 1972). Variations,

however, exist where analysis of changes in heart rate over a specified time (Engel and Hansen,1966) or analysis of raw IBI's is reported (Brener and Hothersall,1967).

2.2.3.2 Heart rate measurement

In the present experiment, heart beat signals were measured by two silver/silver chloride, pre gelled electrodes with self adhesive foam backing (type number R 00 S from Cambic instruments). The electrodes were pre gelled to facilitate contact and improve electrical conductance. The electrodes were attached to an electrocardiogram (ECG) machine. A BBC microcomputer was programmed to receive heart beat signals from the ECG (for a circuit diagram of the out put to computer refer to appendix 2). The computer was also programmed to translate continuous interbeat intervals (IBI's) into beats per minute and to provide these heart beats every five seconds via a matrix printer (Epson LX-80) which was attached to the computer (refer to appendix 3).

2.2.3.3 measurement of "driving style"

A scalextric racing car (12 cm in length) was used for driving around a "figure of eight" model race track (3 meters in length). Electricity at low voltage was picked up by two copper brushes under the front of the cars. The car became mobile when the subject pressed on a hand held

accelerator. The movements of the accelerator made by the subject were used to measure erraticness. This was measured by means of a direct writing pen recorder (Washington 400 MD1 Oscillograph) which was attached to the accelerator and measured its movements in the form of a moving line on graph paper 10 cm in width. The pen recorder also indicated time in seconds on the graph paper (for a diagram of the scalextric car set up refer to appendix 4).

2.2.4 Procedure

Subjects were taken to a soundproof, windowless laboratory and asked to sit on a comfortable chair facing the race track. Subjects were run individually and each subject was asked to fill in the Eysenck Type A questionnaire. The time of the presentation of the questionnaire was altered for each subject. In other words, the questionnaire was presented before the experiment to one subject and after the experiment to the next.

Subjects were told that they would be required to drive the car around the track while their heart rate was being measured. Each subject was then given a full description of how heart rate would be measured and electrodes were attached. One electrode was placed on subject's wrist on the non dominant hand so that the dominant hand would remain free to hold and use the accelerator. One electrode was

placed on subject's inner foot between the ankle and the heel of the foot. Subjects were told to sit comfortably and relax for two minutes so that a baseline heart rate could be obtained. The average heart rate for the two minutes was recorded as "heart rate at rest" for each subject.

Following the baseline measurement, subjects were given the following instructions by the experimenter: "I would like you to drive the car around the track by using the hand held accelerator. This is just to give you a chance to get familiar with the track and to get some practice. So just get a feel of the track. This practice session is necessary because the task involves a certain amount of skill and judgement to avoid crashing the car and to score a respectable lap time. I will call out the number of laps you complete at the each of each lap. After 15 laps, I will ask you to stop. If you crash and fall off the track, I will simply put the car back on the track and you can continue straight away." Following these instructions, the experimenter activated the printer and the pen recording and asked the subject to begin the practice session. The average heart rate during practice, the pen recording of the accelerator movements, time in seconds, and number of times the car fell off the track were recorded.

After the practice session was completed one minute rest period was given after which the following instructions were

given by the experimenter: "Now that you have had some practice, I would like you to drive the car around the track again and complete 15 laps as before. But this time I would like you to complete the race as quickly as possible. To give you some incentive, there is a £10 prize for the fastest overall time and you are competing against thirty nine other people." The following instructions were added before the start of the race: "During the race there will be exactly the same procedure as in the practice session. But this time, every time you fall off the track or crash the car, I will add three seconds to your overall time. So, there will be a three second penalty for each crash". Subjects, therefore, completed 15 laps in a competitive situation. The average heart rate during competition, the pen recording of accelerator movements, time in seconds without penalties for crashes (during both practice and competition time taken by the experimenter to replace the car was taken into account), and the number of crashes during competition were recorded.

A self report rating scale was presented at the end of the experiment. Subjects were asked how much they wanted to win the competition, on which zero represented "not at all" and twenty represented "very much."

At the end of the experiment, the experimenter disconnected the subject from the electrodes and pointed out that the

winner will be announced when all participants have completed the experiment. subjects were paid £2 each for their participation in the experiment.

2.2.5 Data preparation

The average heart rate during practice was subtracted from average heart rate during rest to give a measure of heart rate change from rest to practice (R-P). Likewise, the average heart rate during competition was subtracted from the average heart rate during practice to give heart rate change from practice to competition (P-C). Finally, heart rate change from rest to competition (R-C) was measured by subtracting average heart rate during competition from average heart rate at rest.

Three different aspects of "driving style" were used for analysis: firstly, time in seconds taken by each subject, as indicated by the pen recorder, to complete the 15 laps during practice and competition.

Secondly, erraticness was measured for each subject during practice and competition. The pen recordings of the accelerator movements were in the form of a continuous line comprising of peaks, spikes and troughs. The length of this trace was calculated for the first 90 seconds of driving during practice and competition. A map measure was used for

calculating erraticness; This is normally used to measure distance on survey maps. It is a circular instrument with a small wheel at the bottom. the route or distance to be measured is then traced with the wheel. A pointer will then indicate on the dial the exact distance measured in centimeters.

The third measure of "driving style" was the number of times each subject had crashed and/or had gone off the track during practice and competition.

The following data were collected in this study:

1. Scores on the Eysenck Type A questionnaire
2. Changes in heart rate:
 - a. rest to practice (R-P)
 - b. practice to competition (P-C)
 - c. rest to competition (R-C)
3. "Driving style" measures for practice and competition.
 - a. time in seconds
 - b. erraticness in cm
 - c. number of crashes
4. Ratings of desire to win the competition. Zero represented not at all and twenty represented very much.

2.3. RESULTS

2.3.1 Overall analysis

A series of Pearson product-moment correlation coefficient were computed between the scores on the Eysenck Type A questionnaire and measures of heart rate change and "driving style". The Type A scores correlated significantly with all three measures of heart rate change (R-P $r = 0.29$ $P < 0.05$ P-C $r = 0.35$ $P < 0.05$ R-C $r = 0.44$ $P < 0.01$ $df = 38$).

As far as driving style measures were concerned, the correlation between the Eysenck and erraticness reached significance level during both practice and competition (0.29 and 0.40 respectively) suggesting that the higher the Type A score, the more erratic the "driving style" during practice and competition. The relationship between time and Type A score was not significant during practice. The same pattern can be observed for errors committed during practice. However, a significant negative correlation between time and the scores on the Eysenck scale during competition ($r = -0.28$ $P < 0.05$) indicates that the higher the Type A score the faster the time of the car during the competition phase of the experiment. Also there was a significant correlation between scores on the Type A measure and the number of crashes during competition ($r = 0.33$ $P < 0.05$).

There was also a significant correlation between scores on the Eysenck Type A measure and ratings of the desire to win by subjects ($r= 0.69$, $df= 38$, $P<0.01$). Subjects expressed a greater desire to win the competition as scores on the Type A measure increased.

Finally, related t-tests were carried out to see whether or not overall differences existed between practice and competition. Significant differences were found between erraticness during practice and competition ($t= -4.04$, $df= 39$, $P,0.01$) and between time during competition and practice ($t= 4.21$, $df= 39$, $P<0.01$). However, no significant differences were found between errors during practice and competition. The same pattern was observed for heart rate changes from rest to practice (R-P) and from practice to competition (P-C). Furthermore, there were no significant overall sex differences.

2.3.2 Results for Type A and B subjects

Data for twenty six extreme scorers on the Eysenck Type A scale were analyzed in order to compare Type A and B subjects. An initial t-test was carried out on average baseline heart rate (i.e. heart rate at rest) for Type As and Bs (mean heart rate for Type As was 81.02 $sd= 8.39$ and for Type

Bs was 78.32 sd= 9.37). The difference was not found to be significant.

Analyses of Variance (ANOVAs) were carried out using the statistical computer package BMDP.

Table 1 : Mean changes in heart rate and SDs for Type A and B subjects.

	Type A		Type B	
	Mean	SD	Mean	SD
Rest-Prac	5.30	2.53	2.91	2.33
Prac-Comp	5.75	1.39	3.49	2.67

note: the values above represent average beats per minute (BPM) increases from rest to practice and practice to competition

Three two way ANOVAs with two between subjects factors (Type and sex) were carried out on each measure of heart rate change.

**Table 2 : ANOVA summary table for changes in heart rate
(Rest to Practice)**

FACTOR	ss	df	ms	F	p
Type	26.344	1	26.344	4.16	0.05
Sex	22.858	1	22.858	3.61	0.07
TypexSex	0.197	1	0.197	<1	ns
Error	139.30	22	6.332	-	-

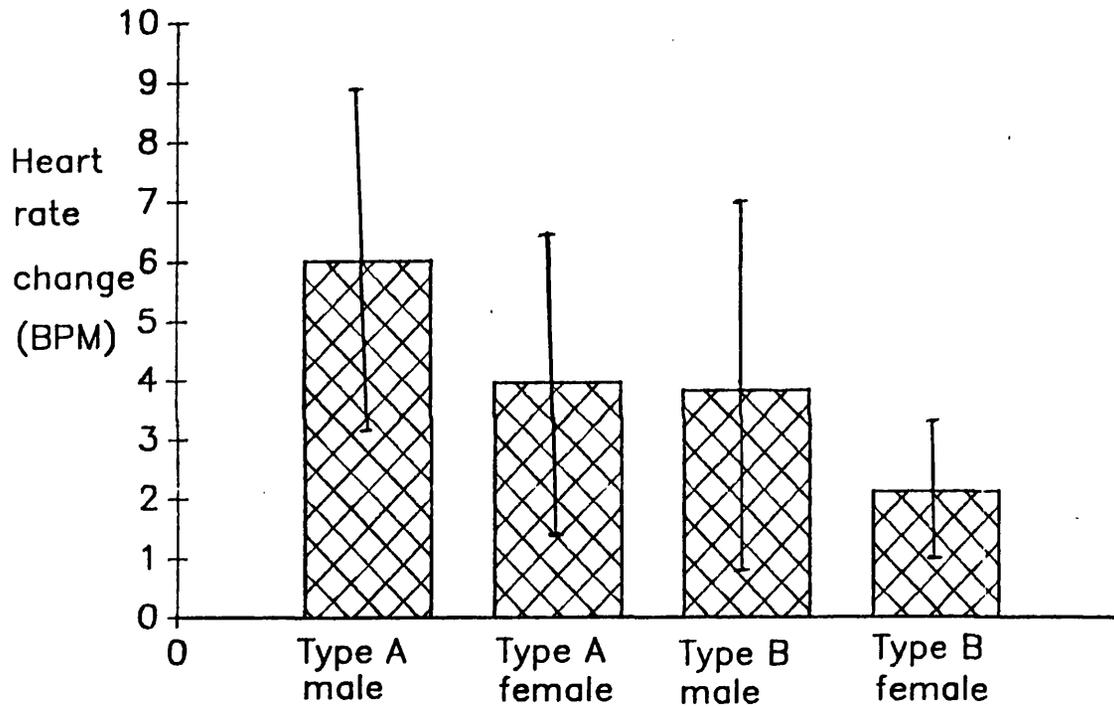
Table 3: ANOVA summary table (Practice to Competition)

FACTOR	ss	df	ms	F	p
Type	44.117	1	44.117	9.39	<0.01
Sex	16.514	1	16.514	3.52	0.07
TypeXSex	0.005	1	0.005	<1	ns
Error	103.338	22	103.338	-	-

As can be seen from tables 2 & 3, there is a significant main effect of Type in both indices of heart rate change. Hence, overall, Type A subjects increased heart rate significantly more than Type B subjects from rest to practice and from practice to competition. There was no significant sex difference in heart rate change from P-C and R-P, although in the latter analysis there seems to be a

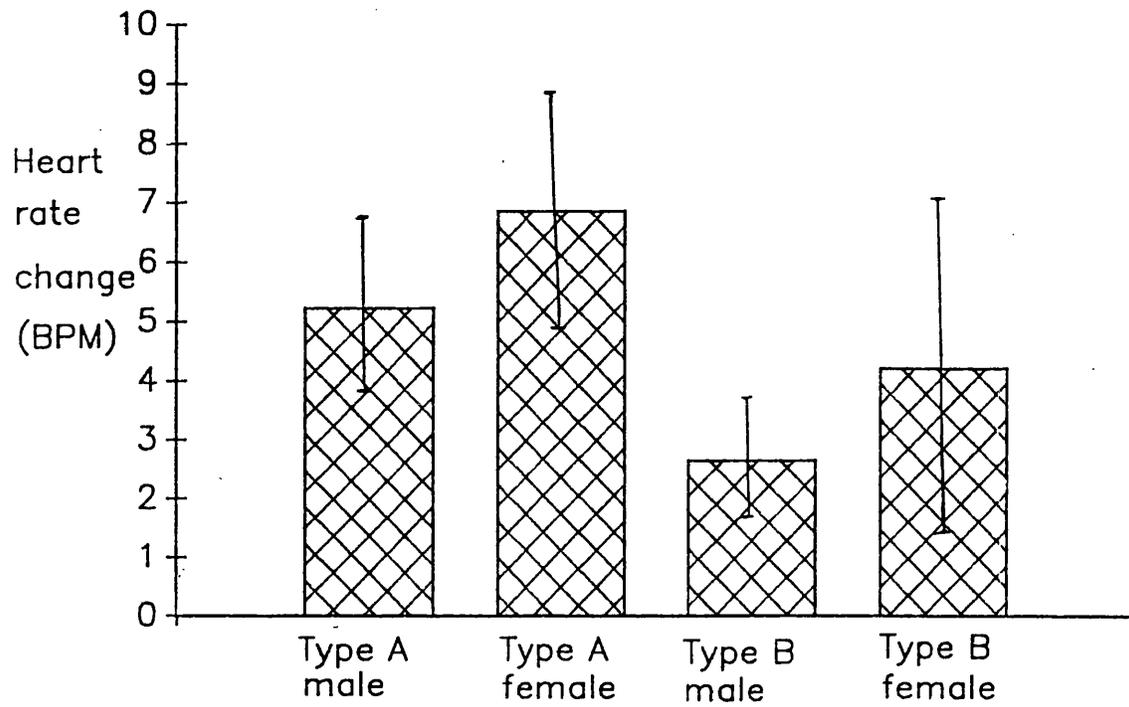
trend towards male Type As showing a more increased change in heart rate. Females show a trend towards higher heart rate than males from P-C (see figures 1 and 2).

figure 1: Heart rate change for Type A & B males and females from rest to practice



Overall males mean= 3.94 sd= 1.12 females mean= 5.54 sd= 2.63

figure 2: Heart rate change for Type A & B males and females from practice to competition



Overall males mean= 4.93 sd= 2.99 females mean= 3.04 sd= 1.86

Table 4: mean erraticness values and standard deviations for Type A and B subjects.

Erraticness (in cm)				
	Type A		Type B	
	Mean	SD	Mean	SD
Prac.	144.31	22.73	125.46	16.42
Comp.	164.23	36.66	130.54	20.44

note: erraticness is the length of the trace of accelerator movements in cm for the first 90 seconds of driving during practice and competition.

A three way ANOVA with one repeated measure (competition) was conducted (Table 5).

Table 5 : ANOVA summary table for erraticness.

FACTOR	ss	df	ms	F	p
Type	8660.165	1	8660.165	7.56	0.01
Sex	319.242	1	319.242	<1	ns

PTO

TypexSex	276.433	1	276.433	<1	ns
Error	25193.941	22	1145.179		
Comp	1998.132	1	1998.132	9.46	0.006
CompXType	770.242	1	770.242	3.65	0.07
CompXSex	192.858	1	192.858	<1	ns
CxTxS	9.363	1	9.363	<1	ns
Error	4645.702	22	211.168		

As can be seen, Type A subjects are significantly more erratic in their "driving style" than Type Bs during both practice and competition. There are no interactions or sex differences.

Table 6: Mean time (in seconds) and sds for Type A and B subjects.

	Type A		Type B	
	Mean	SD	Mean	SD
Prac.	121.85	23.79	120.23	13.08
Comp.	103.31	12.06	118.15	20.14

A three way ANOVA with one within factor (competition) was carried out on the above data (table 7).

Table 7: ANOVA summary table for time taken to complete the race.

FACTOR	SS	df	ms	F	p
Type	495.238	1	495.238	<1	ns
Sex	394.315	1	394.315	<1	ns
TypexSex	437.951	1	437.951	<1	ns
Error	11741.119	22	533.687		
Comp	1392.645	1	1392.645	10.68	0.004
CompXType	884.661	1	884.661	6.78	0.02
CompXSex	4.046	1	4.046	<1	ns
CxTxS	11.722	1	11.722	<1	ns
Error	2869.310	22	130.423		

As might be expected there is a significant overall difference between time during practice and competition across all subjects.

There is no sex difference between Type A and B subjects. Unlike the results found for erraticness, there is no significant overall difference between Type A and B subjects as far as time is concerned. However, as can be seen from the above table, there is a significant interaction between

Type and competition indicating that although Type A and B subjects took almost the same time to complete the circuit during practice, Type As drove significantly faster during the competition phase of the experiment.

Table 8: Mean no. of falls/crashes and SDs for Type A and B subjects.

	Type A		Type B	
	Mean	SD	Mean	SD
Prac.	5.15	2.15	5.54	3.23
Comp.	9.54	5.36	5.62	3.23

A three way ANOVA with two between factors (Type and sex) and one within factor (competition) was conducted on these data (table 9).

Table 9: ANOVA summary table for no. of falls/crashes.

FACTOR	ss	df	ms	F	p
Type	61.000	1	61.000	3.77	0.07
Sex	0.770	1	0.770	<1	ns
TypexSex	35.641	1	35.641	2.21	ns
Error	355.512	22	16.160		

Comp	85.056	1	85.056	9.63	0.005
CompXType	42.037	1	42.037	4.76	0.04
CompXSex	2.576	1	2.576	<1	ns
CXTxS	5.440	1	5.440	<1	ns
Error	194.369	22	8.835		

As can be seen from table 9, there is no overall significant difference in the number of falls/crashes between Type A and B subjects. However, there is an expected significant main effect for competition signifying the fact that across subjects there is an overall difference in the number of falls/crashes during practice and competition. More importantly, there is a significant interaction between Type and competition. As can be seen from tables 8 and 9, Type A subjects had significantly more falls and crashes than Type B subjects during the competition stage. However, there is no significant difference between the two groups during the practice phase of the experiment.

Finally, a two way ANOVA with two between subjects factors (Type and sex) was carried out on the ratings made by Type A and B subjects indicating their desire to win the competition. Mean rating for Type A subjects was 15.35 (sd= 2.89), and for Type B subjects mean rating was 10.17 (sd= 3.08). The difference was significant ($F= 18.25$ $df= 1,22$

$P < 0.01$). There were no significant interactions or sex differences.

2.4. DISCUSSION

In the present experiment, subjects identified as Type A increased their heart rate significantly more than Type Bs from rest to practice (R-P) and from practice to competition (P-C). This supports the "habitual mode" theory of the Type A behaviour pattern. However, for driving style there was no significant difference between Type A and B subjects during practice when no mention of reward or competition was made. Only on one measure (erraticness) there was a significant difference in the absence of competition. Hence, the results tend to suggest that physiological reactivity in Type As are generally exhibited even in the absence of competition. Results on behavioural measures, however, suggest the necessity for the presence of competition for the clear emergence of the behaviour pattern.

Therefore, the hypothesis of the present study concerning the habitual characteristic of the Type A behaviour pattern reveals a somewhat complicated picture insofar as there is a discrepancy between behavioural and physiological measures. If the Type A behaviour pattern is habitual, then Type A subjects would be expected to exhibit Type A behaviour even in the absence of external cues. It must be pointed out that

the view that the Type A behaviour pattern is a habitual characteristic is by no means held by all the researchers in the field (Smith and Anderson, 1986). The present study therefore suggests that Type A is a behavioural propensity which needs an appropriate environmental stimulus in order to be manifested as overt behaviour.

Examining all subjects' data by using correlation coefficients suggested similar conclusions. Type A scores were significantly correlated with measures of erratic and careless "driving style", particularly when subjects were involved in competitive behaviour. Driving more erratically as defined by the more excessive use of the accelerator, driving at significantly higher speed and the number of car crashes in effect means that Type A subjects took more risks and drove more "aggressively" than their Type B counterparts in order to win.

Type A subjects expressed a significantly greater desire to win the competition than their Type B counterparts. This finding is consistent with physiological and behavioural results of the experiment, as well as the extensive self report literature on the Type A behaviour pattern (Furnham and Linfoot, 1987).

Research in the field of road safety seems to suggest that drivers who tend to cause more car crashes and driving

violations on the road seem to exhibit similar characteristics to those described in the Type A paradigm (eg Marsh and Collett, 1987; Perry, 1986). Although it may be argued that the driving task in the present study was a game and equating a certain game behaviour with actual highway behaviour may be viewed as an overstatement, the results of the present experiment showing a distinct driving style for Type A individuals different from that shown by Type Bs, pose possible important implications as far as predicting driver performance and improving road safety are concerned. It is suggested that future research on driving performance should take personality factors in particular the role played by the Type A behaviour pattern into consideration.

Pitariu (1985) has argued that some psychological assessment tools may have useful validity for predicting driving performance. It seems reasonable to suggest that the assessment of the Type A behaviour pattern in particular may be useful in predicting the performance of drivers. Furthermore, it may be possible to reduce road accidents by predicting and modifying the Type A behaviour pattern. In other words, The Type A behaviour pattern may be a potentially useful screening device for drivers. Conversely, driving style, similar to the way it was measured in the present experiment, may be used as one of a number of

possible behavioural non-self report measures of the Type A behaviour pattern.

This latter point is important. As discussed in chapter one, the tools devised for Type A assessment so far are either paper and pen questionnaires (Jenkins, etal. 1965; Haynes, etal. 1978; Bortner, 1969; Eysenck and Fulker, 1983) or a performance based measure, namely the structured interview, devised by Friedman and Rosenman in the 1950's, the questions for which were derived from experimenters' subjective interpretation of subjects' behaviour. The need for an objective measure which will ultimately enhance and facilitate Type A measurement was also emphasized in chapter one. One possible objective method for assessing Type A could be the measurement of behavioural characteristics such as driving style as outlined in the present experiment. However, the value of any objective assessment device depends on its ability to predict CHD. This is beyond the scope of the present research. Nevertheless, devising objective behavioural measures for the assessment of Type A and establishing the predictive validity of such measures should be an important aim of future research.

Sex differences in this experiment, though not significant, revealed some strong tendencies. Overall, males, particularly Type A males seemed to have increased heart rate more than females from rest to practice. However,

females showed a tendency towards increased heart rate more than males from practice to competition. These trends add to the complexity of the existing literature concerning the differences between Type A males and females. It may be speculated that since competition is more acceptable in men than in women, men fail to discriminate as well as women as to when competition is inappropriate. Hence, women's competitiveness may be more a function of specific situations (Price, 1983). It may be that with the increased involvement of women in the competitive work market these sex differences are largely disappearing.

Finally, behavioural and physiological differences between Type A and B subjects were similar to other well documented findings in the field (Jones, 1985; Gasturf, etal. 1980; Glass and Karkoff, 1980). One contribution of the present study to the Type A literature in that it reports a theoretically expected pattern of findings for a relatively new Type A scale, namely the Eysenck Type A questionnaire. Type A subjects increased heart rate under competition significantly more than Type Bs. This latter finding raises an important question concerning the modification of the Type A behaviour pattern. If physiological levels are raised so easily in Type As under competition, then it may be possible to decrease physiological levels such as heart rate by making Type A elements such as competition salient factors in modification programmes. For example, by using

reward in a competitive situation, Type A individuals may be encouraged to reduce heart rate rather than increase it. Some researchers have attempted to employ similar methods in biofeedback programmes (Stern and Elder, 1982; Prior, et al. 1983) but the results of these studies are by no means conclusive. It is of great value to consider taking advantage of the core elements of the Type A behaviour pattern to modify physiological changes that occur when these behaviours are exhibited. This will be the main concern of the next study which will investigate the possibility of reducing heart rate in extreme Type A individuals using contingent and false heart rate biofeedback in a competitive situation where Type A and B subjects will compete to reduce heart rate rather than increase it. Before the study is described, a review of the biofeedback literature will be offered in the next chapter.

CHAPTER THREE

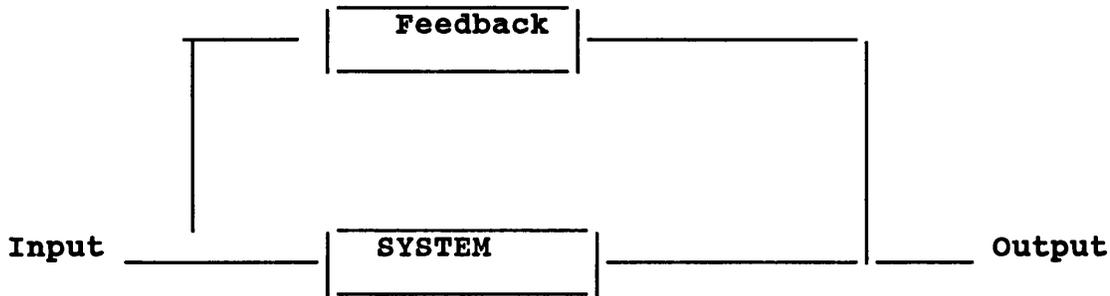
BIOFEEDBACK

The aim of the present chapter is to review the literature on biofeedback. Since it is the ultimate aim of the present research to offer a new approach to the modification of physiological responses in healthy Type A individuals using heart rate biofeedback, it is necessary to outline the significance of biofeedback technology in some detail. The present chapter will deal with the history of biofeedback, outlining and criticizing theoretical models and discussing the contribution of various biofeedback techniques as methods of relaxation and in the treatment of a variety of psychosomatic illnesses.

3.1. DEFINITION OF BIOFEEDBACK

The notion of feedback was brought into specific use by the mathematician Norbert Wiener (1948) who defined it as "a method of controlling a system by reinserting into it the results of its past performances." It is seen most clearly in the form of the feedback loop which is shown in a simplified form below:

Figure 1: the feedback loop



Biofeedback may be considered a special case of this method since the system is a biological one. The organism is placed in a closed feedback loop and information concerning one or more of its bodily functions is continuously made known to it. The feedback is artificial, mediated by man made detection, amplification

and display instruments. The principle underlying biofeedback is a novel yet very simple one. Traditionally when physiological processes, such as cardiovascular activity are monitored in the medical context, the information is presented to the health professional who is involved in the care of the patient. The novel twist in biofeedback is that this information is not fed back to the "experts" but to the patient. Thus, the information concerning the individual's bodily functions is used by the individual so that the functioning of the system is controlled. By continuous exposure (biofeedback training) to organ specific artificial feedback, individuals can learn to bring under conscious control particular bodily functions that ordinarily are not

subject to conscious control (eg. heart rate and blood pressure) or are under minimal conscious control (eg. tension in the frontalis muscle). For example, an individual with tension headache, due to abnormal levels of tension or contraction in the frontalis muscle, only knows that the front of his head hurts. With biofeedback, the patient can know accurately the level of activity at any time while it is being monitored in this muscle.

Biofeedback has been demonstrated to be a practical and successful form of treatment for improving psychosomatic health (Schwartz, 1973). Research in this area envelops a number of bodily processes. The major systems involved in biofeedback are the neuromuscular (electromyographic or EMG), the central nervous system (electroencephalographic or EEG), the sympathetic nervous system as manifest in electrodermal response (Galvanic Skin Response or GSR), and the cardiovascular system (heart rate, blood pressure, skin temperature).

3.2. HISTORY OF BIOFEEDBACK

Biofeedback has a relatively short history. The term biofeedback did not come into widespread use until 1969. It is generally accepted that four fairly distinct lines of research in the late 1960's produced the field of biofeedback. These were studies of operant conditioning of

human heart rate and of human galvanic skin response; studies with curarized animals; and studies on the feedback control of the alpha rhythm of the human EEG.

Kimble in 1961 argued that all the responses typically thought to be involuntary and under the control of or innervated by the autonomic nervous system, such as stomach acid secretion, blood pressure, skin resistance and heart rate were no subject to instrumental or operant conditioning. Instead, the latter form of learning was thought possible only for responses which were under "voluntary" control such as skeletal muscle response. Shortly after the publication of Kimble's assertions reports began to appear in which autonomically mediated responses were shown to be modified by instrumental conditioning methods. Two of the earliest reports (Shern, 1962; Frazier, 1966) each employed a shock-avoidance paradigm in which subjects could avoid mild electrical shocks by making appropriate changes in heart rate.

At about the same time other researchers (e.g. Kimmel and Hill, 1960; Kimmel, 1967) were showing that Galvanic skin response (GSR) could also be operantly conditioned. This line of research however, seemed to die out eventually. This could be due to the general lack of clinical utility of the work in GSR biofeedback.

A third response to Kimble's (1961) statements was that of Miller (1969). Laboratory rats were given injections of curare, a drug which paralyzed all skeletal muscles by blocking the myoneural synapse. The rats were maintained on artificial respiration, which both kept them alive and also regulated their breathing exactly. As a means of delivering a reinforcement to a paralyzed animal, an electrode was implanted in one of the so called pleasure centres of the hypothalamus. It was argued that not only could heart rate be operantly conditioned, but likewise, so could the blood pressure, urine formation, and even the degree of vasoconstriction and dialation in the ear. (Miller, et al. 1970). However these brilliant experiments on operant conditioning of autonomic responses could not be replicated successfully in subsequent attempts (Miller and Dworking, 1974). No ready explanation for these later failures has been given.

The final line of research which lead to the field of biofeedback was work in the field of EEG. Several researcher, all relatively independently, began to study whether subjects could "voluntarily" produce the alpha rhythm, a distinctive 8-13 Hz (cycles per second) rhythm (Kamiya, 1968; Brown, 1970; Hart, 1967). Because of the similarity in the subjective experience of a "high alpha state" with that reported for meditation, the work with self-control of the EEG attracted much attention from the

non scientific world and thus helped the whole field to grow. Furthermore, the field showed considerable promise in clinical applications.

Mainly due to this growing interest, a small group of researchers interested in "feedback effects on biological responses" began to create an informal network in California. This eventually led to the first meeting of this group in October 1969. From this meeting emerged the Biofeedback Research Society which started to publish findings in the area in its own journal "Biofeedback and Self Regulation". In 1976 the name of the Society was changed to Biofeedback Society of America in order to accommodate the growing number of practitioners of clinical biofeedback along with those more interested in research.

3.3. TYPES OF BIOFEEDBACK

The existing ways in which the physiological information may be fed back to the subject are in terms of auditory or visual sensory modalities, or both at the same time. For all of these the information may be analogue, binary, or digital.

Visual analogue feedback may be given through a dial and a moving pointer where the physiological level is translated directly and continuously into a slowly moving (dc) voltage

level which is displayed as a dial reading; or via a computer programme which translates physiological level in the form of a display on the computer screen. In both cases information is given directly and continuously. Auditory analogue feedback also uses a system in which the physiological activity is converted into a continuous voltage, and then is fed into a voltage to frequency converter so that the output is a tone.

Binary feedback is given through a constant signal which is either turned on or off when an already defined threshold is exceeded. Thus, the information an individual gets is whether or not the level of physiological activity is greater or lower than the threshold level.

It has been shown that analogue feedback may be superior to binary feedback for heart rate speeding but not necessarily for heart rate slowing (Blanchard, et al. 1974).

Digital feedback is usually visual where physiological levels are translated into numbers which are displayed to the subject. Other parameters of biofeedback are the delay between the physiological event and the presentation of the information to the subject, the information being related to the instantaneous physiological level or to a running average over a certain amount of time. Feedback presented immediately after the event, leads to more efficient

learning. This view has been supported by Gatchel (1974) for heart rate speeding and by Wells, et al. (1973) for salivation.

Financial rewards have been used in addition to biofeedback in an attempt to improve subjects' performance. There has indeed been considerable support for the notion that financial reward enhances control over physiological events in the case of heart rate control (Lang and Twentyman, 1976) and EEG alpha enhancement (Brolund and Schalloro, 1976).

3.4. APPLICATIONS OF BIOFEEDBACK

The purpose of the following section is to present an overview of the literature on the clinical application of various forms of biofeedback. Since the aim of the present research is to use heart rate feedback to reduce physiological responses in Type A subjects, the research on heart rate biofeedback will be outlined here in more detail.

3.4.1 Electromyographic (EMG) feedback

The basic principle underlying EMG feedback is that electrodes are attached to chosen muscle groups to detect the firing rate of specific motor neurones. EMG feedback studies with on-patient volunteers have provided reasonable evidence that a degree of control can be obtained over

individual motor units and over whole muscle groups. (e.g. Budzynski and Stoyva, 1969; 1973).

The clinical studies have approached a diverse range of clinical problems. One of the earliest approaches involves the use of EMG feedback for direct muscle retraining in patients with diminished or absent muscle activity as the result of strokes, spinal damage or other crippling lesions. A number of studies have shown considerable gains in muscle activity as a result of including EMG feedback as part of therapy (Basmajian and Hatch, 1979).

In contrast, EMG feedback can also be used effectively to diminish excessive muscle activity such as that occurring in spasmodic torticollis (Cleeland, 1973); and facial spasms, for example occurring in Parkinson's disease (Netsell and Cleeland, 1973). The reduction of frontalis muscle activity is so used to modify tension headaches associated with abnormal levels of frontalis activity (e.g. Wickramaskera, 1973; Reinking and Kohl, 1975).

3.4.2 Electroencephalographic (EEG) feedback

The use of EEG biofeedback training in volunteer subjects has already been mentioned briefly. Recent evidence suggests that the highly touted training of the brain to produce increases in alpha waves has accomplished less than was

originally anticipated. Recent studies have generally failed to confirm earlier findings that a state of calmness and well being by producing alpha waves can be reliably induced. Furthermore, some studies have suggested that subjects' expectations of alpha training may be responsible for whatever effects produced (Glaros, 1975). "There is no evidence that this treatment has helped patients or that it allows people to relax any better than if they just sit quietly with their eyes closed" (Paskewitz and Orne, 1973).

A more specific application of biofeedback has been used with epileptic patients (Sterman, 1973). This has involved training epileptic patients to increase the amount of sensori motor rhythm (SMR) found in the EEG. This rhythm is one in the range 12-14 Hz, found in the sensori motor cortex. The occurrence of 12-14 Hz activity was first observed in cats (Sterman, et al. 1969) and later in 1972 Sterman and Friar reported the systematic use of biofeedback training to produce sensori motor rhythm to control epileptic seizures in a human subject. Although Sterman's original findings have been replicated, some studies have failed to show that 12-14 Hz activity can be trained in humans or that such training helps to control seizures (Kaplan, 1975).

SMR training appears to result in reduction in the number of seizures in some but not all patients. Biofeedback training

of certain EEG rhythms (especially the sensori motor rhythm) that increases activity in the cortical inhibitory circuits has potentially the widest sphere of action. However, although this technique has not yet achieved credence (Powell, 1981), it seems to be very promising.

3.4.3 Cardiovascular feedback

The use of biofeedback has been reported in the successful treatment of migraine headaches. Elmore (1979) succeeded in markedly reducing the severity of migraine by teaching sufferers through biofeedback to constrict the extra cranial arteries. This constriction requires sympathetic arousal and might therefore be regarded as the diametric opposite of the control of hypertension by training patients to reduce their blood pressure. For example, in a recent study by McCoy, Blanchard, et al. (1988), hypertensive patients whose blood pressure was controlled by medication received either thermal biofeedback for hand warming or progressive muscle relaxation prior to medication withdrawal. A number of biochemical measures such as plasma norepinephrine and urinary sodium and potassium were taken before and after treatment. It was found that the biofeedback group showed significant reduction in arterial pressure and some of the biochemical measures. The relaxation group, however, showed no significant reductions. Although the results are impressive, they have not been conclusive enough to

establish blood pressure feedback as a standard treatment for the problem since there are contradictory results suggesting no change in the severity of headaches after biofeedback training (Shapiro and Surwit, 1979). Moreover, it has been argued that relaxation training, which is often given along with biofeedback, does more to reduce blood pressure than biofeedback itself (Blanchard, et al. 1979).

3.4.3.1 Heart rate biofeedback

The use of heart rate biofeedback in clinical settings stems from enthusiastic research conducted in the laboratory with normal subjects since the early 1960's. A brief outline of early research in heart rate biofeedback has already been described. It has been demonstrated that subjects can show significant increases in heart rate (Frazier, 1966). Engel, in a series of experiments, which have given rise to extensive use of binary heart rate biofeedback by Engel and his colleagues with patients in clinical settings, has shown that heart rate increase is achieved better using biofeedback than heart rate decrease. In the first study, Engel and Chism (1967) sought to operantly condition heart rate speeding. In this experiment, the amount of increase heart rate in experimental subjects averaged 5.9 ranging from 4.2 to 9.8 BPM. The overall average for the control subjects was 3.0 BPM.

In a second experiment, Engel and Hansen (1966) sought to operantly condition heart rate decreases. Binary feedback of heart rate was given in the form of a red light which was on when heart rate, on a beat to beat basis, was less than a base line level. When heart rate was above the base line level, the light was off. Results showed that the experimental subjects did decrease their heart rate below the base line level as compared to a control group. The average decrease was 0.5 beats per minute (BPM) with a range of -2.6 to +2.6 BPM. The control group who received no feedback actually increased heart rate by +5.6 BPM. From the results it appears that the significant difference between groups is more due to a sizable average increase on the part of the control group than lowering on the part of the experimental subjects.

However, at about the same time Brener, et al. were conducting similar experiments (Brener and Hothersall, 1967; Brener, et al. 1969). Brener's work showed that subjects could obtain significant differences in heart rate between trials on which they were to increase heart rate and trials on which they were to decrease heart rate, both with and, to a lesser extent, without biofeedback. Furthermore, it was shown that subjects control heart rate better with feedback than without it.

Lang, et al. (1967) showed the same general effects in an experiment investigating whether or not subjects could stabilize heart rate, to reduce the beat to beat variability which occurs naturally in most people. They found that only when subjects received tone feedback and only when they were told that the feedback was of their heart rate did they show any reduction in heart rate variability. This finding ended the myth that subjects learn autonomic response control better if they are unaware of what response they are controlling (Frazier, 1966). Hence these early studies showed conclusively that awareness of one's heart rate could significantly increase the ability to control the speed and variability of heart beats.

Heart rate biofeedback has also been applied in clinical settings to control various cardiac dysrhythmias. This occurs when due to some defect or because of cardiovascular disease, the normal rhythmic operation of the heart, known as the "sinus rhythm", is interrupted either regularly or episodically. "Some of these dysrhythmias are very serious and if uncontrolled can lead to death fairly quickly" (Blanchard and Epstein, 1978).

The first published reports on the use of heart rate biofeedback with clinical problems were by Engel and his colleagues during the early 1970's. Weiss and Engel (1970) published data on a group of eight patients suffering from

premature ventricular contractions (PVC). This is when the ventricles of the heart are incompletely filled with blood and overall cardiac output decreases. The training of patients more or less followed Engel's earlier methodology. Patients were told to increase heart rate, then to decrease heart rate, and then to alternately increase and decrease heart rate. Next patients were taught to decrease the variability of their heart rate (similar to Lang's 1967 work) by holding heart rate within a specified range. Binary visual biofeedback gave subjects direct information about the occurrence of a PVC. In the last phase of the study, self control training began. While the patient was keeping his heart rate within a specified range, biofeedback was systematically faded out. Three out of the eight patients completed the entire programme. But the authors obtained follow up data on all patients. In four patients there was a marked decrease in the number of PVC's. In the fifth patient there was noticeable decrease in PVC rate. For the other three cases there was no evidence of reduction in the rate of PVC's. The success of heart rate biofeedback training as applied to treating PVCs has been shown by Engel and his colleagues repeatedly (eg. Engel and Bleecker, 1974).

Recently heart rate biofeedback has been successfully applied to the treatment of essential hypertension, the treatment of which using cardiovascular biofeedback has already been briefly outlined. It has been argued that heart

rate biofeedback may be actually more effective than other forms of cardiovascular feedback such as blood pressure. Achmon, et al. (1989) assessed and compared biofeedback for heart rate control and another behavioural treatment for essential hypertension, namely cognitive group therapy for anger control. Biofeedback aimed at slowing heart rate in stressful situations. A significant reduction in blood pressure for both groups were found compared with the control groups. Furthermore, a significant reduction in blood pressure was found with heart rate biofeedback as compared with cognitive therapy. Therefore, it may be argued that heart rate biofeedback, because of its wide application in clinical problems, may be considered the best technique as compared to other forms of cardiovascular feedback.

3.4.4 Other forms of biofeedback

The use of biofeedback has been reported in the successful management of a variety of illnesses. Welgan (1974) treated patients with duodenal ulcers using brief biofeedback training to control gastric acid secretions. Fecal incontinence has been treated by Engel, Nikoomanesh and Schuster (1974). Recently Radnitz and Blanchard (1988) have used bowl sound biofeedback with patients suffering from irritable bowel syndrome and have reported significant success. Cornsweet and Crane (1973) presented the first evidence of control of accommodation in the modification of

visual acuity by discrete verbal and auditory feedback based on continuous measurement of accommodation.

Biofeedback techniques have also been used in the treatment of respiratory illnesses such as asthma. A forced oscillation technique has been used by Levenson (1974) and Feldman (1976) who reported decreases in respiratory resistance in asthmatic children.

3.4.5 Conclusions

A major question which needs to be addressed in all biofeedback studies is whether or not the effects observed last long enough to be of any clinical use. This point has been raised by Johnson (1984) in a major review of biofeedback literature. Some of the research described above have provided follow up data suggesting that the effects of biofeedback training may be sustained up to one year after the termination of training. However, in most studies using biofeedback training there is either a lack of follow up data or the results of follow up studies are negative suggesting that the effects of biofeedback are not sustainable over a long period of time. The contradiction in results may be due to poor methodology but nevertheless casts a shadow of doubt over the clinical utility of biofeedback training. Although the results of some follow up studies are positive and impressive, further research must

be carried out to specifically test the long term effects of biofeedback. Nevertheless, biofeedback training has shown great promise in that it has been used to reduce stress and tension both in patients with a variety of psychosomatic illnesses and with normal subjects. In this respect, biofeedback training seems to be a possible effective relaxation technique for reducing physiological responses in Type A subjects.

3.5. THEORIES OF BIOFEEDBACK

3.5.1 Biofeedback as operant conditioning

Biofeedback has been considered by most researchers to be a special case of operant conditioning. A measurable physiological change, such as a reduction in heart rate, is followed by a signal denoting the change. This signal is assumed to function as a secondary reinforcer to increase the strength of the response (Engel, 1971).

An important feature of operant theory is its "seeming capacity to include practically anything within its purview" (Davison and Neale, 1982). If someone acquires a new skill, the researcher first looks for environmental reinforcers of a concrete sort, like money, food, or sex. If these are not found, a search is begun for more abstract events, such as receiving praise. If this hunt is similarly fruitless,

people "find" reinforcers such as feelings of well being. Awareness of internal events as they are monitored by highly sensitive bio electrical instruments - knowledge that heart rate is decreasing, blood pressure dropping, or contraction of the frontalis muscle diminishing- is similarly labelled a "reinforcer".

Elmore and Tursky (1978) have argued that adopting an operant conditioning theory of biofeedback does not increase our knowledge and understanding of biofeedback: "It will not tell us what kind of feedback is best, for example, auditory or visual, and whether or not some physiological systems will be more responsive to one kind of feedback signal than to another" (Elmore and Tursky, 1978).

More generally, the question may be posed: are there behavioural phenomena that are not amenable to an operant explanation? If there are not - and many researchers now believe this to be the case - can this view of human behaviour be accused, as behaviourists have accused psychoanalytic theory, of explaining every thing and therefore explaining nothing?

Some researchers have argued that biofeedback may be effective because it allows better processing of information rather than because it rewards (Lang, 1974; Shapiro and Surwit, 1976; Brener, 1974). Brener has argued that the

development of voluntary control is a function of the degree of "awareness of the response to be controlled". Hence, the ability of subjects to discriminate the consequences of their actions is a prerequisite to the development of instructional control over those actions. The feedback signal may be important because it provides information rather than because it functions as reward for a particular response.

Birk (1973), in support of the operant conditioning theory of biofeedback, has pointed out that this kind of reasoning fails to account for the effects observed in animals using feedback techniques (eg. Miller, 1969). Birk argues that in human subjects it is always some sensory signal or stimulus that provides information to the individual about "how he is doing" in attempting to bring this function under voluntary control by indicating to him results of early successes in changing a bodily function in a desired direction. However, Birk points out that curarized animals who do not "know" what they are trying to achieve by biofeedback are also influenced by these same kinds of learning processes through the use of primary reinforcing stimuli with or without instruction, cognitive awareness or conscious intention to change a particular bodily function and therefore the fundamental and irreducible paradigm of biofeedback is the operant conditioning of bodily responses that usually are largely out of awareness. However, in response to this view,

Stroebel and Glueck (1973) have argued that biofeedback has actually objective and subjective components in humans. The participant is placed in a closed feedback loop where information about one or more of his normally unconscious physiological processes is made available to him. The subject's progress in controlling the process (eg. density of EEG alpha or muscle tension) can be monitored objectively with a polygraph for example. The person's subjective mental state in achieving such control and his interpretation of it can not be monitored, except by introspective report. Therefore, Stoebel and Glueck raise the critical point that it is possible to assume that an important part of biofeedback may be due to the subject merely responding to a suggestion-placebo effect.

The authors have therefore argued that if animal studies documenting the active principle of biofeedback were not available, the evidence discussed above would be sufficient to conclude that all of the human results with biofeedback could be explained by invoking a subjective, placebo, cognitive, expectancy, suggestion type rationale. Therefore, It seems that although operant conditioning may play a significant role in biofeedback, explaining biofeedback in terms of operant conditioning alone is not sufficient.

3.5.2 Placebo effects and the self attribution theory

In attempting to offer alternative theories, some researchers have interpreted the effects observed in biofeedback as a result of placebo effects and have in fact viewed biofeedback as "an ultimate placebo" (Stoebel and Glueck, 1973). This view of biofeedback has emerged from developments made in the conceptualization of a self attribution theory of emotions. The theory was first proposed by Schachter (1964) under the influence of the American philosopher William James (1842-1910). James (1890) presented a theory which attempted to explain feelings of emotion as feedback from peripheral effects of the autonomic nervous system and from skeletal muscles. Schachter argues that given a state of physiological arousal for which the individual has no immediate explanation, he will describe his feelings in terms of cognitions available to him. Also given a state of physiological arousal for which there are immediate explanations, no evaluative needs will arise; and finally, given the same cognitive circumstances, the individual will react emotionally or describe his feelings as emotion only to the extent that he feels a state of physiological arousal. From this point of view Schachter (1964) developed a self attribution theory of emotion. There have also been extensions of this argument in the general context of self attribution theory (eg. Bem, 1967). Thus, the individual may use the information from his or her own

expressive behaviour as well as physiological cues, to interpret and label his or her emotional experience. Schachter was therefore able to integrate much of the research on the James-Lange theory of emotion. Hence, Schachter's theory suggests that emotional terms are used as labels attached to various feeling states by the individual.

Nisbett and Schachter (1966) demonstrated that the same bodily states may be labelled differently in different situations. Subjects were given a placebo before being given electric shocks and half were told that the side effects would cause arousal symptoms such as palpitation, tremor, and sweating. The other half of subjects expected no such symptoms. Subjects believing themselves to be in an artificial state of arousal, failed to attribute their shock created arousal to the shock and reported the shock to be less painful.

It has also been shown that the use of self attribution and placebo effects can alter physiological states. For example, Sternbach (1964) gave three sets of instructions in a "drug experiment" stating that one kind of pill would relax the stomach, that a second was a placebo pill having no effects, and that a third was a stimulant to the stomach. In fact, all three pills were plastic coated magnets used to monitor gastrointestinal activity. In most subjects, the stomach

responded in accordance with the anticipated effects of the drug.

These studies have lead some researchers to believe that some of the effects observed in biofeedback studies may be due to similar placebo effects. This poses an important theoretical challenge. To test the hypothesis that nothing more than placebo explanations can be offered to explain the effects observed in biofeedback, researchers have used false, non-contingent or sham feedback (Valins, 1969; Stern, Herrick and Botto, 1972; Shahidi and Powell, 1988). These studies have generally supported the self- attribution theory of biofeedback and have suggested possible implications for the use of false feedback in clinical settings. At the same time, some recent false feedback studies have generally rejected the self attribution theory (Beck, etal. 1988; Parkinson and Colgan, 1988). These studies will be discussed in the next section.

3.5.2.1 The concept of non-contingent (false) biofeedback

In some biofeedback experiments non-contingent, false, or sham feedback has been used in an attempt to control for the placebo and non-specific effects (for example, Finley (1977; Engel and Hansen, 1966). These studies, however, have used false feedback in a random manner. In other words,

information has not been systematically manipulated. The process involved in biofeedback is when the information concerning bodily functions is continuously made known to the subject so that unconscious bodily events are brought at least under partial control. The concept of false biofeedback refers to the manipulation of this information so that the individual is somehow misinformed about his or her bodily events. The subject is thus lead to believe that physiological activity is decreasing or increasing as the case may be. A classical experiment by Valins (1966) using false biofeedback of this kind has been reported particularly in support of the self attribution theory discussed in the previous section.

Valins (1966) conducted an experiment which was concerned with "some of the cognitive effects of internal events". The objective was to ascertain whether the labelling of emotional stimuli would be affected by information concerning internal reactions. Subjects viewed slides of semi nude women while hearing sounds which were allegedly their heart beats. One group of subjects heard their "heart rate" increase to some of the slides and not to others. The control group heard no change to the "heart rate". Using rating scales, Valins found that subjects in the experimental group rated slides which were associated with increased heart rate more favourably than the other slides. Valins concluded that subjects who were presented with false

biofeedback assumed that they were excited and, therefore, rated the slides as exciting. Therefore, in support of Schachter's (1964) self attribution theory, it was argued that the individual uses external cues such as physiological reactivity to explain and label emotional states.

During the early 1970's, a number of false feedback experiments (Stern, Botto and Herrick, 1972; Decaria, Proctor and Malloy, 1974) generally supported Valins' results supporting the self attribution theory. These studies were almost unanimously rejecting the operant conditioning model of biofeedback. Thus false feedback has been used to demonstrate the shortcomings of the operant conditioning model and emphasize placebo effects and the self attribution theory.

Recently, however, the self attribution theory has been criticized as a result of renewed interest in the field of false biofeedback and the replication of many of these experiments including Valins' original study (Parkinson and Colgan, 1988; Beck, et al 1988). These recent experiments have argued that the Valins effect is due to experimenter demand rather than self attribution. It is argued that given the elements involved in a typical false feedback experiment, the aim of the experiment is made salient so that the subject may guess what is demanded of him and respond accordingly. Evidence for the experimenter demand

hypothesis comes from studies in which subjects have been given only very brief false feedback (five seconds) which is thought to be too brief a time to search the slides for cause of the apparent physiological arousal (Barefoot and Straub, 1971). Here significant feedback effects in terms of subjective reports have been found, suggesting that subjects may be giving high ratings to slides associated with heart rate change only to please the experimenter. Although these results offer criticism on the experimental manipulations reported by Valins and his associates, experimenter demand may not necessarily be the salient feature in all false feedback studies. For example, some experiments using false biofeedback have found subjective ratings by subjects to be congruent with actual physiological change following false feedback (Shahidi and Powell, 1988). In other words, it has been found that subjects receiving, for example, false heart rate biofeedback suggesting that heart rate is decreasing across a relaxation session, report more relaxation but also show a significantly more decreased heart rate than control subjects receiving no feedback. Furthermore, as pointed out earlier, actual physiological changes have been reported in experiments using placebo effects (Sternbach, 1964). It is difficult to assume that physiological changes are also due to experimenter demand since pleasing the experimenter in effect means denying actual heart rate change and reporting one's state of relaxation as congruent with the direction of false feedback. If there is no experimenter effect there is

no desynchrony between subjective reports and actual heart rate change. Therefore, it may be argued that the experimenter demand hypothesis may play an important role in some non-contingent biofeedback studies but its significance is very much questionable for the effects observed in most false feedback experiments. Furthermore, false physiological feedback has been used in clinical studies where some psychological problems such as chronic shyness and speech anxiety have been successfully alleviated (Borkovec, et al 1974). It seems therefore that if experimenter demands are carefully disguised in false feedback studies, then self attribution seems to be the most likely explanation for the effects observed.

3.6. CONCLUSIONS

The development of the concept of biofeedback and theoretical models of biofeedback were discussed in the present chapter. It was argued that the operant conditioning model fails to account for the subjective effects of biofeedback training and that the self attribution theory is superior in this respect. Evidence for the self attribution theory was discussed primarily in terms of studies which have used false biofeedback. The self attribution theory seems to offer the best explanation for the effects observed in biofeedback and the next study of the present research

will be designed using contingent (true) and non contingent (false) biofeedback.

The present chapter was also concerned with the applications of biofeedback in alleviating a vast range of illnesses. It was argued that various types of biofeedback training can be used successfully as a relaxation technique to reduce physiological activity. It was pointed out that the clinical use of biofeedback in terms of its long term effects needs clarification but research suggests that biofeedback has significant potential in the area of stress management.

In view of the above points, it is surprising that to date only a very few studies have attempted to use biofeedback to reduce physiological reactivity in Type A subjects. Results of the first experiment (chapter two) showed clearly that Type As exhibit heightened physiological reactivity, particularly when involved in competition. It was argued in the previous chapter that it may be possible to take advantage of the core elements of the behaviour pattern such as competition to actually help Type As relax. Biofeedback seems to be an ideal relaxation technique for reducing physiological reactivity in Type A individuals.

The next chapter will be concerned with describing an experiment using contingent and non-contingent heart rate biofeedback to facilitate relaxation in healthy Type A and B subjects.

CHAPTER FOUR

BIOFEEDBACK TRAINING OF RELAXATION IN TYPE A AND B
ADULTS: THE EFFECTS OF FALSE FEEDBACK AND COMPETITION

4.1. INTRODUCTION

The result of the first experiment (chapter two) showed distinct behavioural and physiological differences between Type A and B subjects particularly when involved in competition. In particular, it was found that Type A subjects exhibited significantly higher heart rate than Type Bs. The results were similar to well documented findings (Jones, 1985; Ward, 1986) suggesting that Type A subjects show excess physiological reactivity during competition.

In chapter one, attempts to modify the Type A behaviour pattern since the mid 1970's, in both post infarction patients (eg. Suinn, 1976; Friedman, et al, 1982; Powell, et al, 1984) and healthy subjects (eg. Roskies, 1977, 1986) were discussed. It was pointed out that attempts to modify the Type a behaviour pattern in healthy subjects have produced mixed results (Haaga, 1987) suggesting that healthy Type As are very resistant to change. Furthermore, the most difficult aspect of the behaviour pattern to change has been physiological reactivity (Seraganian, etal 1987). It was further pointed out that a salient feature in most

modification programmes has been the extensive use of stress management techniques such as deep muscle relaxation exercises (Thompson, 1976), autogenic training and rhythmic breathing (Curtis, 1974), meditation (Muskatell, etal 1984) and biofeedback training (Prior, etal 1983). In this context, Rosenman and Friedman (1977) have supported the use of biofeedback training in modifying the Type A behaviour pattern. They have argued that since Type As persist in their quest to quantify and to compete, biofeedback training may serve as a particularly attractive therapy because its very nature presents them with a new challenge of quantification and competition except the new goal will be improvement in their own psychological state.

There have been only a few studies exploring the possibility of using biofeedback as a means of training healthy Type A subjects to reduce physiological reactivity. These studies generally support the notion that biofeedback training could play an important role in the modification of the Type A behaviour pattern. (Prior, etal 1983; Stern and Elder, 1982; Anchor etal 1979).

Prior, etal (1983) used EMG biofeedback training to reduce muscle tension levels in healthy Type A subjects. Subjects were trained for six sessions and returned for a seventh session to perform without biofeedback a series of easy and difficult tasks. It was found that subjects who received

biofeedback training reduced muscle tension levels more than the control group. Furthermore, although Type A biofeedback subjects had EMG levels as high as Type B control subjects for the actual duration of the performance tasks, they maintained significantly lower EMG levels than either group prior to, between, and after performance tasks. The authors conclude that biofeedback training may be considered to be a very promising means of modifying certain aspects of physiological reactivity in the Type A individuals since significant reductions in EMG levels in Type A subjects were only in the biofeedback condition.

Anchor, Anchor and Sandler (1979) have also used EMG biofeedback as relaxation training for Type A individuals. Subjects attended relaxation sessions twelve times. Each session consisted of EMG biofeedback training while listening to a relaxation tape. Results showed that although high Type A subjects reduced EMG levels significantly during and after relaxation sessions when compared to a control group, there was no significant overall difference in EMG reduction between high and low Type A subjects. This may be because the sample used was relatively homogeneous consisting of women with a history of stress related symptomology. Despite this, it must be pointed out that the results highlight biofeedback training as a form of teaching behavioural self control (Schwartz, 1973).

The most promising attempt to reduce physiological reactivity in healthy Type A subjects using biofeedback training was made by Stern and Elder in a series of experiments in 1983. In these studies the researchers found that Type As were capable of reducing physiological responses when put in challenging situations in which they would normally be expected to show increased physiological responses. For example, when Type As were told that reducing heart rate was a rare ability, Type As reduced heart rate more than Type Bs. Similarly when subjects were told to reduce heart rate as quickly as possible (time urgency) Type As were significantly more successful than Type Bs. Interestingly, when these challenging incentives were not available, Type B subjects were far superior to Type As. The authors suggest that "in light of the behavioural and physiological effects that Type A individuals show when striving to excel, it is surprising that no attempts have been made to study whether Type As can modify their physiological responses in a less stressful direction if challenged to do so."

In view of the difficulty in altering the Type A behaviour pattern in healthy subjects, what seems to be needed is an alternative approach. The above discussion points to the fact that exploiting the core elements of the behaviour pattern in order to encourage reductions rather than increases in physiological responses in Type As is a step in

the right direction. Furthermore, the use of biofeedback to motivate Type As seems most appropriate. In chapter three the biofeedback literature was extensively reviewed. In particular, the role played by placebo effects in the biofeedback paradigm were outlined. It is therefore reasonable to assume that placebo may play a significant role in the effects produced by biofeedback. Particularly with respect to Type A, the question of non contingent feedback may be associated with the need to control (Glass, 1977). Several studies have raised the importance of the need for control associated with the Type A behaviour pattern (Burke, 1982; Furnham, etal 1986). It is suggested, for example, that Type As tend to blame themselves more than Type Bs for negative events (Rhodewalt, 1984) and that Type As are found to process information in a way that bolsters self esteem. In this context, non contingent feedback where heart rate is shown to increase, may be viewed as an uncontrollable situation where heart rate seems to be increasing despite repeated attempts by the subject to reduce it. In such a situation Type As would not be expected to perform as successfully as Type Bs. On the other hand, false feedback showing decreasing heart rate may encourage and positively reinforce Type As to perform better than others.

The aim of the present experiment is to offer an alternative approach to modifying the Type A behaviour pattern in the

context of contingent and non-contingent (false) heart rate biofeedback which could be used as part of modification programmes with healthy Type As. It is suggested that the core elements of the Type A behaviour pattern such as competitiveness can be manipulated so that Type A persons may be encouraged to reduce heart rate rather than increase it. In other words, competition may be used to encourage Type As to relax rather than exhibit heightened cardiovascular activity.

In the present experiment Type A and B individuals are required to reduce their heart rate under competitive and non competitive biofeedback training. there are four experimental conditions. One group receives true contingent signals of heart rate (Cont-F) and another group is attached to the apparatus in the same way but does not receive feedback (No-F). Then there are two non contingent groups. One receives false feedback suggesting that heart rate is going down across the training session (Down-F) and one group receives false feedback suggesting that heart rate is going up across the training session (Up-F).

It is hypothesized that Type A individuals will reduce heart rate more successfully under competition that the no competition condition. Secondly, it is predicted that Type A individuals will reduce heart rate more than Type B subjects under competition.

The next set of hypotheses concerns the role of contingent and non contingent biofeedback in general. In chapter three it was pointed out that several studies have shown that biofeedback training as a method of relaxation is effective (Lang, etal 1967; Brener, etal 1969). The efficiency of biofeedback as treatment for a variety of psychosomatic illnesses was also discussed (Engel and Bleeker, 1974; Levenson, 1974; Achmon, etal 1989). In the present experiment it is predicted that overall, subjects receiving contingent heart rate biofeedback (Cont-F) will reduce their heart rate significantly more than the group receiving no biofeedback at all (No-F).

Experimental studies have also emphasized the important role played by placebo effects in biofeedback training (Stroebel and Glueck, 1973). It has been shown that the mere belief that one's physiological state is changing may be sufficient to affect both behaviour (Valins, 1966; Stern, etal 1972) and actual physiological responses (Shahidi and Powell, 1988). Therefore, it is hypothesized that subjects to whom it is suggested that heart rate is decreasing (Down-F) will reduce heart rate more than the group to whom it is suggested that heart rate is increasing (Up-F).

4.1.1 Hypotheses of the present study

The hypotheses of the present experiment may therefore be summarized as follows:

1. Type A subjects will be more successful at reducing heart rate when competing than when competition is not mentioned.
2. Type A subjects will reduce heart rate significantly more than Type B subjects during competition.
3. Type A subjects who are in the Up-F group will not be successful at heart rate reduction as compared to subjects in other feedback groups.
4. Overall, the Cont-F group will reduce heart rate significantly more than the No-F group.
5. The Down-F group will reduce heart rate significantly more than the Up-F group.

Finally, in a clinical context, one hopes that any relaxation procedure will cause the subject to relax on both subjective and objective measures. In this study, one would expect in general that self report of relaxation would be highly congruent with actual heart rate. But potentially, some interesting situations could arise in the two false feedback conditions. For example, it is possible for a subject to be relaxed during a relaxation session and have lowered heart rate but be told by the false feedback signal that heart rate is increasing. Hence the present experiment will examine the relative influence, upon subjective report, of heart rate and false heart rate information under

conditions in which these two are incompatible (Valins,-
1966; Stern, etal, 1972).

4.2. METHOD

4.2.1 Subjects

102 University students (mean age 23.5 age range 17-41) from the psychology department and other departments of University College London were contacted by letter asking them to take part in a psychology experiment on relaxation. Each subject filled in the Eysenck Type A Questionnaire (Eysenck and Fulker, 1983). Of these 37 subjects were extreme scorers that is they scored one standard deviation above or below the mean (mean= 17.44, sd= 5.5; mean for Type As= 24.75 sd=2.18 Type Bs= 7.99 sd= 2.50) and were asked to take part in the main study. There were no refusals. However, five subjects failed to complete the four relaxation sessions or were rejected from the sample in order to create equal cells. Therefore, data for 32 (17 females and 15 males) was analyzed. 16 subjects were labelled Type A and 16 were labelled Type B. The mean age was 23.13 years and the age range was 18-41 years. Each subject was paid £2 for completing the experiment.

4.2.2 Design

A repeated measures design was used where each subject took part in four biofeedback training sessions. In session Cont-F the subject received true contingent heart rate biofeedback. In session No-F the subject received no feedback at all. In session Down-F the subject received false non contingent heart rate biofeedback suggesting that heart rate was going down across the training session. In session Up-F the subject received false biofeedback suggesting that heart rate was going up across the training session. The biofeedback sessions were conducted on four consecutive days for each subject. A Latin square design was used to alter the order of each biofeedback session. The standard form of a Latin square is, by definition, that square obtained by rearranging the rows and columns until the letters in the first row and the letters in the first column are in alphabetical order (Winer, 1971 p 688). The following arrangement as an example of a Latin square was used in the present experiment: a b c d

b c d a

c d a b

d a b c

where a= Cont-F, b= No-F, c= Down-F, d= Up-F.

Half of the subjects (8 Type As and 8 Type Bs) were given competitive instructions as described below and the other half were given no competitive instructions at all. Mean age

and Eysenck scores for each of the four groups is given below.

Table 1: Mean age and Eysenck scores for Type A and B competition and no competition groups

	Mean (sd)	
	Age	Eysenck (out of 34)
Type A Comp	22.63 (4.24)	24.63 (1.87)
Type A No comp	21.75 (1.98)	24.88 (2.52)
Type B Comp	24.00 (6.27)	7.75 (2.77)
Type B No comp	24.13 (6.90)	8.13 (2.26)

4.2.2.1 False feedback

The false feedback display was prepared in a previous study at the University of Surrey (Shahidi, 1986; Shahidi and Powell, 1988) where 10 subjects (4 males and 6 females, mean age 22.4 years) were asked to participate in a study intended (1) to establish the optimum duration of the biofeedback session; (2) to create a false heart rate biofeedback programme; and (3) to modify the programme so that the feedback appeared realistic to subjects.

Ten minutes was regarded to be the optimum duration for a training session as most subjects in the study reported fatigue after ten minutes (Shahidi, 1986).

False feedback was created by carefully manipulating one subjects heart beats which had been recorded on disc and could be displayed and altered on a BBC computer. It was designed to be as realistic as possible. Several versions of false feedback were presented to to the ten subjects and each version was modified after it was presented until a point was reached where it was decided that the display was realistic and that it resembled true heart rate biofeedback displays (Shahidi, 1986). The average beats per minute (BPM) was 72 for the ten minute session with a gradual decrease of 10 beats (75 BPM in the first minute to 65 BPM in the last minute) for the Down-F condition. A mirror image of this was presented for the Up-F condition (65 BPM in the first minute, 75 BPM in the last minute). The increase and decrease in heart rate was noticeable to subjects.

4.2.3 Apparatus

Heart beat signals were picked up by a Tunturi TPM-200 pulse meter which has a light sensitive clip. This is a very useful method of heart rate measurement and is particularly convenient for psychological experiments since it is far less intrusive and easier to use than the traditional method

of using surface electrodes (experiment one). This method is based on the principle of photo-plethysmography. The pulse meter contains a clip which is placed on the ear lobe and contains a transducer containing a small lamp and photo-cell. The pulsating blood flow passes more or less red light to the cell which produces an electrical change in sympathy with the heart beats.

A BBC computer programme (appendix 5) was prepared to display this signal on a BBC computer as an analogue signal to be presented to subjects (see below). Heart rate in the form of beats per minute (BPM) was also made available through an interface which amplified the signals and measured the interbeat intervals (IBI's) by means of timers to 1/100 seconds of successive heart beats. The IBI's were transformed into beats per minute (BPM) averaged out over one minute. The BPM was displayed continuously to the subject.

The biofeedback display consisted of two axes. The horizontal axis showed beats per minute (40 to 120). A fluctuating line extending from the vertical axis indicated to the subject what his/her heart rate was. Thus, continuous visual feedback was provided for ten minutes (for a photograph of the visual biofeedback display and the set up of laboratory equipment see appendix 6).

4.2.4 Procedure

Subjects were initially seen individually to arrange suitable times for the four relaxation training sessions on four consecutive days and were given a brief introduction to the biofeedback apparatus. For each session, subjects were taken to a laboratory with subdued lighting and were seated on a large comfortable reclining arm chair. Then the following instructions were given verbally by the experimenter: "I am interested to see if people can control their heart beats. What I'd like you to do is to sit here for ten minutes and try to relax as much as possible. I'd like you to reduce your heart rate as much as you can".

Subjects were run individually and took part in four relaxation sessions each as described in the design section. Before each biofeedback session the subject was told: "You will see a display of your heart rate on the computer monitor in front of you. I'd like you to keep your eyes open all the time and concentrate on the display of your heart rate". During the No-F control condition, the monitor was switched off and a circular piece of paper (2 cm in diameter) was placed at the centre of the computer monitor. Subjects were told to keep their eyes open and concentrate on the piece of paper. No biofeedback was given during the No-F session. Half the subjects, selected randomly, were told that they were involved in a competition with other

subjects and the person with the highest reduction in heart rate across the four sessions would win a prize of £10. They were reminded of the prize every time they attended a session. To the other half competition was not mentioned at all. The allocation of subjects to the competition or the no competition groups was done randomly. Following these instructions, the experimenter activated the apparatus and left the laboratory. After ten minutes of each session, the experimenter returned to the laboratory and presented the subject with a rating scale of 0 to 20. The rating scale was a continuous 20 cm long line. Subjects were asked to indicate on the scale how relaxed they were at the end each session. 0 represented "not at all relaxed" and 20 represented "extremely relaxed".

The following data were collected:

(1) Scores on the Eysenck Type A Questionnaire. Subjects scoring one standard deviation above the mean (23 or above out of 34) were labelled Type A and those scoring one standard deviation below the mean (13 or below out of 34) were labelled Type B.

(2) Average heart rate for each minute of each relaxation session in the form of beats per minute (BPM). There were four sessions for each subject and 10 minutes in each session.

(3) Scores out of 20 on the relaxation rating scale for each session. 0 represented "not at all relaxed" and 20 represented "extremely relaxed."

4.3. RESULTS

An initial 2 (Type)x 2(competition)x 4(Condition) was conducted for heart rate in the first minute of the relaxation session. No significant effects were found.

A 2x2x4x4x10 ANOVA with fitted regression slopes (over the successive minutes of the 10 minute sessions) was conducted on the raw data. The GENSTAT5 statistical computer program was used. The following factors were included in the analysis: 1.Type (A vs B), 2. Competition (Competition vs No competition), 3. Condition (Cont-F vs No-F vs Down-F vs Up-F) 4. Days (the four consecutive days on which the sessions were conducted) 5. Minutes (10 minutes for each session). The last three factors: Condition, Days and Minutes were repeated measures and a linear trend was fitted to the minutes factor. This was done to see if there are any gradual learning effects during the ten minute relaxation sessions.

Because of the complex nature of the analysis, only significant results will be presented here. For a full ANOVA

table refer to appendix 7). There were no significant main effects or interactions for the overall Type, Comp and Days factors. However significant linear trends were found when the Minutes variable was added. Table 2 shows the summary table for main results.

Table 2 : ANOVA summary table for main results.

Source	df	F	P
Minutes	9	8.22	<0.001
Linear	1	68.02	<0.001
MinutesxType	9	3.30	<0.001
Linear MinsxType	1	20.57	<0.001
MinutesxCompetition	9	0.52	ns
Linear MinsxCompetition	1	0.27	ns
MinsxTypexComp	9	1.30	ns
Linear MinsxTypexComp	1	4.07	0.045
Error	252		
MinutesxCondition	27	1.61	0.028
Linear MinsxCondition	3	8.12	<0.001
Error	648		

There is a significant linear trend for the overall Minutes factor. This confirms the suggestion in Figure 1 that subjects gradually reduced heart rate across the ten minute relaxation sessions. Also the significant interaction of linear trend of MinutesxType means that there is a significant difference in the rate of heart rate reduction

of Type As and Bs over the ten minutes. As can be seen from figure 2, Type As reduced heart rate more rapidly than Type B subjects.

There is a significant linear trend for the Minutes \times Types \times Competition interaction. This is shown in figure 3. As can be seen from the figure, Type As seem to have a higher starting heart rate during competition than other groups. However, this difference was found to be statistically insignificant. Contrasts of mean linear coefficients were calculated using GENSTAT. It was found that, during competition, Type A subjects' heart rate decreased linearly more than Type B subjects' heart rate across the ten minutes ($t= 6.54$ $df= 14$). Furthermore, Type As showed a significantly greater reduction in heart rate when competing than when no competition was encouraged ($t= 2.52$, $df= 14$). Even when not competing, Type A subjects performed significantly better than Type B subjects during both competition and no competition ($t= 4.02$ and 2.52 respectively). There was no significant difference between Type Bs during competition and no competition.

As can be seen from table 2, there is also a significant Minutes \times Conditions interaction effect, of which the linear component is significant. This is shown in figure 4. As can be seen, the Cont-F group decreased heart rate significantly more rapidly than the other groups except the Down-F.

Contrasts of mean linear coefficients confirmed this. Subjects in the Cont-F condition reduced heart rate significantly more than subjects in the No-F condition ($t=4.0$, $df=62$) and the Up-F condition ($t=3.88$, $df=62$). There was no significant difference between the Cont-F and the Down-F and the Up-F and the No-F conditions. Subjects in the Down-F condition showed a significantly greater linear decrease in heart rate than both the No-F and the Up-F groups ($t=2.98$ and 2.85 respectively). Hence, false heart rate biofeedback where heart rate is seen to decrease seems to be as effective as contingent heart rate biofeedback. Finally, there was no significant Minutes \times Type \times Condition interaction effect.

figure 1: Overall heart rate (BPM)
for each minute

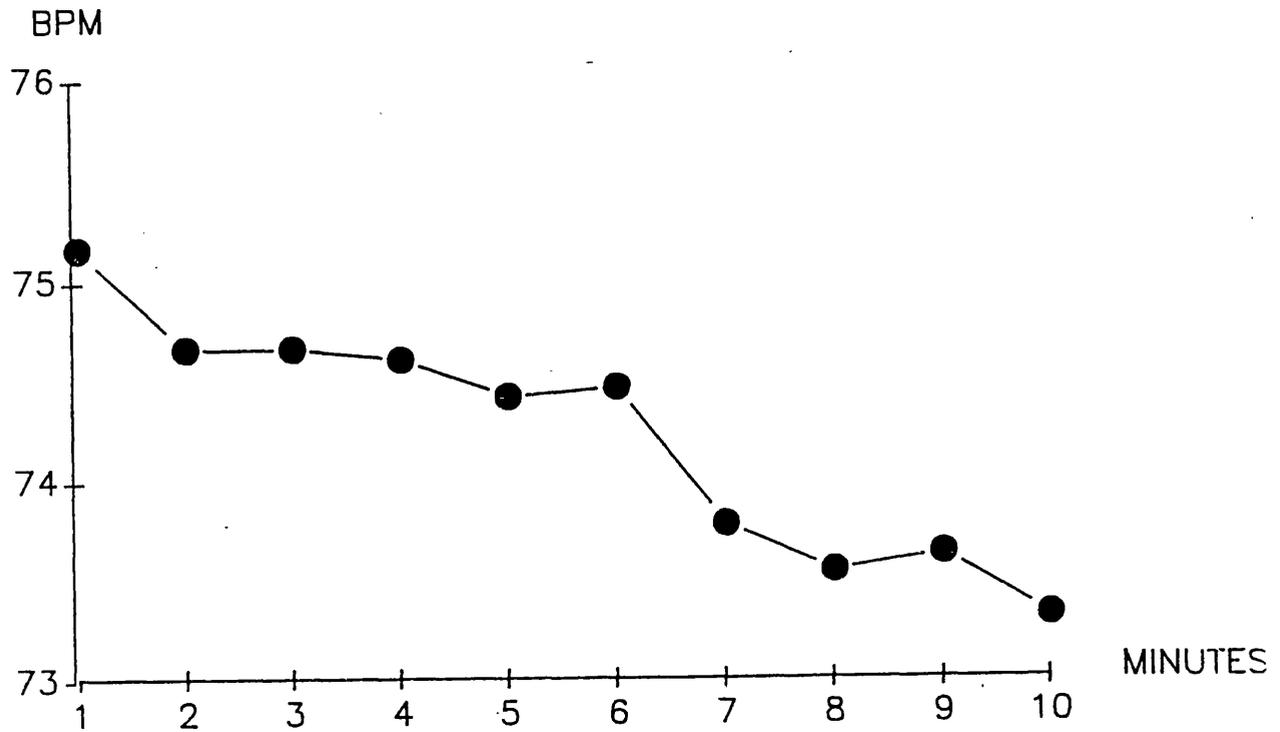


figure 2: Heart rate (BPM) for Type A and B
subjects

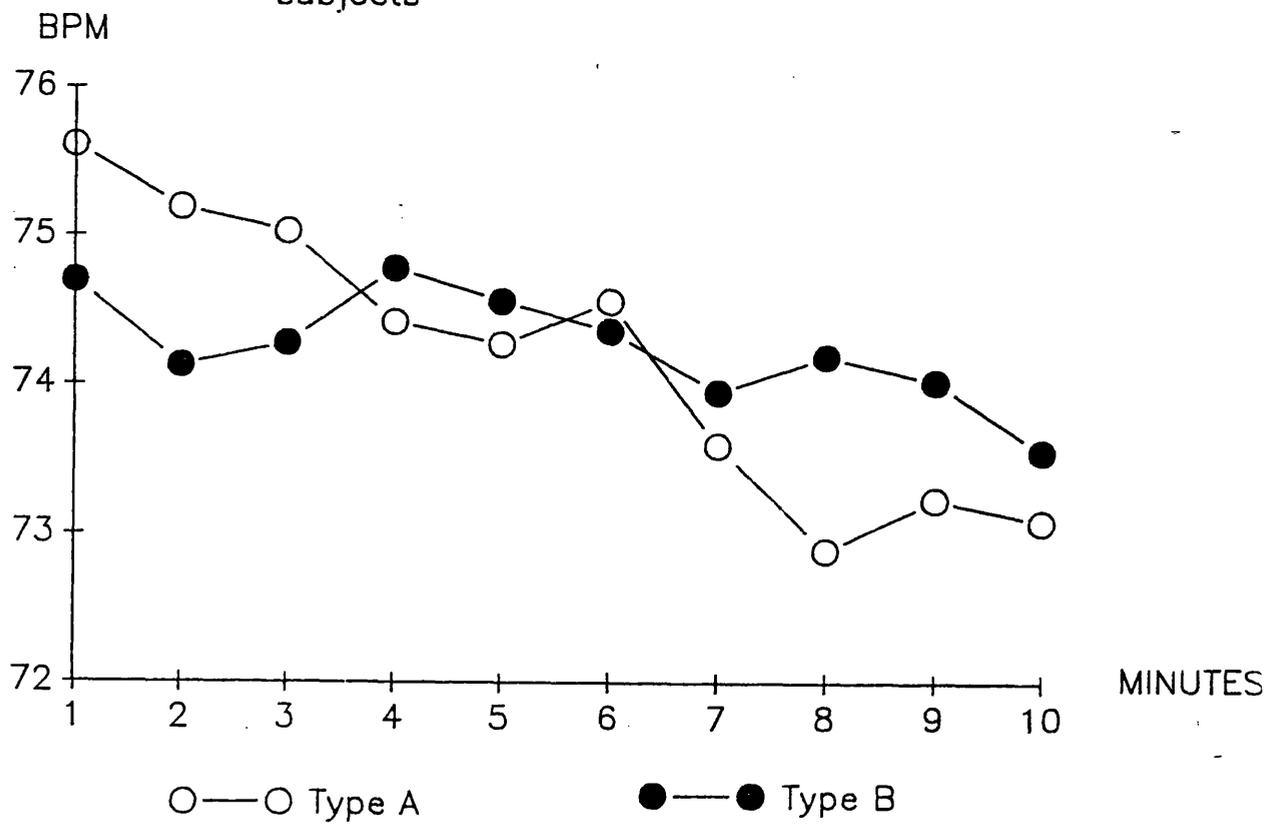


figure 3: Heart rate (BPM) for Type A & B subjects during competition and no competition

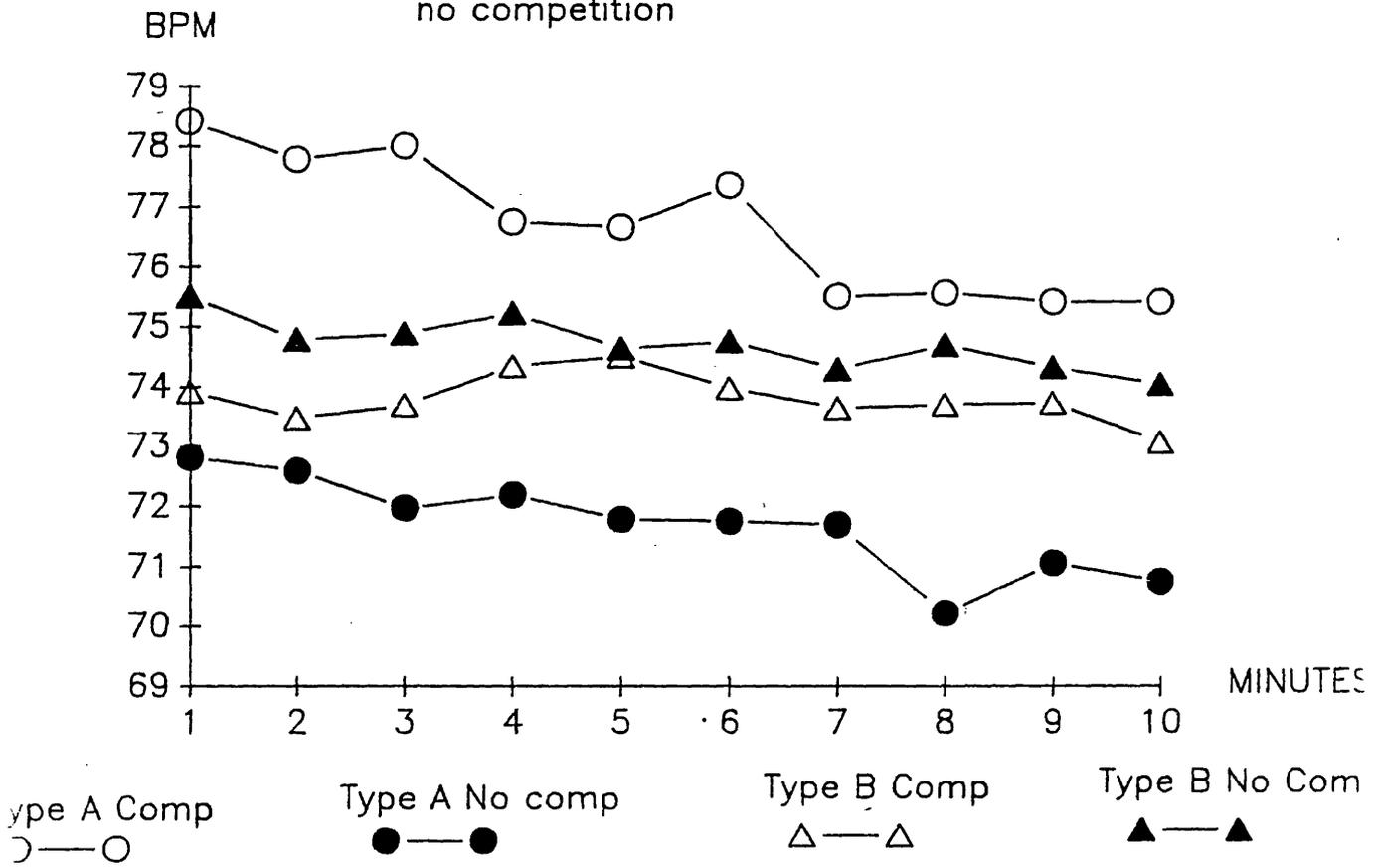
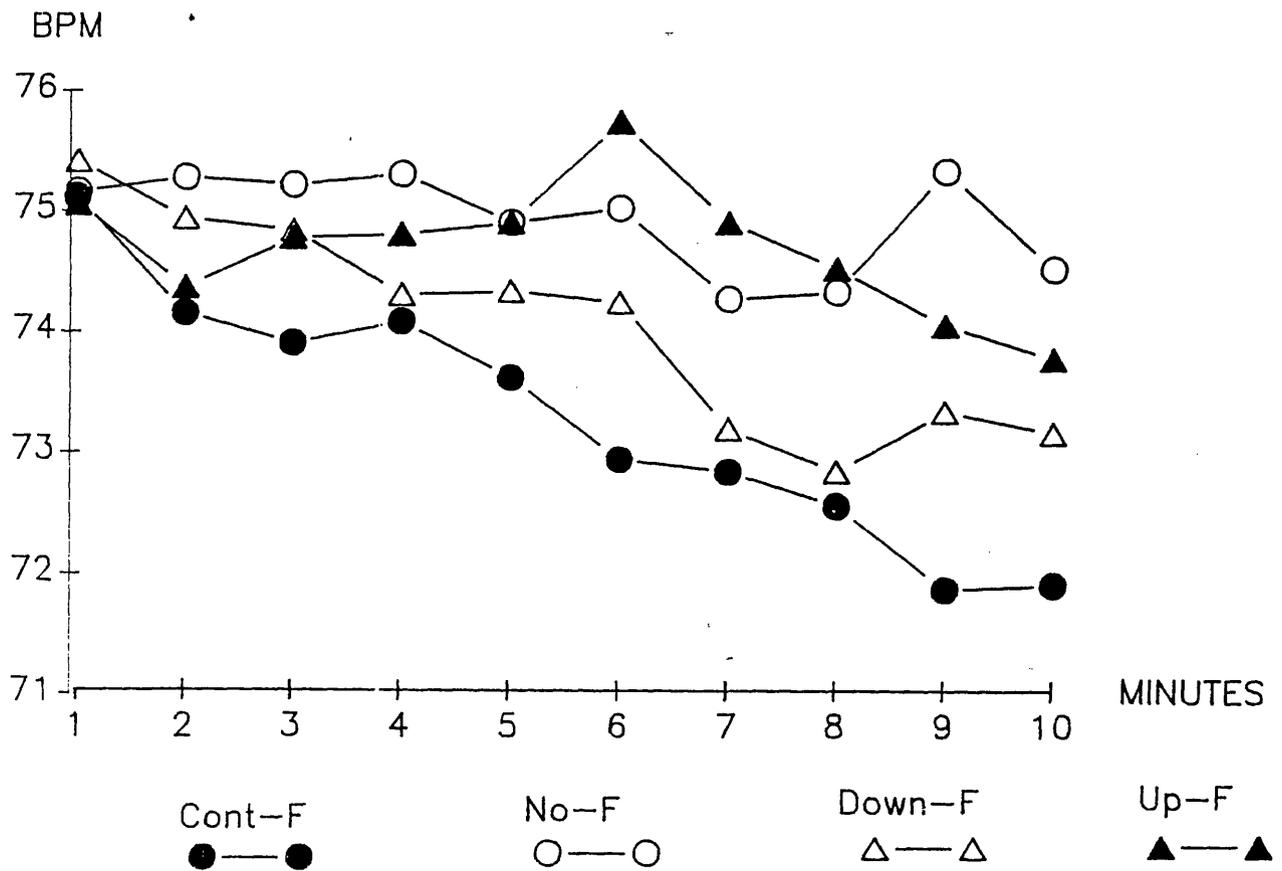


figure 4: Heart rate (BPM) for each feedback condition



The above analysis is complicated because of the complex nature of the design of the experiment. The major problem with the design is the presence of three levels of repeated measures. As a check on the above results by eliminating the principal repeated measure from the analysis, the regression slope of Minutes for each subject was calculated. The b value in the simple regression line formula $y = bx + c$ was calculated where $y =$ beats per minute (BPM) $x =$ minutes (1 to 10) $c =$ constant and $b =$ the gradient of the linear regression line which is the rate of change of heart rate with respect to time. A negative sign indicates a decreasing regression slope.

The GENSTAT5 statistical program was used for the calculation of the b values and the subsequent ANOVA analysis. The ANOVA was a 2 (Type)x 2 (Comp)x 4 (Condition)x 4 (Days) analysis and Table 3 shows the summary table.

Table 3: the ANOVA summary table for b values

Source	df	ss	ms	F	P
Type	1	1.441	1.441	9.71	0.004
Competition	1	0.019	0.019	0.13	ns
Type x Comp	1	0.285	0.285	1.92	ns
Error	28	4.157	0.146	-	-
Days	3	0.383	0.126	0.85	ns
Condition	3	1.710	0.570	3.80	0.014

DaysxType	3	1.669	0.056	0.37	ns
DaysxComp	3	0.141	0.047	0.31	ns
TypexCond	3	0.189	0.063	0.42	ns
CompXCond	3	0.147	0.049	0.33	ns
DaysxTypexComp	3	0.046	0.015	0.10	ns
TypexCompXCond	3	0.129	0.043	0.29	ns
Error	72	10.809	0.150	-	-
Total	127	19.623	-	-	-

Table 4: Mean b values and standard deviations for Type A and B subjects during competition and no competition.

	Mean (sd)
Type A	-0.30 (0.38)
Type B	-0.09 (0.38)
Type A Comp	-0.36 (0.28)
Type A No comp	-0.24 (0.45)
Type B Comp	-0.05 (0.39)
Type B No Comp	-0.12 (0.34)

There is a significant main effect in Type. This confirms the previous analysis suggesting that, overall, Type A subjects have significantly lower b values than Type B subjects. In other words, the rate of decrease of heart rate with respect to time is greater among Type As than Type Bs.

Table 5: Mean b values and standard deviations for subjects in the four feedback conditions.

Condition	Mean (sd)
Cont-F	-0.34 (0.34)
No-F	-0.08 (0.35)
Down-F	-0.27 (0.39)
Up-F	-0.08 (0.43)

Type A Cont-F	-0.41	(0.36)
Type A No-F	-0.15	(0.38)
Type A Down-F	-0.39	(0.36)
Type A Up-F	-0.25	(0.33)
Type B Cont-F	-0.28	(0.30)
Type B No-F	-0.02	(0.33)
Type B Down-F	-0.15	(0.39)
Type B Up-F	-0.08	(0.43)

As can be seen there is a significant main effect in Condition confirming the previous finding of a significant difference between the b values of subjects in the four biofeedback groups. However, the interaction between Type and Condition does not reach significance. This is contrary to the main analysis in which this interaction was significant. Since the present analysis is more conservative, that interaction should be regarded with caution. Nevertheless, table 3 shows clearly that Type A subjects in the competition condition, show a steeper regression slope than their Type B counterparts (-0.36 vs -0.05) and Type A subject in the no competition group (-0.36 vs -0.24).

Finally, a 2x2x4 ANOVA with one repeated measure (Condition) was carried out on the ratings of relaxation made by subjects. This was carried out using the BMDP 4V statistical package. Table 6 shows the summary table.

Table 6: ANOVA summary table for subject's relaxation ratings.

Source	df	ss	ms	F	P
Type	1	18.98	18.98	0.76	ns
Comp	1	38.39	38.39	1.54	ns
TypexComp	1	0.80	0.80	0.03	ns
Error	28	698.92	24.96	-	-
Condition	2.09@	233.09	77.70	9.23	<0.01
ConxType	2.09	9.59	3.20	0.38	ns
ConxComp	2.09	27.05	9.02	1.07	ns
ConxTypexComp	2.09	3.85	1.28	0.15	ns
Error	58.42@	707.27	8.42	-	-

@ Greenhouse-Geisser adjusted degrees of freedom for repeated measures (Greenhouse and Geisser, 1959).

Table 7: Relaxation means and standard deviations

	Mean (sd)
Type A	13.05 (4.28)
Type B	13.82 (2.99)
Type A Competition	12.42 (3.83)
Type A No Competition	13.67 (4.36)
Type B Competition	13.35 (2.72)
Type B No Competition	14.29 (2.66)
Overall Competition	12.88 (3.57)
Overall No Competition	13.98 (3.78)
Cont-F	13.78 (4.04)
No-F	13.00 (3.79)
Down-F	15.34 (2.64)
Up-F	11.60 (3.27)

As can be seen from tables 6 and 7 the only significant effect is that there is an overall difference in the relaxation ratings made by subjects in the four feedback conditions. It seems that subjects in the Down-F condition reported significantly more relaxation than any other group. Subjects in the Up-F condition reported less relaxation than other groups. Interestingly, the relaxation ratings of the Cont-F and the No-F groups are almost identical.

4.4. DISCUSSION

The results of the present experiment concerning the role of contingent and non contingent feedback are quite straight forward. Results indicate that subjects who were given contingent heart rate biofeedback reduced heart rate significantly more than subjects receiving no feedback at all, thus confirming previous research suggesting that biofeedback is an effective tool for self control of heart rate (Engel and Chism, 1967; Brener, 1974; Engel and Bleeker, 1975). In view of the relatively short period of biofeedback training provided (ten minutes), the results are very striking. Secondly, false feedback showing heart rate going down (Down-F) was significantly more effective than false feedback showing heart rate going up (Up-F). The results reinforce the view that there is a major suggestibility component to the feedback paradigm (Shahidi and Powell, 1988). It is interesting to note that similar

manipulations and procedures as those described in the present experiment have been used in therapeutic procedures to eliminate maladaptive emotional behaviour (eg Wolpe, 1959; Lang and Lazovik, 1963). Valins (1966) suggests that manipulation of the cognitive representation of internal events may be the primary factor contributing the successful treatment of phobic patients and the present study reinforces that important point.

More importantly, the results showed that non contingent biofeedback was as effective as contingent feedback in the sense that subjects who were presented with false feedback suggesting that heart rate was going down reduced heart rate to the same extent as subjects who were given true contingent heart rate biofeedback. This finding supports the claim that the biofeedback paradigm operates through mechanisms other than operant conditioning or skill acquisition, at least under the conditions of the present experiment. This, to some extent, reinforces the view put forward by Stroebel and Glueck (1973) who have argued that many of the effects produced by biofeedback may be explained by recourse to a placebo effect. It may be argued that awareness of one's physiological state via biofeedback, whether contingent or not, may at least result in some internal control.

Although subjects in the Down-F group reported themselves to be more relaxed than subjects in the Up-F group, subjects receiving contingent heart rate did not report themselves to be any more relaxed than subjects receiving no feedback at all. This finding seems to refute the assumption by some authors (eg Scott, etal 1973; Lang, 1969) that lowered heart rate would lead to lowered tension and heightened relaxation. It seems that learning to reduce heart rate does not necessarily lead to reports of more relaxation by subjects. There were no other significant effects in the subjective reports of relaxation. However, the results must be viewed with caution since the relaxation ratings were taken at the end of each session and ratings at the beginning were not made. In any case the measurement of relaxation was done in a crude manner (single rating scale). The clinical importance of the effective measurement of both subjective and physiological aspects of relaxation has been emphasized by some authors (eg Prigatano and Johnson, 1972). In other words, it is clinically useful if both subjective and physiological measures are available. The reliable measurement of subjective reports of relaxation needs to be investigated systematically in future experiments.

The results of the present research concerning the effects of biofeedback upon heart rate reduction in Type A and B subjects suggests that Type A individuals are generally

better able to reduce their heart rate than their Type B counterparts. This effect was observed regardless of whether or not subjects were presented with contingent or non-contingent biofeedback. Further, the hypothesis that Type A subjects would be less successful than the other groups in the Up-F condition was not supported. Biofeedback training seemed to significantly facilitate heart rate reduction for both Type A and B individuals. This result is contrary to other findings in the field where Type As have been only successful in the presence of biofeedback (Stern and Elder, 1982; Prior, et al 1982; Anchor, et al 1979). The reason may be the relatively short period of biofeedback training provided in the present experiment.

Analysis of subjects' heart rate across time indicated that during competition, Type As reduced heart rate more during competition than during no competition. Type B individuals were less able than Type A subjects to reduce heart rate whether competing or not. However, the interaction with competition could not be confirmed statistically when b values were analyzed suggesting that the above results should be treated with caution until replicated with larger numbers. Nevertheless, the present results suggest the importance of challenging incentives in the modification of physiological responses in healthy Type A subjects (Stern and Elder, 1982). In the present experiment, competitive instruction seems to have motivated Type As to reduce heart

rate successfully when normally the opposite would be expected (ie heart rate increase). The results, therefore, have implications for the modification of Type A behaviour pattern in healthy Type A individuals. As mentioned in chapter one, attempts to modify Type A in healthy subjects have produced mixed results suggesting that compared to individuals already suffering from CHD, the behaviour pattern is much more difficult to alter in healthy individuals (Haaga, 1987; Friedman, Thoresen and Gill, 1981). It is reasonable to argue that in the light of the present results an alternative approach might be to use Type A characteristics such as competition in order to motivate Type A subjects to reduce stress related physiological symptoms. Together with the pioneering work of Stern and Elder (1982), the present research takes a significant step in this direction. Several questions, however, remain to be answered in future research.

One question which must be addressed concerns the generalizability of heart rate reduction to other cardiovascular symptoms of Type A. For example, research suggests that a significant precursor of CHD is the excess excretion of catecholamines (Glass, 1977, Friedman and Rosenman, 1964). One possible index of catecholamine excretion may be the transit time of the pulse wave (Obrist, etal 1978). Future research should concentrate on discovering whether or not challenging incentives facilitate

pulse transit time increases followed by heart rate reduction in Type A individuals. Furthermore, the question of whether or not reductions in heart rate achieved by the use of challenging incentives would actually generalize to aspects of Type A behaviour warrants further investigation.

The above discussions and suggestions may be even more valuable if the focus of research was shifted from adults to children and adolescents. As discussed previously, healthy Type A individuals may be very resistant to change either because the negative effects of the Type A behaviour pattern are unknown to them or these effects may not seem relevant since their health and success may outweigh the risk of illness (Haaga, 1987). Furthermore, the "habit" of exhibiting the Type A behaviour pattern may be extremely difficult to overcome after the individual has reached adulthood (Price, 1983; Roskies, 1986). Because of these problems, the importance of concentrating on the younger population has been emphasized by Price (1983). However, any modification programme will be faced with several difficulties. Firstly, and most importantly, although the Type A behaviour pattern has been measured in adults and adolescents, to date, there has been no reliable theoretical framework to guide researchers. It is not known how the behaviour pattern is maintained and encouraged in the younger population. Secondly, it may be difficult to obtain the consent or active participation of significant adults

which is so important to the success of any modification attempt (Price, 1983). Secondly, the ethical issues which are involved in attempting to change behaviour in children and adolescents pose the question of how the behaviour pattern should be modified. For example, since Type A children seem to be high achievers at school (Matthews and Woodall, 1988), would reduction in Type A characteristics have any impact on achievement? With regard to these issues, the present research seems to offer an ideal alternative for the modification of physiological responses in the younger population.

The ultimate aim of the present research is to discover if challenging incentives, for example, competition, could be manipulated in the same way as described above so that Type A adolescents are encouraged to relax rather than exhibit increased physiological responses. However, what is missing in the field is a comprehensive understanding of Type A behaviour in adolescents. The aim of the next study is to bridge this gap by concentrating on examining the role played by the core beliefs and fears thought to maintain the Type A behaviour pattern through out a person's life and to see whether or not Type A adolescents actually hold these

beliefs and fears. If the association between these beliefs and fears and their relationship with Type A is indeed similar between adults and adolescents, the next step will be to discover if the effects of using competition as a challenging incentive to help Type A adults relax are observed in Type A adolescents.

CHAPTER FIVE

BELIEFS AND FEARS UNDERLYING THE TYPE A BEHAVIOUR PATTERN IN ADOLESCENTS.

5.1. INTRODUCTION

Recent research has revealed that the origins of atherosclerosis, which was discussed in some depth in chapter one, can be traced back to early childhood (McGill, 1984). Fatty acids are seen in the aortas of even three year olds and are thought to become grossly visible microscopically in the coronary arteries after the age of ten (Matthews and Woodall, 1988). By the age of twenty, fibrous plaques begin to appear. Beginning in the 15 to 24 age group, white men have about twice as many raised lesions as white women at every age, a relationship paralleled by sex differences in CHD mortality twenty years later (Matthews and Woodall, 1988). Therefore, some researchers have argued that high levels of lipids, lipoproteins and blood pressure in children and young adults may accelerate the progression of atherosclerosis in the second and third decades of life (Berenson, 1980). In fact a number of epidemiological studies have shown that children and adolescents can be reliably classified on the major biological and behavioural characteristics of adults at risk of CHD (eg. Clarke, etal 1978). Type A as a behavioural risk

factor in children has been found to predict increased cholesterol in adults (Hubert, etal, 1987). It has been shown that both biological and behavioural risk factors are relatively stable across time. In particular, it has been found that the Type A behaviour pattern which can be identified in childhood can also be recognized later in life. In this chapter a review of the research on the early origins of the Type A behaviour pattern will be given. In this context, the genetic and familial factors will be briefly discussed. The present chapter will also emphasize the importance of identifying and modifying the Type A behaviour pattern in children and adolescents.

5.1.1 Identifying the Type A behaviour pattern in children and adolescents

The Type A behaviour pattern in children and adolescents has been identified and measured in recent years. Matthews and Angulo (1980) have developed a teacher rating scale. This rating scale known as the Matthews Youth Test for Health (MYTH) consists of 17 questions measuring Type A in children as young as five years old. Teachers are asked to rate children's hostility, competitiveness and impatience. Based on a sample of 485 children aged between 5 to 10 years old a test retest reliability of 0.82 has been reported for the total Type A score after a period of three months. Also a

very high internal consistency (Cronbach's alpha= 0.90) for the MYTH has been established (Matthews and Angulo, 1980).

Wolf, etal (1982) have developed a self report measure of the Type A behaviour pattern in 10 to 11 year old children. The scale is based on the Bortner scale described in previous chapters. The measure consists of 24 self anchoring items in the form of a seven rung ladder which was developed to convey the idea about different forms of behaviour on a continuum. A test retest (6 weeks) reliability of 0.53 has been reported.

Siegel, etal (1981) have developed a structured interview which aims to measure the Type A behaviour pattern in adolescents known as the adolescent structured interview (ASI). This is a modified version of the original adult structured interview developed by Friedman and Rosenman (1960). The format of some of the questions is altered and the mannerisms of the interviewer are slightly different from the original version. Interviewer agreement of 75% has been reported. The correlation between the ASI and the Bortner scale is 0.57.

Based on these instruments, Type A children and adolescents have been the subject of a great deal of recent research. Most studies have concentrated on behavioural and physiological differences between Type A and B children and

comparing the findings with their adult counterparts. The results generally suggest that, like Type A adults, Type A children and adolescents make greater effort to assert environmental control (Matthews, 1979) and are aggressive and competitive during play with others (Wolf, etal 1982; Matthews and Angulo, 1980). Furthermore, Type A children and adolescents have been found to express more anger (Thoresen and Pattillo, 1988) and more symptoms of stress (Eaglestone, etal 1986) than their Type B counterparts.

It is interesting to note that recent research on overt Type A in children and adolescents suggests that the behaviour pattern seems to be composed of a prosocial dimension, best characterized as achievement striving, and an antisocial dimension, characterized as impatience/aggression (Steinberg, 1986; Matthews and Woodall, 1988). In this context it has also been found that Type A children and adolescents tend to receive better grades than Type Bs (Bachman etal 1986). Studies on the social competence of young Type As, however, have revealed a somewhat bleak picture. These studies suggest that Type A children and adolescents, mainly due to the impatience aggression component, tend to lack certain social skills that may make them unlikable (Matthews and Woodall, 1988). It has also been found that Type As are seen by their peers to be more noisy, causing trouble, less fun to be with and making more negative contact with others (Whalen and Hencker, 1986).

These findings are important because they provide strong support for the existence of the Type A behaviour pattern in the younger population since the behaviours observed in children and adolescents are consistent with those behaviours seen in Type A adults.

5.1.2 The stability of the Type A behaviour pattern

The stability of the Type A behaviour pattern has been examined by several investigators. It has been found, for example, that aggression and competition are more stable in boys than in girls (Block, 1971). Several studies have specifically investigated the stability of the behaviour pattern across time. Bergmann and Magnusson (1986) found that Type A related behaviours rated by class room teachers when the subjects were 13 years old correlated significantly with subjects' self reported Type A fourteen years later.

In a five year longitudinal study, Visintainer and Matthews (1988) followed up children from kindergarten age up to the age of 12 by gathering annual ratings of Type A from their teachers. It was found that total Type A scores were significantly correlated across the duration of the study, as were ratings of competition and impatience aggression. In a similar study, Steinberg (1986) examined the stability of overt type A behaviour from childhood to young adulthood.

Results showed that the behaviour pattern was stable between adolescence and adulthood but not between childhood and adulthood.

Therefore, it seems that despite the fact that overt Type A behaviour seems to be relatively stable during various stages of life, it is only possible to predict reliably which Type A adolescents as opposed to children are likely to become Type A adults. The Type A behaviour pattern between childhood and adulthood does not seem to enjoy continuity. The conclusion seems to be that although there may be Type A children, these children do not necessarily grow up to be Type A adults.

5.1.3 The etiology of the Type A behaviour pattern

Twin and family studies have aimed to examine the extent of familial similarity of Type A (Matthews and Woodall, 1988). Generally, there seems to be disagreement among researchers as to whether or not there is a genetic component in the Type A behaviour pattern. For example, Matthews and Krantz (1976) studied adult twins and concluded that there is a genetic component in the Type A behaviour pattern even in twins reared apart. However, Rahe, Hervig and Rosenman (1975) reported no evidence of a genetic component of the Type A behaviour pattern.

The most recent study on the etiology of the Type A behaviour pattern was reported by Pederson, et al (1989) who studied twins reared together and apart in Sweden. It was found that heritability (the proportion of total variance due to genetic effects) was significant for competition and ambition (40%) but not for hostility (12%). Furthermore, the study suggested that there is an important environmental as well as a genetic effect. 60% of the variance could be attributed to non shared environmental experiences unique to the individual. Indeed, some researchers have pointed out that even if the type A behaviour pattern should have a heritable base, early environmental factors are likely to play a significant role in the expression of Type A characteristics (Price, 1983; Matthews and Woodall, 1988).

The above assertion comes mainly from family studies which have found correlations between the Type A behaviour pattern in parents and that of their children (Matthews, 1977; Matthews and Siegel, 1983). For example, observational studies have shown that mothers of Type A children make infrequent positive remarks, as compared to mothers of Type B children, about their children's performance on achievement tasks, despite the fact that Type A children tend to perform better than Type B children (Glass, 1977). In another study, Brack (1986) found that parents of Type A children tended to push their children to perform better. Parents of Type A children were more critical and exhibited

more tension than parents of Type B children. Furthermore, they were more likely to evaluate the performance of their children through comparison to the children's peers during task performance. It has also been found that even when children interact with women other than their mothers, they are more often encouraged to do better than Type B children are (Matthews, 1977).

Although no cause and effect conclusions can be drawn from the above studies, these results do provide a picture of how the Type A behaviour pattern may be encouraged and indeed maintained through the family. Price (1983) has developed a social learning model of the acquisition and maintenance of the Type A behaviour pattern which was discussed briefly in chapter one. Price argues that the Type A behaviour pattern may be learned through the family in different ways. The most important ways are through instruction and social modelling. There is a great deal of evidence which shows that Type A parents seem to have Type A children. In a study of over 200 families, mothers' and fathers' Type A scores were significantly associated with the scores of their elementary school aged children (Matthews, etal 1986). Although data seem to suggest that boys' and not girls' Type A may be related to similar behaviours in their parents (Weidner, etal, 1988), some researchers have argued that one important way in which the Type A behaviour pattern is transmitted to the individual is through the family.

Price (1983) argues that apart from the family, other factors influence the etiology of the Type A behaviour pattern. These include the mass media (particularly television) and the educational system. She points out that a great deal of television programmes are based on promoting characters who are portrayed to be young, aggressive, assertive and violent. The emphasis yet again seems to be on material success and the importance of working and competing hard in order to be successful and popular.

Schools as primary socializing agents teach competitive achievement striving from a child's earliest age throughout his academic life. As Price points out, the child who achieves high grades generally receives high recognition, special privileges, and the security which derives from knowing that he is considered valuable and worthwhile. Hence, a great number of children are taught to compete for high grades. Also a sense of time urgency is reinforced by the emphasis on rapid learning and performance (Cohen, etal 1978).

Based on these observations, Price (1983) has proposed a set of beliefs and accompanying fears which are thought to maintain and encourage the Type A behaviour pattern. The cognitive social learning theory of Type A has already been discussed elsewhere. Here a detailed outline of the specific

beliefs and fears will be given together with studies done to test Price's theory.

5.1.4 Beliefs and fears underlying Type A

According to Price, there are four beliefs accompanied by three fears. The first belief is that the individual must constantly prove himself. Since society strongly encourages achievement and material success by emphasizing the importance of individual effort and competitive spirit, it may be that positive self evaluation is seen by the individual a function of material success. Therefore, personal esteem may be equated with the number and the quality of achievements. If the person believes that the way in which people evaluate him depends on his achievements, then it is reasonable to postulate that the individual may develop a fear of being judged as not having sufficient worth and therefore not being valued or esteemed (Price, 1983).

A second belief is that there is no ordinary relationship between intentionality and consequences. Hence, the person believes that no universal moral principles exist (Price, 1983). The accompanying fear is that good may not prevail. In other words, the person fears that good actions may produce negative consequences. This belief that good individuals never win is indeed a very frightening one in a

society where losing is severely punished and failure is penalized.

Similar to the belief of good not prevailing is the belief that justice may not prevail. This in effect means that it is up to the individual to ensure justice for himself. The notion of revenge is commonly observed in Type As (Van Egeren, 1979). Revenge behaviour can be seen as consciously arranging aversive consequences to match or compensate previously experiences consequences (Price, 1983). Thus, the belief that justice may not prevail may result in a vengeful behaviour which could be an important source of hostility in Type A individuals.

According to Price's theory, the fourth belief is that all resources worth having are in limited supply. The individual believes that because the resources are scarce, others' gain means the individual's loss. The fear of insufficient supply which accompanies this belief results in the individual trying harder and harder and competing more and more in order to gain his fair share.

In order to explain the importance of these beliefs and fears, Price (1983) has argued that the Type A behaviour pattern can be viewed as an iceberg. Observable speech and psychomotor behaviours indicative of Type A constitute the tip of this iceberg, the proportion which is visible above

the water. The layer just beneath the surface is comprised of psychological and interpersonal characteristics and behaviours such as ambition and competition. But the foundation of the iceberg, the bottom layer hidden from view, consists of the cognitive basis for the Type A behaviour pattern, the beliefs and fears which maintain the behaviour pattern. These are the hardest to get at and the most difficult to change. Price believes that the individual copes with these fears through Type A behaviour. It is hypothesized that a great deal of these beliefs and fears are not necessarily in the person's awareness. It is further believed that these seven beliefs and fears form a constellation and therefore should be positively inter correlated.

To date there has only been one attempt to empirically test Price's cognitive social learning model. In a series of studies, Burke (1984 a,b) and Burke and Deszca (1984) developed a 50 item questionnaire aimed at measuring the beliefs and fears underlying the Type A behaviour pattern in adults and validating them against Type A measures. In one study, Burke (1984 a), 220 police officers (mean age= 36 years) responded to this questionnaire. The beliefs and fears were assessed together with four measures of the Type A behaviour pattern, measures using the 52 item Jenkins Activity Survey (JAS). High reliabilities were reported for each of the beliefs and fears ranging from 0.42 for the fear

that good may not prevail to 0.88 for the fear that the individual may not get his share. The average reliability for the seven beliefs and fears was 0.73. In a replication of the study, Burke (1984 b) based on 137 subjects an average reliability of 0.77 was reported. However, only low correlations were obtained between the beliefs and fears and Type A measures. The beliefs and fears did not correlate significantly with overall Type A scores except for the belief that self worth is a function of one's accomplishments. ($r= 0.13$). The correlation between overall beliefs and overall Type A scores was significant but low ($r= 0.11$).

It seems that two subscales of the JAS correlated most strongly with the measures of beliefs and fears. Time urgency correlated significantly, albeit moderately, with nearly all the beliefs and fears with an average correlation of 0.21 (Burke, 1984 b). Speed and impatience correlated significantly with all the beliefs and fears with an average of 0.16 (Burke, 1984 a).

Burke (1984 b) concludes that the relationship between beliefs and fears underlying the Type A behaviour pattern and Type A scores provide modest validation for Price's hypotheses. However, Burke (1984 a) points out the fact that although some aspects of the Type A behaviour pattern such as time urgency seem to support Price's model, other aspects

such as job involvement produced results opposite to expectations. Furthermore, none of the correlations reported in Burke's studies exceed 0.30.

Although Burke's findings do not seem to be as conclusive in supporting the cognitive social learning model as expected, they are suggestive nevertheless. It must be pointed out that further research on different populations with more participants should be undertaken. These different populations should include non American participants and children and adolescent populations.

Based on the strong evidence suggesting that the Type A behaviour pattern does exist in the younger population and the fact that the first signs of CHD can be detected at a very early age, Price (1983) has strongly recommended the examination of these beliefs and fears in the younger population. The aim of the present research is to test Price's cognitive social learning theory in adolescents. This will be done by administering measures of beliefs and fears proposed by Price and correlating them against two measures of Type A, namely the Bortner Adjective Rating Scale (BARS) and the student version of the Jenkins Activity Survey (SJAS). The BARS has been used in various formats with children and adolescents (Wolf, 1982) and the SJAS has been developed specifically as a modified version of the

adult JAS and is particularly suitable for the younger population (Glass, 1977; Yarnold and Mueser, 1988).

It is hypothesized that the measures of beliefs and fears will form a constellation (Price, 1983; Burke and Deszca, 1984). In other words, the beliefs will correlate significantly with other beliefs and that fears will correlate significantly with other fears. Also the beliefs and fears will correlate with each other. Further, that the measures of beliefs and fears will possess acceptable levels of reliability. Secondly, it is predicted that the beliefs and fears will correlate significantly with the measures of the Type A behaviour pattern.

5.2. METHOD

5.2.1 Subjects

Subjects were 350 pupils from three private secondary schools in the London area. Of these 23 were eliminated from the sample either because they failed to complete a large proportion of the questionnaires or because parental permission could not be obtained. Therefore, 327 pupils were included in the sample. The mean age was 14.85 years (range 12 to 18 years). There were 266 males and 61 females in the final sample.

5.2.2 Materials

Subjects completed the student version of the Jenkins Activity Survey (SJAS) (Jenkins, etal 1965) and the Bortner Adjective Rating Scale (BARS) (Bortner, 1969). Additionally, a measure of beliefs and fears was developed and administered.

5.2.2.1 The Bortner Adjective Rating Scale (BARS)

The 14 item Bortner scale has already been fully described in chapter one. BARS has been widely used with adolescents (Wolf, etal 1982; Steinberg, 1981) since it is a very simple questionnaire to score and administer. Furthermore, the questions are easy to understand by the subject (O'Looney, 1984). The same version that is used with adult populations was used in the present study. Only one item was slightly altered: item fourteen on the questionnaire "satisfied with job" was changed to "satisfied with studies" (for a copy of the BARS see appendix 8).

5.2.2.2 The student Jenkins Activity Survey (SJAS)

The SJAS is a self report measure of Type A behaviour which was modelled after the adult version of the JAS (Jenkins, etal 1965) except that items on the adult JAS measuring job involvement were deleted or modified in the SJAS (Glass,

1977). The SJAS consists of 44 multiple choice items having two to four response alternatives. It is common practice, however, to use the short version of the SJAS, as done in the present study, by computing a total A/B score using a unit weighting procedure with 21 of the items (Yarnold and Mueser, 1988) The other items are ignored. Therefore, individuals may be located on an A/B continuum ranging from extreme Type B (a score of zero) to extreme Type A (a score of 21). In addition, based on a sample of 4072 undergraduate students, Bryant and Yarnold (1988) have validated the existence of two subscale scores (speed and hostility, 4 items and hard driving and competitive, 11 items).

Yarnold et al (1986) have reported high test-retest reliability for the SJAS ranging from 0.96 over a two week interval to 0.70 over a three month interval. The SJAS has moderate internal consistency, with Cronbach's alpha ranging from 0.40 to 0.72 (Yarnold and Mueser, 1988). For a copy of the SJAS see appendix 9).

5.2.2.3 The Beliefs and Fears Questionnaire

The original 50 item questionnaire compiled by Burke and Deszca (1984) aimed at measuring beliefs and fears underlying the Type A behaviour pattern in adults was used. The questionnaire was first administered to 15 adolescents

(mean age= 13.2, age range= 12 to 15 years) in order to determine if the questionnaire was suitable for this population. These respondents were selected randomly from three independent secondary schools. Respondents were asked which questions, words or phrases they found difficult to understand. Based on their responses, ten of the questions were deleted and several questions were modified until it was decided that the questionnaire was suitable for the population for which it was intended. The following two questions are examples of the ten deleted questions: "A person who has not achieved economic prosperity is often considered a failure as a human being" and "I worry about how often my own actions and behaviour are guided by expediency rather than standing up for what I believe is right". The following statement is an example of a modified question: The statement "there is little inherently valuable or good about a human life outside of effort and successful accomplishments" was changed to "what is really good and valuable in a person's life is hard work and success". For a comparison of Burke's original questionnaire and the modified version used in the present study refer to appendix 10).

The following beliefs and fears were included in the questionnaire: 1. The belief that one's worth is a function of one's material achievements (6 items) 2. The fear of negative evaluation (6 items) 3. The belief that no

universal moral principles exist (5 items) 4. The fear that justice may not prevail (4 items) 5. The belief in revenge (5 items) 6. The belief that things worth having are in limited supply (8 items) 7. The fear that the individual may not get his share (6 items).

Subjects had to indicate to what extent they agreed or disagreed with each item. Each item was followed by five choices ranging from 1 representing "very true" to 5 representing "not at all true". Therefore, a minimum total score of 40 meant complete agreement with the beliefs and fears and a total maximum score of 200 meant complete disagreement.

5.2.3 Procedure

Forty five private secondary schools in and around London were contacted through letter asking the Headmaster/mistress whether or not they would be willing to allow some of their pupils to take part in the study. A description of the study, together with general information concerning the Type A behaviour pattern and copies of questionnaires were enclosed. The reason for contacting private schools was that it was decided that contacting other schools through Education Authorities would take too much time.

Out of the forty five schools which were contacted, thirty schools replied and six asked for more information or an interview. Of these three schools agreed to allow their pupils to participate in the study. Subjects' consent and parental permission was obtained by the teachers of respective schools. A large proportion of subjects (n=219) came from a large boys' school which accounts for the large difference in the number of males and females. The other two schools were mixed.

Testing was conducted in two sessions. In the first session (respondents were allowed a maximum of 30 minutes to complete questionnaires), pupils completed the BARS followed by the belief and fears questionnaire. The following day pupils completed the SJAS (subjects were allowed 20 minutes to complete the questionnaire). This arrangement was requested by the school authorities so that disruption to school time table would be minimized. The questionnaires were administered by teachers who were fully briefed as to what instructions to give. Subjects were told that the study was concerned with attitudes towards aspects of every day life and about how people would behave in certain situations. They were told that there were no right or wrong answers and that they shouldn't take too long on any question. Furthermore, subjects were assured of confidentiality and were told that their individual responses would not be seen by any member of staff at the

schools. This was at the end of a lesson in classrooms containing 10 to 30 pupils each.

5.3. RESULTS

5.3.1 Relationship among beliefs and fears

Table 1 shows means and standard deviations for each of the beliefs and fears. Internal reliabilities in terms of Cronbach's alpha (Cronbach, 1951) were computed using the SPSSx statistical computer package. The alpha reliabilities are also shown in table 1.

Table 1: Means, standard deviations, and internal reliabilities for beliefs and fears

BEL1 self worth is a function of one's achievements
 FEAR1 fear of negative evaluation
 BEL2 no universal moral principles exist
 FEAR2 fear that justice may not prevail
 REV revenge
 BEL3 things worth having are in limited supply
 FEAR3 fear that the individual may not get his share
 BELIEF total beliefs
 FEAR total fears
 OVERALL total beliefs and fears

	No. items	Mean	Sd	alpha
BEL1	6	20.68	4.44	0.65
FEAR1	6	19.36	4.90	0.68
BEL2	5	14.50	3.24	0.24
FEAR2	4	11.56	3.01	0.45
REV	5	15.03	4.40	0.70
BEL3	8	26.01	5.96	0.70
FEAR3	6	19.39	6.08	0.84
BELIEF	24	76.21	13.03	0.80
FEAR	16	50.31	11.55	0.85
OVERALL	40	126.52	18.28	0.82

In order to see if alpha values for BEL2 and FEAR2 can be improved, items were eliminated and alphas recalculated.

For BEL2 eliminating one item (question 32) improved the alpha value but only from 0.24 to 0.32. For FEAR2

elimination of items did not improve the alpha value of **0.46**

As can be seen from table 1, all the beliefs and fears possess reasonably high alpha values with the exception of BEL2 (no universal moral principles exist) and possibly FEAR2 (fear that justice may not prevail).

According to Price (1983) and Burke (1984 b), the seven beliefs and fears should form a constellation and should therefore be positively interrelated. Table 2 shows the correlation matrix of the beliefs and fears.

Table 2: correlations between beliefs and fears

	BEL1	FEAR1	BEL2	FEAR2	REV	BEL3	FEAR3
BEL1	1.00						
FEAR1	0.05	1.00					
BEL2	0.24*	0.04	1.00				
FEAR2	0.07	0.29*	0.19*	1.00			
REV	0.36*	-0.12#	0.25*	-0.03	1.00		
BEL3	0.48*	0.13*	0.27*	0.30*	0.39*	1.00	
FEAR3	-0.02	0.68*	0.12#	0.40*	-0.07	0.13*	1.00

P<0.05 * P<0.01

As can be seen, of the 21 correlations, 18 are positive and 15 are statistically significant (71.4%). The average intercorrelation is 0.20 ranging from -0.12 to 0.68. As expected, there are significant correlations between different beliefs and between different fears. An interesting correlation is that between FEAR1 and REV

($r = -0.12$). This in effect means that as the fear of negative evaluation increases, the less the individual believes in a philosophy of revenge.

A principal components analysis with varimax rotation was performed on the seven measures using the GENSTAT statistical computer program. The scree test (figure 1) revealed two clearly separate components which accounted for 27% of the total variance and had eigenvalues of 5.8 and 4.9 respectively. These were retained for rotation. Items loading more than 0.30 were used to interpret the components. The first component contained 15 questions, 13 of which were fears and two of which were beliefs. This component was labelled "fear". The second component contained 18 questions all of which were belief questions. This component was labelled "belief". Seven questions (4 fear and 3 belief) did not emerge in either component. This is shown in table 3.

figure 1: The Scree Test

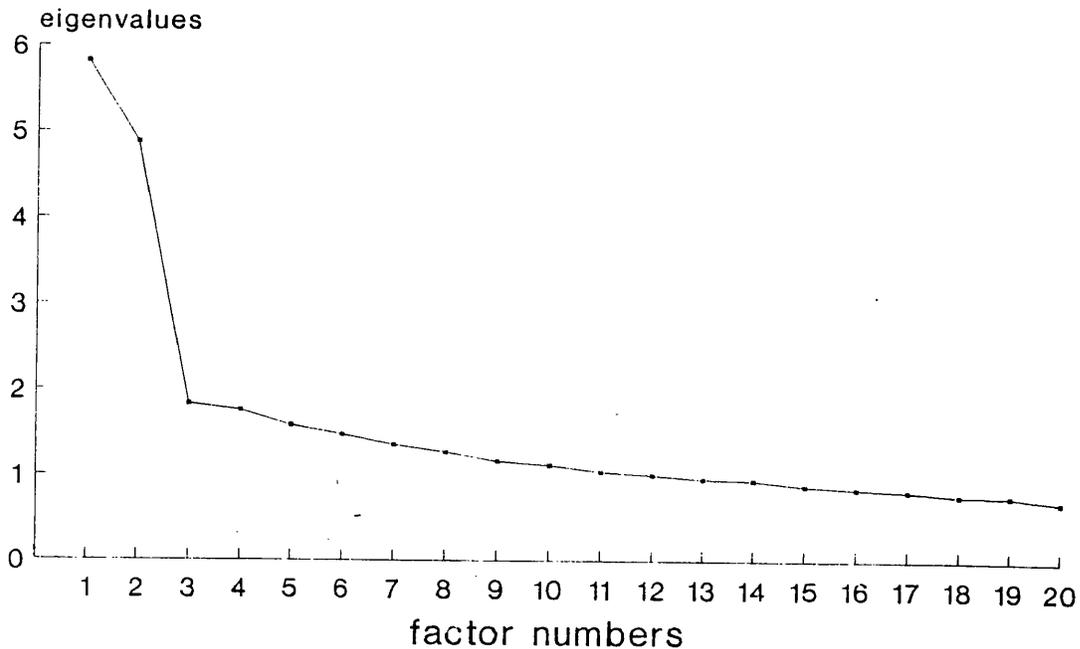


Table 3: Principal components analysis

	Component I FEAR	Component II BELIEF	
I worry about not making it	-0.83	----	I worry about nice guys finishing last
I worry that I may not get the things I want	-0.77	----	When others do well, I am likely to be judged as less able
I worry that I may not be able to be successful	-0.76	----	Revenge is sweet
I worry that I am not talented enough to be successful	-0.73	----	When someone hurts me, I will get even
I worry that I may not be as good as most people	-0.73	----	I believe that your gain is my loss
I worry that I might not be smart enough to make it	-0.68	----	The way to get what you want is to do better than others
I worry that I may not be as worthy as most people	-0.65	----	My chances of being successful increase as others fail
I worry that it will take too long to get the things I want	-0.56	----	People should have respect for those who are successful
I worry that good things won't last	-0.53	----	You can tell how worthy a person is by achievements
I worry about what other people think of me	-0.51	----	A lot of my friends are rivals for the things I want
My friend think I have confidence but I don't	-0.43	----	A person's value or worth depends on the things he owns
I worry about what my teachers think about me	-0.42	----	It is better to get revenge than to forgive and forget
When I do something, the result is different from what I expect	-0.39	----	There aren't enough goodies for everyone.

I don't think there are enough goodies in life	-----	-0.39
People are measured by what they own	-----	-0.37
You have to achieve a lot to be a success	-----	-0.35
I think that if someone does something wrong, he should pay	-----	-0.33
There aren't enough goodies to go round	-----	-0.33
No one can tell you what is good or bad	-----	-----
Doing what is good doesn't always make you happy	-----	-----
I worry that real justice doesn't exist.	-----	-----
I believe that criminals should get what they deserve	-----	-----
I don't usually do what religion tells me to	-----	-----
I worry about guilty people being found innocent	-----	-----
It is important to make other people like me	-----	-----

5.3.2 Relationship among the Type A measures

Table 4: Means and standard deviations for the Type A measures

BARS Bortner Adjective Rating Scale
 SJAS Student Jenkins Activity Survey
 COMP Competition/ambition scale of the SJAS
 HOST Hostility/time urgency scale of the SJAS

	No of items	Mean	Sd
BARS	14	60.30	8.56
SJAS	21	7.83	3.61
COMP	11	4.21	2.34
HOST	4	1.51	1.10

Table 6(a): Relationship of each belief & fear with
 Type A measures (the effects of other beliefs & fears
 have been controlled for using partial correlations)

	BARS	SJAS	COMP	HOST
BEL1	-0.10	-0.08	-0.08	-0.07
FEAR1	-0.19*	-0.14*	-0.15*	-0.05
BEL2	-0.01	-0.04	-0.05	-0.08
FEAR2	-0.03	-0.06	-0.07	-0.02
REV	-0.04	-0.03	-0.02	-0.12@
BEL3	-0.09	-0.10	-0.02	-0.14*
FEAR3	-0.00	-0.10	-0.16*	-0.03
BELIEF	-0.15*	-0.17*	-0.09	-0.17*
FEAR	-0.19*	-0.06	-0.00	-0.04
OVERALL	-0.30*	-0.19*	-0.07	-0.18*

@ P<0.05

* P<0.01

Table 5: Correlations between the Type A measures

	BARS	SJAS	COMP	HOST
BARS	1.00			
SJAS	0.52	1.00		
COMP	0.48	0.85	1.00	
HOST	0.28	0.53	0.19	1.00

As can be seen from Table 5, there is reasonably high correlations between the BARS and the SJAS and between the competition/ambition subscale of the SJAS and the two measures of Type A. However, the hostility/time urgency subscale of the SJAS seems to correlate poorly with the BARS and the competition/ambition subscale.

5.3.3 Relationship between the beliefs and fears and Type A measures

Table 6: Relationship of beliefs and fears to Type A measures (N= 327)

	BARS	SJAS	COMP	HOST
BEL1	-0.20*	-0.18*	-0.12#	-0.07
FEAR1	-0.27*	-0.13*	-0.08	-0.04
BEL2	-0.06	-0.02	-0.02	-0.14*
FEAR2	-0.08	-0.10	-0.05	-0.04

REV	-0.10	-0.10	-0.04	-0.19*
BEL3	-0.21*	-0.20*	-0.08	-0.21*
FEAR3	-0.17*	-0.02	-0.06	-0.02
BELIEF	-0.21*	-0.19*	-0.09	-0.22*
FEAR	-0.23*	-0.09	-0.01	-0.04
OVERALL	-0.30*	-0.19*	-0.07	-0.18*

P<0.05 * P<0.01

Table 6 presents an interesting pattern of results. Firstly, no correlation exceeds -0.30, suggesting that relationships are modest. Nevertheless, it seems that the strongest relationship is between the BARS and the beliefs and fears. Competition seems to possess the lowest association. The relationship between revenge and hostility indicates that the higher the score on the hostility scale, the more the individual seeks revenge and the correlation between BEL1 and competition suggests that the higher the score on the competition/ ambition scale, the more the individual believes that self worth is a function of one's accomplishments.

Total belief correlates significantly with the BARS, the SJAS and the hostility/time urgency subscale of the SJAS but not with the competition subscale. Overall fear correlates significantly with the BARS only and the total beliefs and fears correlates with the BARS, SJAS and HOST. The strongest predictor of Type A seems to be the overall beliefs and fears measure.

5.3.4 Sex Differences

Because of the large difference in the number of males and females, the following analyses should be treated with some caution. However, since Burke (1983) has reported some similar analysis, the results of the present research will be presented here. As a first step, a principal components analysis was performed on the data excluding females from the sample. This was done to see if the structure of the components would be altered without the female population. There were not enough females (n=61) to allow a separate analysis on them. The analysis on the 266 males revealed almost identical results. Two clear components emerged collectively accounting for 25.10% of the total variance with eigenvalues of 5.82 and 4.88 respectively and the same items loaded on each component as in the overall analysis.

Sex differences were examined by comparing males and females on the four Type A measures and on the measures of beliefs and fears. No significant differences were found between males and females on the Type A measures (Table 7).

Table 7: Means and standard deviations for males and females on the Type A measures

PTO

	Males (N=262)		Females (N=65)	
	Mean	Sd	Mean	Sd
BARS	60.10	8.32	61.12	8.75
JENKINS	7.96	3.22	7.28	3.73
COMP	4.31	2.35	3.78	2.36
HOS	1.51	1.12	1.51	1.07

As can be seen from table 7, there are no significant differences between males and females on any of the Type A measures. Striking sex differences, however, were observed when examining the measures of beliefs and fears (Table 8).

Table 8: Means and standard deviations for males and females on the measures of beliefs and fears

	Males (N=262)		Females (N=65)	
	Mean	Sd	Mean	Sd
BEL1	20.36	4.50	21.95	3.96
FEAR1	20.05	4.64	16.58	4.94
BEL2	14.48	3.35	14.57	2.77
FEAR2	11.69	2.97	11.02	3.15
REV	14.54	4.31	17.00	4.21
BEL3	25.31	5.86	28.83	5.58
FEAR3	20.16	5.73	16.29	6.45
BELIEF	74.69	12.76	82.35	12.36
FEAR	51.90	10.73	43.89	12.55
OVERALL	126.60	18.06	126.20	19.31

Although there was no significant difference between males and females on the overall score of the beliefs and fears, a

clear difference was observed between the two sexes on the overall beliefs ($F=19.03$ $df=1,326$ $P<0.001$). Males held stronger beliefs than females. However, females reported significantly more overall fear than males ($F=27.07$ $df=1,326$ $P<0.001$). This pattern was observed across the subscales of beliefs and fears. Males believed more than females that self worth is a function of one's achievements ($F=6.85$ $P=0.009$) and that things worth having are in scarce supply ($F=19.14$ $P<0.001$). Furthermore, males believed in seeking revenge more than females ($F=17.12$ $P<0.001$). Females on the other hand, expressed significantly more fear than males of negative evaluation ($F=28.34$ $P<0.001$) and of the individual not getting his share. There were no significant difference between males and females on BEL2 (no universal moral principles exist) and FEAR2 (fear that justice will not prevail).

Finally sex differences were examined by comparing the patterns of relationship between the various measures.

Table 9: Correlations between beliefs and fears and Type A measures for males and females

PTO

NB: Because of the large number of correlation coefficients, the possibility that Type II error may occur is inevitably increased. Therefore, caution should be taken when interpreting these results.

	BARS		JENKINS			COMP		HOS	
	M	F	M	F	M	F	M	F	
BEL1	0.25*	0.02	0.18*	0.13	0.13#	0.06	0.05	0.16	
FEAR1	0.25*	0.31*	0.14#	0.20#	0.11	0.09	0.01	0.25#	
BEL2	0.08	0.01	0.05	0.08	0.01	0.09	0.16*	0.08	
FEAR2	0.04	0.22#	0.08	0.21#	0.04	0.14	0.01	0.14	
REV	0.14#	0.02	0.08	0.08	0.01	0.07	0.22*	0.09	
BEL3	0.22*	0.26#	0.16*	0.28#	0.05	0.15	0.18*	0.39*	
FEAR3	0.13#	0.29*	0.03	0.06	0.04	0.07	0.02	0.18	
BEL	0.26*	0.13	0.18*	0.18	0.07	0.09	0.21*	0.28#	
FEAR	0.19*	0.32*	0.10	0.16	0.04	0.04	0.01	0.22#	
TOTAL	0.30*	0.30*	0.19*	0.22#	0.07	0.08	0.15*	0.32*	

Note: All correlations are negative in sign

P<0.05 * P<0.01

As can be seen, for male subjects, the overall beliefs and fears seems to be the best predictor of the BARS ($r = -0.30$) and for females the total fear score seems to be the best predictor ($r = -0.32$).

5.4. DISCUSSION

The present study had two objectives. The first was to develop measures of beliefs and fears thought to underlie the Type A behaviour pattern in adolescents and the second aim was to validate these measures against standardized Type A measures.

The first objective was reasonably well met. As predicted, the multi item measures of the seven constructs were generally found to have a high level of internal consistency

reliability. Further, beliefs correlated more strongly with other beliefs and fears with other fears, and the pattern was that the seven constructs formed a constellation congruent with Price's hypothesis. However, principal components analysis revealed two distinct components which were labelled "Fear" and "Belief" and most of the questions loaded on these components. It seems therefore that belief and fear measures form a distinct but separate pattern in the present study. It seems that the beliefs as measured in the present study are not necessarily accompanied by the fears.

The second objective of the present study was to correlate the belief and fear scores with Type A measures. This provided moderate support for Price's (1983) cognitive social learning theory. The relationship between the beliefs and fears with Type A measures suggested that the measures of the beliefs and fears best predicted scores on the BARS as compared with the SJAS and its competition/ambition subscale which did not seem to correlate as highly with measures of beliefs and fears. The hostility/time urgency subscale correlated moderately with the beliefs and fears.

Furthermore, some striking sex differences were found. The differences were important and congruent with previous research. Females reported significantly stronger fears than males, and males expressed stronger beliefs than females.

There were no significant differences between actual Type A scores between the sexes.

Nevertheless, it is well known that men tend to admit fear less than women (Price, 1983). This is presumably due to societal pressures and influences. Lakarczyk and Hill (1969) studying a large sample of primary school aged children, found that boys had higher scores on the Lie and Defensive scales of the MMPI with respect to fear. The results of the present study are congruent with the findings reported by Burke (1984a) on a sample of adult respondents. Unlike the results of the present study, however, Burke found females to be significantly more hostile than males.

There were several factors which may have reduced the statistical significance of the present results. As Price (1983) points out, the individual may not be necessarily aware of the beliefs and fears. Therefore, it is possible that questionnaires alone may not be sufficient in measuring these beliefs and fears and that other methods such as semi structured interviews may be a better method of assessing the proposed beliefs and fears. Hence, the present study should be viewed as exploratory. Moreover, as discussed previously, the measurement of the Type A behaviour pattern itself may be greatly improved if more objective assessment tools are used. This is clearly seen in the discrepancy between the two Type A measures used in the present study.

Firstly, the correlation between the two was lower than expected and, secondly, the beliefs and fears correlated strongly with one measure and not with the other. Future research should aim at measuring the proposed beliefs and fears by minimizing the use of questionnaire and using more objective methods.

Nevertheless, the results of the present study highlight two major points. Firstly, that the Type A behaviour pattern is associated with beliefs and fears in the younger population. This association suggests that the observable behavioural and physiological characteristics of Type A may indeed be maintained and encouraged by the presence of certain beliefs and fears. The results are congruent with similar studies carried out on adult populations suggesting that a pattern of beliefs and fears exists in Type A adolescents and that the relationship between these beliefs and fears and Type A measures is as strong as that observed in adults. Secondly, by showing an association between Type A and the beliefs and fears, the present study lends modest empirical support for the cognitive social learning theory proposed by Price (1983).

Finally, the results of the present study have implications for the modification of the behaviour pattern. The present research has shown that a pattern of specific beliefs and fears exists in adolescents and is associated with the Type

A behaviour pattern. Hence, attempts to modify Type A should take cognitive aspects into account. One major modification programme which was discussed in chapter one (Powell, et al 1984) has paid some attention to the cognitive social learning theory. A part of the programme was concerned with teaching Type As the cognitive underpinnings of overt Type A behaviour. One specific topic included characteristic beliefs about one self. However, the authors do not provide information as to whether or not any significant changes in these beliefs were observed in patients. It seems reasonable to argue that in the light of the present results, the modification of beliefs and fears may play a significant role in the modification of the Type A behaviour pattern.

Furthermore, the present research seems to suggest that the cognitive basis of Type A is as well established in adolescents as it is in adults. Therefore, the need to concentrate on the younger population is once again emphasized.

Price (1983) suggests that psychological intervention can provide a useful context for altering specific Type A beliefs and fears. This may be done by firstly concentrating on aspects of the behaviour pattern which are widely accepted as personally and socially negative and secondly, by repeatedly drawing connections between the Type A behaviour in problem situations and underlying beliefs and

fears so that ultimately these beliefs and fears can be replaced by alternative ones. It is reasonable to assume that these therapeutic programmes aimed at modifying beliefs and fears in Type A children and adolescents should be conducted with the support and active participation of significant adults and should be offered in a school setting or as part of a family based intervention (Price, 1983; Matthews and Haynes, 1986).

Nevertheless, there may be problems in trying to alter beliefs in fears in healthy Type As. In fact, generally, the results of modification programmes aimed at healthy Type A individuals have been far from conclusive. Firstly, as discussed earlier, the healthy Type A individual may not even be aware of these cognitive characteristics and secondly, the individual may be reluctant to change since some of these beliefs and fears may be not only socially acceptable but also may in fact result in success and therefore, be reinforced. Thirdly, there are ethical issues in attempting to actually alter the beliefs and fears in particular and the Type A behaviour pattern in general in healthy individuals. This is particularly applicable to the younger population.

Roskies (1986) points out that compared to other populations for whom modification programmes have been devised, the healthy Type As do not live in highly stressful life

circumstances; nor do they manifest obvious deficiencies in coping resources. On the contrary, these men and women can be considered as prototypes of the competent copers. Hence, seeking to intervene in the lives of individuals as well-functioning as these would seem to be potentially irresponsible, unless there was strong evidence of health risk serious enough to outweigh the psychological and social dangers inherent in upsetting an existing equilibrium.

As far as the younger population is concerned, these ethical issues play a more central role. For example, since Type A children and adolescents seem to be high achievers at school, there is a fear held by the authorities that any attempt to actually alter Type A would inevitably have an impact on achievement. Although at present there is no evidence to support this assumption, any modification programme must address this question very carefully.

The second experiment of the present research has offered an alternative approach to the modification of the Type A behaviour pattern in healthy Type As. It was suggested that the core elements of Type A could be exploited so that tension is reduced rather than increased. In this way, the aim is not to alter Type A behaviour per se but to reduce physiological reactivity in situations which encourage heightened arousal. The use of a similar technique with the

younger population seems appropriate in the light of the problems discussed above. The next experiment attempts to use heart rate biofeedback with Type A adolescents and take advantage of competition in order to motivate Type A subjects to relax.

CHAPTER SIX

EFFECTS OF COMPETITION AND HEART RATE BIOFEEDBACK ON TYPE A ADOLESCENTS.

6.1. INTRODUCTION

The results of the third study (Chapter five) showed that the beliefs and fears thought to constitute the core of the Type A behaviour pattern in adults, do exist in Type A adolescents. It was argued that since the mechanisms of the behaviour pattern seem to be very similar between adults and adolescents, the younger population seem to be an ideal target for the modification of Type A. Research has also pointed to the fact that the Type A behaviour pattern is significantly more stable from adolescence to adulthood than from childhood to adulthood (Steinberg, 1986). However, as was discussed in the previous chapter, there exist ethical and practical problems which seem to have generally dissuaded researchers from attempting to modify Type A in children and adolescents. In light of these problems, it is perhaps not surprising that to date no attempts have been made to test the possibility of modifying the Type A behaviour pattern in the younger population.

In view of the results of the second experiment (Chapter four), the aim of the present experiment is to explore the

possibility of modifying physiological responses in Type A adolescents using heart rate biofeedback. Results of experiment two showed that Type A adults were more successful than Type Bs in reducing their heart rate. Furthermore, results suggested that competition could be valuable in the modification of physiological responses in Type A adults. It was found that when put under competitive conditions, Type A adults were better able to relax (ie reduce heart rate) than Type B subjects. The present experiment aims to discover if a similar technique would produce similar results with Type A adolescents.

6.1.1 A note on the assessment of Type A adolescents

A major issue which warrants attention in the present study is the problem of assessing the Type A behaviour pattern in the younger population. This has already been discussed in some depth in previous chapters. In the present study, the Bortner scale will be used for selecting Type A and B subjects.

There is general agreement among researchers in the field (eg Contrada and Glass, 1988; Seraganian, etal 1987) that the strength of Type A associations with physiological reactivity depends on the method used to assess the behaviour pattern. The majority of studies showing clear physiological differences between Type A and B individuals

have used the structured interview as a method of assessment (Contrada and Krantz, 1988). Although some studies, such as the first two experiments described in the present research, have shown clear physiological differences between Type As and Bs using conventional Type A questionnaires, studies using the JAS have more consistently reported negative findings (Matthews and Haynes, 1986). This is despite the fact that the JAS has been found to predict the incidence of CHD quite accurately. This will be the main reason for not using the student version of the JAS for subject selection in the present study.

However, it can not be concluded that the Bortner scale itself is an ideal Type A assessment tool. Firstly, some studies have found no predictive validity for the Bortner in terms of CHD mortality (eg Koskenvou, etal 1983). Secondly, other self report Type A measures such as the Bortner have not received sufficient attention in psychophysiological research to permit comparisons between them (Contrada and Krantz, 1988). Despite this, the Bortner Scale has been used repeatedly for Type A assessment particularly with adolescents (eg Wolf, etal 1982). This is mainly because it is an easy questionnaire to administer and the questions are worded in a simple format (see chapters one and five). This is why the scale will be used in the present experiment. Nevertheless, the difference in results obtained between scores on the Bortner Scale and the SJAS reported in chapter

five highlights the assessment problems faced. In fact there was only moderate correlation found between the SJAS and the Bortner and several subjects classified as Type A or B on one questionnaire would not be classified as such on the other.

6.1.2 Biofeedback training with children and adolescents

There has been relatively little published research on the effectiveness of biofeedback training as a method of relaxation in children and adolescents. Most of these studies have used biofeedback in clinical settings. These include working with disabled children and adolescents (Murphy, etal 1977; Guralnick and Mott, 1976; Hunter, etal 1976), asthma (Kotses,etal 1976), drug abuse (Volpe, 1977) and migraine headache (Burke and Andriasik, 1989).

The first attempts to use biofeedback with children produced mixed results. A series of early studies (Fruhling, etal 1969; Simard, 1969) showed that sensorimotor control could be taught to children even under the age of six years old. Later studies found no such effect (Englehardt, 1976; Blovin, 1977).

A series of studies have evaluated the effectiveness of biofeedback in treating asthmatic children (eg Kotses, etal 1976). These studies have generally evaluated the effects of

frontalis muscle relaxation on peak respiratory flow rates in asthmatic children. The results show that significantly improved peak respiratory flow rates are obtained using EMG biofeedback training (Lynch, 1976).

The most promising attempt at using biofeedback in a clinical setting with children and adolescents has been the use of EMG and thermal feedback in treating migraine headache (eg Diamond and Franklin, 1976). More recently, Duckro, Cartwell and Simmons (1989) reviewed recent literature on treatment of headache in children and adolescents and concluded that frequency of headache was most consistently reduced by behavioural therapies, in particular biofeedback training.

The use of biofeedback with children and adolescents has also been successful in the area of stress management. Volpe (1977) reported feedback facilitated relaxation in early adolescent drug addicts and concluded that biofeedback training of relaxation could be used as primary prevention of drug abuse in adolescence. Angus (1989) in a review of the literature argues that biofeedback training (particularly thermal feedback) is an ideal stress management technique for children.

Therefore, it seems that although early studies on biofeedback training with children produced conflicting

results, the use of biofeedback technology in clinical settings has generally been successful. Nevertheless, it must be pointed out that only one of the studies has provided follow up data (Burke and Andriasik, 1989). This lack of follow up information leaves the question of the long term effects of biofeedback unanswered. Nevertheless, in relation to the present experiment, biofeedback technology seems an ideal method to employ with Type A adolescents, since it has been a popular and successful method in the area of stress management in both adult (Johnson, 1984) and adolescent populations (Angus, 1989).

In the present experiment, Type A and Type B adolescents will take part in a 10 minute relaxation session where half the subjects will be given contingent auditory heart rate biofeedback while the other half will be provided with no feedback at all. Half the subjects will be given competitive instructions as described in experiment two and the other half will be asked to reduce heart rate without any competitive instruction. The hypotheses of the present experiment are as follows.

6.1.3 Hypotheses of the present study

1. Overall, Type A adolescents will reduce heart rate more than Type B subjects

2. Type A adolescents will be more successful in heart rate reduction under competition than the no competition condition.

There is some evidence suggesting that biofeedback training is an effective technique for relaxation in adolescents (Duckro, etal 1989;Lynch, 1976). In the area of stress management, biofeedback has been particularly successful (Angus, 1989).

3. Type As will reduce heart rate more successfully when provided with heart rate biofeedback than when no feedback is provided.

The next hypotheses concerns the role of heart rate biofeedback as a method of relaxation. It is predicted that

4. Subjects receiving heart rate biofeedback will reduce heart rate more than subjects who receive no biofeedback at all.

Finally the role of subjective reports of relaxation will be investigated in the present experiment. Research on subjective effects of biofeedback have been mixed. Biofeedback has been reported by some researchers to reduce levels of anxiety and stress. For example, Grazzi, etal (1988) found that after EMG biofeedback, subjects' scores on the MMPI and state-trait anxiety inventory were

significantly reduced as compared to scores obtained before training. On the other hand, several studies have found that although subjects are more successful in controlling physiological responses following biofeedback training, they do not necessarily report more relaxation (Shahidi and Powell, 1988; Prigatano and Johnson, 1972). More specifically, a recent study by Kratsky (1988) found that following GSR biofeedback training, there was no difference in the ratings of relaxation between Type A and B females. The results of the second experiment of the present research seems to support the latter study. However, it must be pointed out that neither in experiment two in the present work nor in most of the published studies investigating the subjective effects of biofeedback, has a reliable index of relaxation been used. The problems faced in interpreting the results of experiment two concerning the role of subjective reports have already been discussed. The present experiment aims to investigate subjective reports of relaxation in Type A and B adolescents by using a more reliable index, namely the stress and arousal check list developed by Mackay et al (1978).

6.2. METHOD

6.2.1 Subjects

Subjects were selected from an original sample of 327 adolescents who filled in Type A questionnaires (see Chapter five). 80 respondents who were extreme scorers on the Bortner Adjective Rating Scale (BARS) were selected to take part in the present experiment. Those subjects who scored one standard deviation above the mean were labelled Type A and those who scored one standard deviation below the mean were labelled Type B (Mean= 60.30, sd= 8.56). Out of the 80 subjects selected, three were absent when the experiment was conducted and the parents of five subjects did not give permission for their children to take part. Therefore, 72 adolescent subjects (56 males and 16 females) took part in the experiment. The mean age was 14.94 years and the age range was 12-18. The ratio of males to females was kept constant across the experimental groups (there were 7 males and 2 females in each group) and the mean age for subjects was roughly the same for each group (see table 1).

Table 1: Mean age, Bortner scores and sd's for each group

	Mean (sd)	
	Age	Bortner score
Type A Comp	15.16 (1.13)	73.84 (3.85)
Type A No Comp	15.28 (0.69)	73.56 (4.58)
Type B Comp	14.73 (1.40)	49.78 (4.21)
Type B No comp	14.75 (1.48)	48.23 (4.75)

6.2.2 Design

A 2 (Type A vs Type B) x2 (Competition vs No Competition) x2 (Biofeedback vs Control) x10 (ten minutes of the relaxation session) design was used. The first three factors were between subjects and the last factor (minutes) was within subjects factor. Each subject took part in one ten minute relaxation session. Half of subjects (18 Type As and 18 Type Bs) were selected randomly and were given competitive instruction and half were given no competitive instruction at all. Similarly, half of subjects were given contingent auditory heart rate biofeedback as described below and half were given no feedback at all (control).

6.2.3 Materials

6.2.3.1 The stress and arousal check list

Subjects were asked to complete the stress and arousal check list (Mackay, Cox, Burrows and Lazzerini, 1978) before and after the ten minute session as a measure of subjective relaxation. The check list was developed as an inventory for the measurement of self reported stress and arousal and was adopted for British use from one originally constructed by Thayer (1967). The scale was administered to 145 subjects and two bipolar factors were extracted and labelled stress and arousal. Further factor analytic studies (Cox, Mackay and Page, 1982) modified the original version so that the loading on none of the adjectives is less than 0.60. The check list consists of thirty adjectives which are common descriptors of two aspects of mood. Stress is defined as feeling tense, uncomfortable, unpleasant and bothered. Arousal is defined as being alert, awake, attentive, and lively. The respondents are asked to indicate how they feel "at this moment" using a four point response scale ranging from "definitely feel" to "definitely do not feel". Eighteen of the adjectives load on the stress factor, and twelve on the arousal factor. The scoring of the scale is dichotomized and the adjectives are bipolar, that is the adjectives describing stress and those describing arousal are separate and different from each other (see appendix 11).

6.2.3.2 Heart rate measurement

Heart rate signals were measured by a Tunturi Pulse meter as described in chapter four and recorded via a tape recorder (Tensai, model CRE-102) on magnetic tape (Ampex, C30). This was done with the aid of modulator and demodulator systems which together with circuit diagrams are presented in appendix 12. Continuous auditory heart rate feedback was provided for ten minutes to half of subjects. Heart beat signals were presented to the subjects through headphones in the form of clicking sounds 300 Hz in frequency.

A computer programme in BBC basic was prepared to decode heart rate from tape (see appendix 13). The procedure was as follows. The measuring interval was set at one minute. The programme was designed to search for a stable pulse by ignoring the first three pulses before timing. A rising edge of a pulse was waited for and then its falling edge. This was repeated three times. Then the programme simply waited for the rising edge of the next pulse, set the timer to zero, and waited for the falling edge. The programme looped waiting for each rising edge of a pulse before incrementing a counter and displaying it along with the inter beat interval, then waiting for a falling edge. Elapsed time was checked for each loop and if a minute had expired the heart

rate was calculated as beats per minute (BPM) and recorded. After each session the programme switched off the cassette motor, then listed the results for each minute of each session on the computer monitor.

6.2.4 Procedure

The experiment was conducted in three independent secondary schools who had agreed to take part in the study. Subjects' consent was obtained after they were informed about the experiment by their teacher and were asked to take a consent form home to be filled in by their parents. The experimenter was allowed a period of approximately thirty minutes with each subject. This period was specified by the school authorities. Each subject was taken to a quiet room in the school and was given a brief introduction to the biofeedback apparatus and the measurement of heart rate. At this stage a one minute base line heart rate was taken and each subject filled in the stress and arousal check list. Subjects in the feedback condition were given an extra thirty second trial feedback in order to become familiarized with auditory heart beat signals. Subjects in the competition condition were told that they were competing with other subjects and the person who could reduce heart rate more than others would win a prize of £2. Subjects in the no competition condition were given no competitive instruction at all and were told

simply to relax and try to reduce heart rate as much as possible. All subjects were told to keep their eyes closed and those who were given biofeedback were told to concentrate on the sound of their heart beats. The duration of the relaxation session was ten minutes and subjects filled in the stress and arousal check list for a second time at the end of the session. Subjects were not paid for taking part in the experiment.

6.3. RESULTS

2 (Type A)X2 (Competition)X2 (Condition) Anova's were carried out on average baseline heart rate and average heart rate during the first minute of the ten minute relaxation session and no significant differences were found. Similarly, no significant differences were found for initial scores on the stress and arousal questionnaire.

A 2 (Type)X2 (Competition)X2 (Condition)X10 (10 minute session, repeated measure) ANOVA with fitted regression slopes was carried out on the raw data using the GENSTAT programme. Table 2 shows the summary table.

Table 2: ANOVA summary table for heart rate data

Source	df	ss	ms	F	P
Type	1	5.89	5.89	<1	ns
Competition	1	372.95	372.95	<1	ns
Condition	1	56.81	56.81	<1	ns
TypexCompetition	1	629.01	629.01	<1	ns
TypexCondition	1	127.32	127.32	<1	ns
CompXCondition	1	1997.83	1997.83	1.76	ns
TypexCompXcond	1	0.47	0.47	<1	ns
Error	64	72504.83	1132.89	-	-
Minutes	9	75.57	8.40	1.71	ns
Linear of Mins	1	17.24	17.24	3.52	0.06
MinutesxType	9	73.52	8.17	1.67	ns
Lin MinsxType	1	4.49	4.49	<1	ns
MinutesxComp	9	84.95	9.44	1.93	0.046
Lin MinsxComp	1	60.46	60.46	12.35	<0.01
MinutesxCondition	9	97.08	10.78	2.20	0.02
Lin MinsxCond	1	66.20	66.20	13.52	<0.01
Lin MinxTypexComp	1	8.73	8.73	1.78	ns
MinsxTypexCond	9	77.46	8.61	1.76	0.07
Lin MinxTypexCond	1	32.58	32.58	6.65	0.01
MinsxCompXCond	9	26.04	2.89	<1	ns
Lin MinxCompXCond	1	0.12	0.12	<1	ns
Error	585	2864.50	4.90	-	-
Total	719	79115.76	-	-	-

The linear effect for the overall Minutes factor just fails to reach the 0.05 significance level suggesting that subjects' gradual reduction in heart rate across the ten minute relaxation session is not significant.

As can be seen from table 2, there is no overall difference in heart rate reduction between Type A and B subjects. Furthermore, competition does not seem to significantly affect heart rate reduction in Type A subjects. However, there is a significant MinutesxCompetition interaction, of which the linear component is significant (figure 1). This

indicates that overall, subjects' heart rate decreased linearly more during competition than during no competition. The Minutes \times Condition interaction and its linear component is also significant (figure 2). As can be seen, subjects in the biofeedback condition have reduced heart rate more rapidly than subjects in the control condition.

There is a significant linear trend for the Minutes \times Type \times Condition interaction. Figure 3 shows this interaction. Contrasts of mean linear coefficients were calculated using GENSTAT and post hoc t values were calculated using the overall Error degrees of freedom. Type As receiving biofeedback reduced heart rate across the ten minutes significantly more than Type Bs receiving feedback ($t= 3.95$) and Type Bs in the control condition ($t= 3.15$). Furthermore, Type As in the feedback group reduced heart rate significantly more than Type A subjects in the control group ($t= 4.43$).

Finally the scores of subjects on the stress and arousal check list were analyzed. The scores before and after the ten minute relaxation session were analyzed using the BMDP programme. Two split plot ANOVA's (Type \times Competition \times Condition \times Time, before and after) were conducted separately for stress scores and for arousal scores.

during competition and no competition

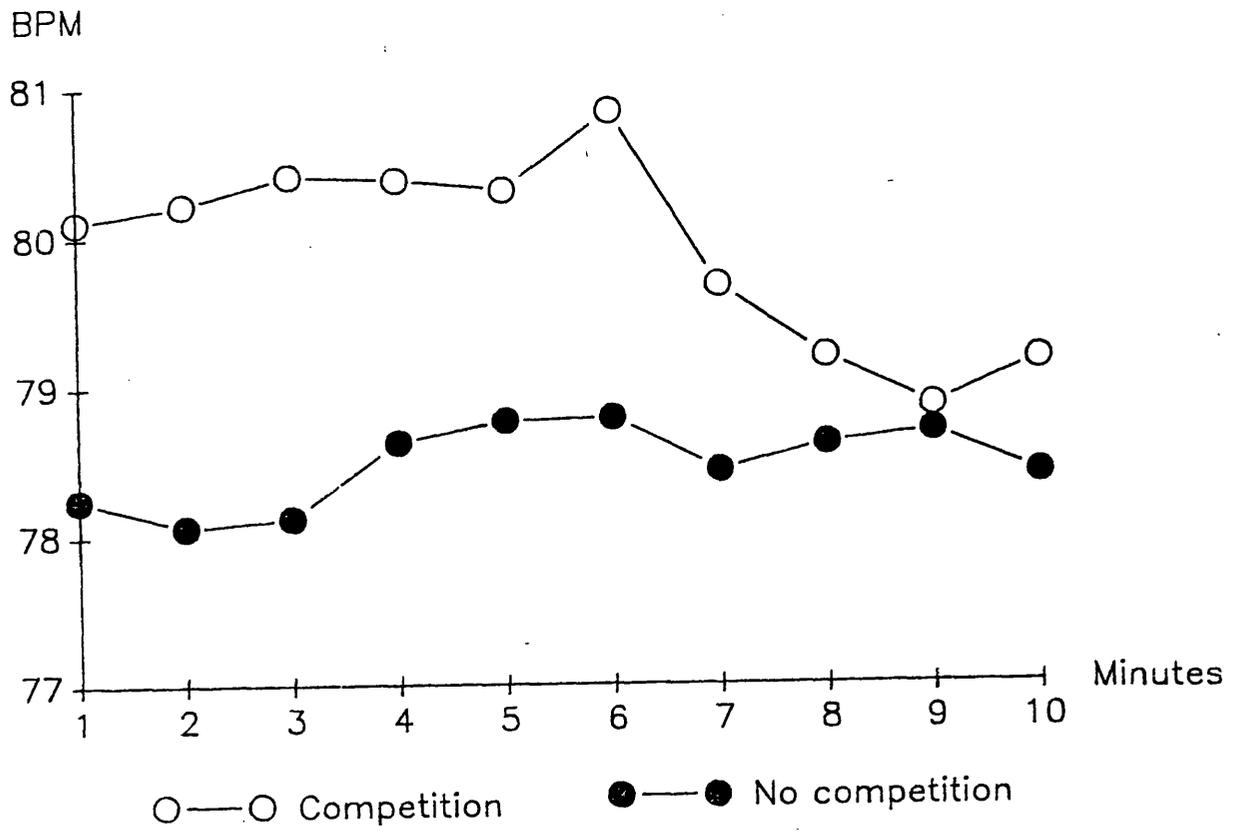


figure 2: Heart rate (BPM) for subjects in the feedback & control conditions

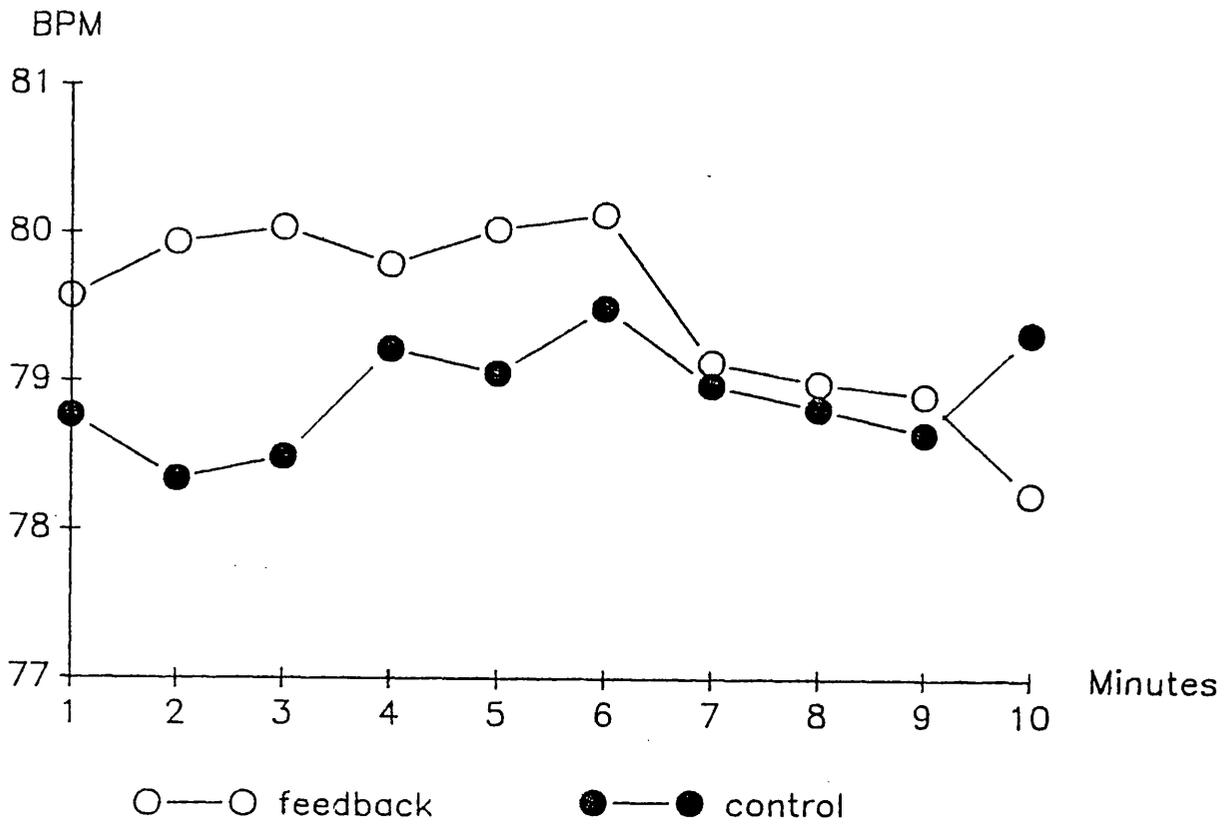


figure 3: Heart rate (BPM) for Type A & B subjects in the feedback & Control conditions

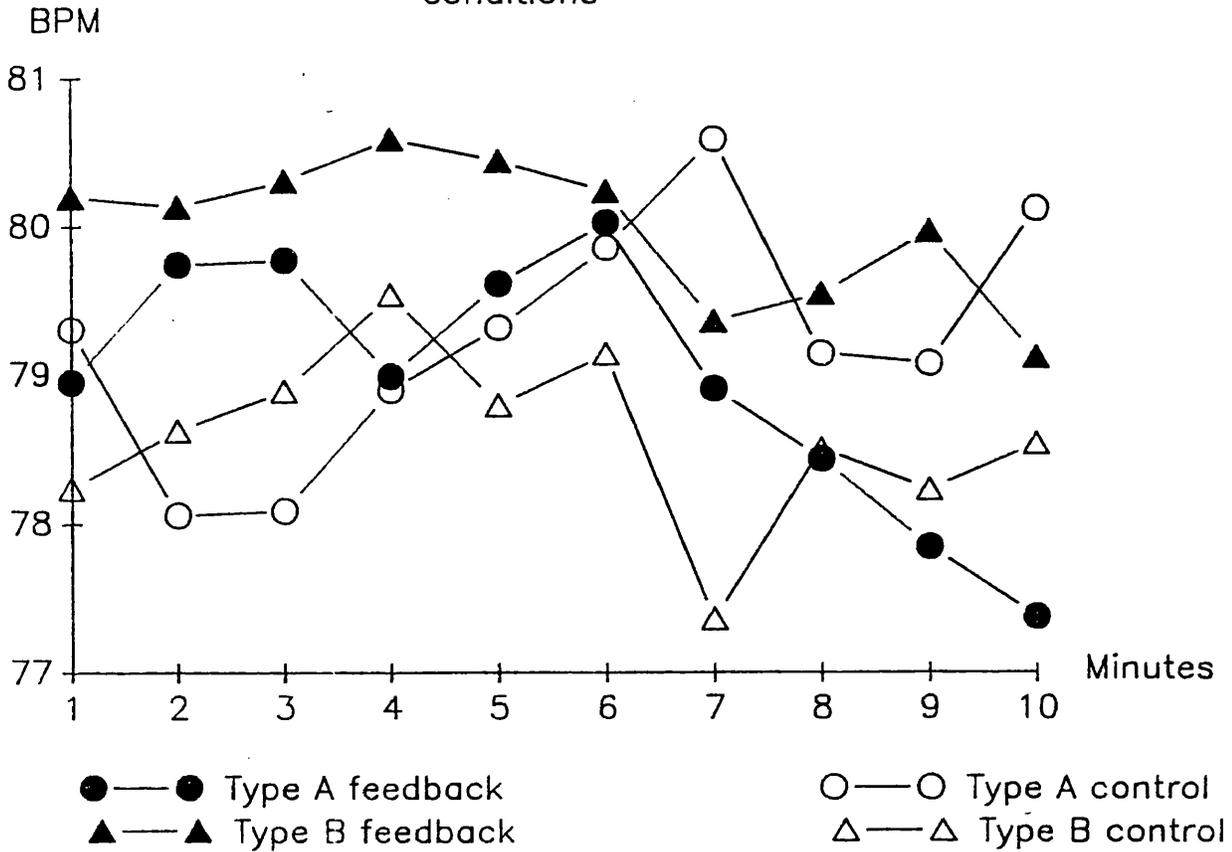


Table 3: Means and sd's for stress and arousal before and after the relaxation session.

Arousal (maximum score=12)	
Before	After
Mean	Mean
6.40 (2.85)	3.57 (3.21)
Stress (maximum score=18)	
Before	After
Mean	Mean
6.83 (4.00)	2.60 (3.56)

Overall, subjects reported significant reductions in both stress and arousal after the ten minute relaxation session as compared to the beginning (for arousal $F=62.67$ $df=1, 64$ $P<0.01$ and for stress $F=70.77$ $df=1, 64$ $P<0.01$).

There were no significant differences in stress scores between groups (table 4). However, as far as arousal scores were concerned, Type A subjects reported significantly higher scores after the relaxation session than did Type Bs ($F=4.54$, $df=1,64$ $P<0.05$). Although the interaction between Type and competition does not reach significance, as can be seen from table 5, Type As in the competition condition reported more arousal after the relaxation session than the other groups.

Table 4: Means and sd's for stress after the relaxation session.

	Mean
Type A	2.72 (2.85)
Type B	2.47 (3.66)
Type A Competition	2.56 (2.60)
Type B Competition	2.50 (3.15)
Type A No competition	2.89 (3.11)
Type B No competition	2.45 (4.17)
Type A Feedback	3.39 (3.56)
Type A Control	2.06 (2.15)
Type B Feedback	2.34 (2.43)
Type B Control	2.61 (3.01)

Table 5: Means and sd's for arousal after the relaxation session.

	Mean
Type A	4.33 (3.03)
Type B	2.81 (2.84)
Type A Competition	3.89 (2.57)
Type B Competition	3.61 (3.52)
Type A No competition	4.78 (3.50)
Type B No competition	2.00 (2.16)
Type A Feedback	3.89 (3.07)
Type A control	3.78 (3.00)
Type B Feedback	2.67 (2.20)
Type B Control	2.95 (3.42)

6.4. DISCUSSION

As compared to the second study, the results of the present experiment present a similar picture. Biofeedback was found to be an effective method for heart rate reduction. Subjects who were given biofeedback showed a significantly greater reduction in heart rate than those who were given no feedback at all. More importantly, Type A subjects were found to be responsive to biofeedback and reduced heart rate more when given heart rate feedback than during the control condition when no feedback was given. Furthermore, Type As receiving biofeedback reduced heart rate more than Type Bs receiving biofeedback.

The other hypothesis of the experiment concerning the role played by competition in Type A subjects was not supported. There was no significant difference in heart rate reduction between Type A and B adolescents during competition and no competition. Furthermore, there was no overall difference in heart rate reduction between Type A and B subjects. Nevertheless, there was an overall linear effect for competition suggesting that overall subjects reduced heart rate significantly more when competing than during no competition. As far as subjective reports of relaxation were concerned, an expected result was that overall, subjects reported significant reductions in both stress and arousal after the ten minute relaxation session as compared to the beginning. However, there were no differences between groups in self reported stress and arousal except that Type As reported to be more aroused after the relaxation session than Type Bs.

One problem, however, with interpreting the above results is the fact that there were different starting heart rate for different groups. Although initial analyses showed no significant difference in initial heart rate, it may be that for example, subjects receiving biofeedback have shown a more linear reduction in heart rate merely because of the "ceiling effect". These results therefore must be treated with some caution.

Nevertheless, the above results are very encouraging indeed. Firstly, it was shown that Type A adolescents in the feedback group showed a greater reduction in heart rate than the other groups and therefore they were responsive to biofeedback. Secondly, although there was no specific competition effect for Type As, overall subjects reduced heart rate more during competition than during no competition. Subjective reports of relaxation showed that Type As reported more arousal after the relaxation session than Type Bs. Therefore, Type A subjects report to be more alert, awake and lively after the relaxation session and after competition than Type Bs. Low stress scores indicate feelings of reduced tension and increased comfort and relaxation (Mackay et al 1978). The present results suggest that modification programmes such as in the present experiment can usefully be aimed at Type A adolescents.

It is important to point out that the present experiment faced some problems which will be discussed.

It is important to point out that auditory biofeedback was used in the present experiment instead of the much preferred method of visual feedback. Many researchers using biofeedback as a method for relaxation have recommended the use of visual feedback and several studies have shown that the presentation of physiological events visually is far

superior in terms of effectiveness to auditory biofeedback (Blanchard, et al 1974). Because of the various limitations faced, visual feedback could not be provided in the present study. Firstly, it was necessary to avoid the use of large complex equipment such as a computer monitor which would be necessary in providing visual feedback. This was mainly for practical reasons. The advantage of presenting heart rate in graphic form on a computer monitor to adolescents over heart rate presented as sound through head phones is suggested by previous experiments (eg Simard, 1969) where such presentation techniques have been more successful. Despite this, the present results suggest a clear difference in heart rate reduction between subjects receiving auditory heart rate feedback and the control group.

The other problem faced by the present experiment was the short period of time each pupil was allowed to participate in the experiment. Ideally, several relaxation sessions with and without biofeedback should have been included as a part of a more comprehensive programme. However, as is the case with most field studies of this kind, the design of the experiment had to be modified to suit school time tables. Subjects would certainly have been more comfortable and a better rapport would have been established had there been more time per session available. Nevertheless, the results of the present experiment supports two major issues. Firstly, the results support experimental studies in the field

(including the experiment conducted on adults in the present research) suggesting that physiological control can be achieved in one biofeedback training session (eg Shahidi and Powell, 1988; Stern and Elder, 1982). Secondly, the effective application of biofeedback techniques can be established by experiments such as the present study, where the usefulness of biofeedback is tested in practical conditions where both time and resources are scarce.

In view of the problems discussed above, the results of the present experiment are impressive. It is important to emphasize, however, that biofeedback studies on adolescents have been scarce and by no means conclusive. Furthermore, to date Type A adolescents have not been the target of any modification programmes and as discussed in the introduction, the problem of subject selection using conventional Type A questionnaires applies to adolescents as well as adults. Further experimentation using a more rigorous methodology is needed. In the context of the present experiment, future studies should establish whether or not Type A adolescents react to competition and other core elements of Type A, and if so programmes should be

devised to exploit these characteristics. Secondly, the long term effects of the manipulations used in the present experiment must be evaluated. For example, it must be established whether the effects of biofeedback can be sustained in subjects, in particular Type A subjects, after the termination of training.

CHAPTER SEVEN

SUMMARY AND CONCLUSIONS

The major contribution of the present research is to offer a new technique for the modification of physiological reactivity in healthy Type A individuals. The present research also emphasized the need for researchers to concentrate on the younger population by aiming modification programmes at children and adolescents.

Four studies were described. The results of the first experiment supported the literature on Type A showing distinct behavioural and physiological differences between Type A and B adults. In particular, Type As showed significantly greater increases in heart rate than Type Bs when involved in a driving game. Physiological reactivity was particularly exaggerated in Type As when subjects were involved in competition. The results suggest that the assessment of Type A can have implications for predicting driver performance. Furthermore, given the above results, it was pointed out that it is surprising that to date no modification programme has attempted to exploit the core characteristics of the Type A behaviour pattern such as competitiveness (Stern and Elder, 1982).

The second experiment outlined a technique, using contingent and non contingent (false) heart rate biofeedback, where Type A and B subjects were put in a competitive situation and were asked to relax. The efficacy of biofeedback training as a relaxation technique was supported by the data. Nevertheless, non contingent feedback suggesting a decrease in heart rate was found to be as effective as true, contingent biofeedback. This latter result supports the view that there is a strong suggestibility component in the biofeedback paradigm (Stroebel and Glueck, 1973) and thus the operant conditioning model of biofeedback (eg Lang, etal 1967) is challenged. The results seem to suggest that many of the effects produced by biofeedback can be explained by a recourse to a placebo effect. It may be argued that awareness of one's physiological state via biofeedback, whether contingent or not, may result in at least some internal control (Shahidi and Powell, 1988).

The second study also found that type As reduced heart rate more than Type Bs. Furthermore, in a situation where Type As would generally be expected to show heightened physiological reactivity (ie competition), they showed lowered heart rate by comparison with Type B subjects. Challenging incentives seemed to play an important role. Type As reduced heart rate more than Type Bs when the goal of the competition was relaxation rather than excitement.

The present research also emphasized the importance of the Type A behaviour pattern in adolescents. The third study showed that the core elements of the behaviour pattern, that is, the beliefs and fears which have been identified in Type A adults (Burke and Deszca, 1984; Burke, 1984) do cluster together in adolescents. These beliefs and fears also form the back bone of the cognitive social learning theory proposed by Price (1983) and Smith and Anderson (1986). The present research has therefore offered data in support of the theory. It was therefore suggested that the behaviour pattern seems to be learned at least as early as the onset of adolescence (Matthews and Woodall, 1988) and that scores of Type A measures in adolescents were associated with the beliefs and fears which are thought to maintain and encourage the behaviour pattern. In view of the above results, and taking into consideration the fact that CHD develops through out a person's life and that its development starts at a very early age (Matthews and Woodall, 1988), it follows that the modification of Type A should also begin at an early age. Future research should concentrate on developing the efficacy of measuring these beliefs and fears, particularly in adolescent populations. This could be achieved by using interviews as well as questionnaires. Furthermore, the actual mechanisms by which these beliefs and fears are learned are still unknown (Price, 1983). Research is needed to evaluate the role played by various socializing agents such as the family, the

educational system and the mass media in transmitting these beliefs and fears to the individual.

The final study of the present research attempted to reduce heart rate using competition and contingent heart rate biofeedback in Type A adolescents. The results were quite straight forward and similar to the second experiment on adults. The effectiveness of biofeedback as a method of relaxation was confirmed. Subjects receiving auditory heart rate biofeedback reduced heart rate significantly more than subjects receiving no feedback at all. Moreover, Type A adolescents were found to be responsive to biofeedback relaxation training. Contrary to the experiment on adults, the results showed no overall significant difference in heart rate reduction between Type A and B subjects, and Type As did not seem to be more responsive to competition than Type B adolescent. Nevertheless, there was an overall competition effect suggesting that overall subjects, including Type As, reduced heart rate significantly more during competition than when not competing. Furthermore, analysis of subjective reports of relaxation suggested that Type As reported significantly more arousal than Type Bs at the end of the relaxation session.

However, it must be pointed out that to date no attempts have been made to reduce any aspect of the Type A behaviour pattern in children or in adolescents. Therefore, firm

conclusions cannot be made about the above results. The few studies using biofeedback with children and adolescents certainly provide mixed results (see chapter six). Although the use of biofeedback in clinical settings has generally been successful (Angus, 1989), future research should confirm whether or not heart rate biofeedback, in the form used in the present experiment, is effective with healthy Type A adolescents.

There is no doubt that any attempt at modifying the Type A behaviour pattern in adolescents will face a great number of problems. These difficulties have already been discussed in some depth in previous chapters. The most complex dilemma facing researchers is the ethical issues involved in changing behaviour in an apparently healthy population. Most researchers (eg Price, 1983; Matthews and Woodall, 1988) agree that a significant factor which constantly dissuades researchers from concentrating modification attempts on the younger population is the resistance of significant adults to cooperate in such programmes.

Resistance to change is also a problem faced by many researchers. It is often reported that many healthy Type A individuals are either unaware of being Type A or do not wish to change their behaviour. This problem is discussed in great depth by Roskies (1987) and has already been outlined in chapter four. It is mainly because of this that many

attempts at modifying the behaviour pattern in healthy individuals have failed or have been only partially successful. The use of cognitive-behavioural techniques such as Rational Emotive therapy (Woods, 1987), anger management (Thurman, 1985) and stress management training (Roskies, 1987) with healthy Type As has generally been successful in reducing Type A behaviour as measured by Type A questionnaires. However, most of these studies suffer from a variety of methodological weaknesses ranging from lack of control groups to failure to measure Type A directly by using questionnaires not designed to measure Type A (Bennett and Carroll, 1990). Moreover, most of these studies have repeatedly failed to alter physiological reactivity in healthy Type As. Seraganian, et al (1987) have pointed out that the constant failure to modify psychophysiological reactivity using stress management training (Roskies, et al 1986) or other methods such as aerobic exercise (Seraganian et al, 1987) may reflect that, in general, behavioural programmes are unsuitable vehicles for producing psychophysiological change in healthy, reactive Type A individuals.

The most promising line of research on changing physiological reactivity comes from programmes using behavioural techniques such as meditation (Muskatel, et al 1984) and biofeedback training (Stern and Elder, 1982; Prior, et al 1983). Here, it has been shown that at least in

the short term, Type As are responsive to relaxation training. Moreover, the results of these studies suggest that heart rate reactivity and muscular tension can be significantly reduced in Type A subjects. Unfortunately, only a very few studies of this kind have been reported.

It seems reasonable to argue that, in view of the points discussed above, research should concentrate on exploring new techniques which would attempt to change reactivity in Type As rather than changing the behaviour pattern per se.

It seems that as far as modifying physiological reactivity is concerned, biofeedback training as used in the present research may be the most promising technique to be employed as compared to the more popular methods such as stress management training. Nevertheless, it must be pointed out that, as is the case in the present research, all biofeedback programmes using Type As as subjects have provided no follow up data. As far as the present study is concerned, as a first step, replication studies are needed, in order to confirm the results obtained.

Finally, a great deal of recent research has suggested that there may be just one or two components of the Type A behaviour pattern that account for the coronary risk associated with Type A (Price, 1988). Many studies have pointed out to hostility as a leading contender (Price,

1988; Shekelle, et al 1983). In a recent review of this body of research, Matthews and Haynes (1986) concluded that hostility seems to be the most toxic element of the Type A behaviour pattern and that future studies should include specific measures of hostility to determine whether this is the case. The procedures described in the present research should be employed in future studies taking advantage of other core elements of the behaviour pattern in particular competition and hostility as challenging incentives. An outline of recommendations for further research is summarized below:

1. It is suggested that Type A research should concentrate on children and adolescents and possible means of modifying the behaviour pattern should be examined including the modification of Type A beliefs. But any attempt to change these beliefs in an apparently healthy population will be faced with a variety of ethical and practical problems. Any modification programme, particularly those aimed at children and adolescents, should take these problems into consideration.

2. The beliefs and fears as measured in the present research need to be studied in more detail, particularly in the younger population. Specifically, the actual mechanisms by which these beliefs and fears are transmitted to the individual through socialization should be examined.

3. Modification of these beliefs and fears may play a significant role in the reduction of the Type A behaviour pattern in patients suffering from heart disease and ultimately reducing the risk for CHD. Research should concentrate on a) evaluating the feasibility of modifying the Type A beliefs in patients with CHD and b) discovering whether or not modification of the beliefs would actually lead to a significant reduction in the incidence of heart disease.

4. In view of the ethical problems faced in changing behaviour pattern in healthy Type As, it is recommended that as a first step, research should evaluate the feasibility of reducing physiological reactivity in healthy Type A adults and adolescents rather than trying to alter the behaviour pattern per se. One way of doing so was outlined in the present research. Future research should examine the role of other challenging incentives such as hostility and time urgency in reducing physiological reactivity.

5. Heart rate biofeedback was shown to be an effective method of relaxation for Type As. Research is needed to confirm the efficacy of other forms of biofeedback and other techniques such as meditation and hypnosis in the reduction of physiological reactivity in Type As, particularly Type A children and adolescents.

6. The clinical utility of the methods outlined must be evaluated. Replication studies are needed to confirm the results obtained. More importantly, follow up studies should evaluate the long term efficacy of the methodology described in the present research.

7. As discussed in chapter one, research suggests that the link between Type A and CHD may be due to independent dimensions such as hostility and competition rather than a global Type A behaviour pattern (Shekelle, etal 1983). The multidimensionality of Type A is seen clearly in the relationship between Type A measures. For example, low correlations are often reported between Type A questionnaires (as in the present research). This may be due to the fact that these measures simply assess different dimensions of the behaviour pattern. The need for developing new and objective methods of Type A assessment has been repeatedly emphasized in the present research. Future studies should address the issue of multidimensionality of Type A closely.

BIBLIOGRAPHY

- Achmon, J Graneck, M Golomb, M and Hart, J (1989)
Behavioural treatment of essential hypertension: a
comparison between cognitive therapy and biofeedback of
heart rate. *Psychosomatic Medicine*, 51(4), 152-164.
- Albright, GL Andreassi, JL and Steines, SS (1988)
Interactive effects of Type A personality and
psychological and physical stressors on human cardio-
-vascular functions. *International Journal of Psycho-
-physiology*, 6(4), 315-326.
- Anchor, F Anchor, K and Sandler, H (1979) Biofeedback train-
-ing, psychophysiological disorders and Type A behaviour
with working and nonworking women. *American Journal of
Clinical Biofeedback*, 2(2), 55-59.
- Angus, SF (1989) Three approaches to stress management for
children. *Elementary School Guidance and Councelling*,
23(3), 228-233.
- Appels, A Jenkins, CD and Rosenman, RH (1982) Coronary heart
disease in the Netherlands: a cross cultural validation
study. *Journal of Behavioural Medicine*, 5(1), 83-90.

- Bachman, EE Sines, JO Watson, JA Lauer, RM and Clarke, WR
(1986) The relation between Type A behaviour, clinical
relevent behaviour, academic achievement and IQ in
children. *Journal of Personality Assessment*, 50, 186-
192.
- Bandura, A (1977) *Social Learning Theory*. Englewood Cliffs,
NJ Prentice-Hall.
- Barefoot, JC and Straub, RB (1971) Opportunity for
information search and the effect of false heart rate
feedback. *Journal of Personality and Social
Psychology*, 17, 154-157.
- Basmajian, JV and Hatch, JP (1979) Biofeedback and
modification of skeletal muscular dysfunction. In
Gatchel, RJ and Price, KP (eds) *Clinical Application
of Biofeedback: Appraisal and Status*, NY Pergamon
Press.
- Beck, RC Gibson, C Elliott, W Simms, C Matteson, N and
McDaniel, L (1988) False physiological feedback and
emotion: experimenter demand and salient effects.
Motivation and Emotion, 12(3), 217-235.

Belgian-French Pooling Project (1984) Assessment of Type A behaviour by the Bortner scale and ischaemic heart disease. European Heart Journal, 5, 440-446.

Bem, DJ (1967) Self perception: an alternative interpretation of cognitive dissonance phenomena. Psychological Review, 74, 183-200.

Bennett, P and Carroll, D (1990) Stress management approaches to the prevention of coronary heart disease. British Journal of Clinical Psychology, 29(1), 1-12.

Berenson, GS (1980) Cardiovascular Risk Factors in Children. NY, Oxford.

Bergman, LR and Magnusson, D (1986) Type A behaviour: a -longitudinal study from childhood to adulthood. Psychosomatic Medicine, 48, 134-142.

Birk, L (1973) Biofeedback: specific learning component vs non specific learning component (placebo effect). In Birk, L (ed) Biofeedback: Behavioural Medicine, Grune and Stratton.

Blanchard, EB and Epstein, LH (1978) A Biofeedback Primer. Addison Wesley Publishing Company.

- Blanchard, EB Scott, RW Young, LD etal (1974) Effect of knowledge of response onthe self control of heart rate. *Psychophysiology*, 11, 251-264.
- Blanchard, EB Miller, ST Abel, GG Haynes, MR and Wicker, R (1979) Evaluationof biofeedback in the treatment of border line essential hypertension. *Journal of Applied Behaviour Analysis*, 12, 99-109.
- Block, J (1971) *Lives Through Time*. Berkeley, CA Bancroft.
- Blovin, S (1977) Anattempt to place children under the operant conditioning of a harlequin puppet. *Psycho-physiology*, 14, 107.
- Borkovec, TD Wall, RL and Stone, NM (1974) False physio-logical feedback and the maintenance of speech anxiety. *Journal of Abnormal Psychology*, 83(2), 164-168.
- Bortner, RW (1969) A short rating scale as a potential measure of pattern A behaviour. *Journal of Chronic Diseases*, 22, 87-91.
- Brack, PE (1986) Parental child rearing practices and the development of Type A behaviour pattern. Unpublished Doctoral Dissertation, Stanford University.

Brener, J (1967) Heart rate analysis. In Venables, PH and Martin, I (eds) A Manual of Psychophysiological Methods North Holland Publishing Company Amsterdam.

Brener, J (1974) A general model of voluntary control applied to the phenomenon of learned cardiovascular change. In Obriest, P etal (eds) Cardiovascular Psychophysiology, Chicago Aldine.

Brener, J and Hothersall, D (1967) Paced respiration and heart rate control. Psychophysiology, 4, 1-6.

Brener, J Kleuria, RA and Goesling, WJ (1969) The effects of different exposures to augmented sensory feedback on the control of heart rate. Psychophysiology, 5, 510-516

Brolund, JW and Schallow, JR (1976) The effect of reward on occipital alpha facilitation by biofeedback. Psychophysiology, 13, 236-241.

Brown, BB (1970) Recognition aspects of consciousness through association with EEG alpha activity represented by a light signal. Psychophysiology, 6, 442-452.

Budzinsky, TH and Stoyva, JM (1969) An instrument for producing deep relaxation by means of analog information feedback. *Journal of Applied Behaviour Analysis*, 2, 231-237.

Budzinsky, TH and Stoyva, JM (1973) Biofeedback techniques in behaviour therapy. In Shapiro, D etal (eds) *Biofeedback and Self Control*, Chicago Aldine.

Burke, EJ and Andriasik, F (1989) Home vs clinical based biofeedback treatment for pediatric migraine: results of treatment through one year follow up. *Headache*, 29(7) 434-440.

Burke, RJ (1982) Interpersonal behaviour and coping style of Type A individuals. *Psychological Reports*, 51, 971-979.

Burke, RJ (1983) Career orientation of Type A individuals. *Psychological Reports*, 53(3), 979-989.

Burke, RJ (1984 a) Beliefs and fears underlying Type A behaviour. *Psychological Reports*, 54(2), 655-662.

Burke, RJ (1984 b) Beliefs and fears underlying Type A behaviour: correlates of time urgency and hostility. *Journal of General Psychology*, 112(2), 133-145.

- Burke, RJ and Weir, T (1980) Personality, value and behavioural correlates of the Type A individual. *Psychological Reports*, 46, 171-181.
- Burke, RJ and Deszca, E (1984) What makes sammy run- so fast and aggressively? beliefs and fears underlying Type A behaviour pattern. *Journal of Occupational Behaviour*, 5(3), 219-227.
- Burnham, MA Pennebaker, JW and Glass, DC (1975) Time consciousness, achievement striving and the Type A coronary prone behaviour pattern. *Journal of Abnormal Psychology*, 84(1), 76-79.
- Byrne, JM Beckman, JE and Smith IM (1986) Developmental assessment: the clinical use and validity of parental report. *Journal of Pediatric Psychology*, 11(4), 549-559.
- Carver, CS and Glass, DC (1978) Coronary prone behaviour pattern and interpersonal aggression. *Journal of Personality and Social Psychology*, 36(4), 361-366.
- Clarke, WR Schrott, HG Leaverton, PE Connor, WE and Laver, RM (1978) Tracking of blood lipids and blood pressures in school aged children: the Muscatine study. *Circulation*, 58, 626-634.

Cleeland, CS (1973) Behavioural techniques in the modification of spasmodic torticollis. *Neurology* Minneapolis, 23, 1241-1247.

Cohen, JB Matthews, KA and Waldron, I (1978) Coronary prone behaviour: developmental and cultural considerations. In Dembroski, TM Weiss, SM Shields, SG etal (eds) *Coronary Prone Behaviour*, NY Springer-Verlag.

Condon, T (1988) The assessment of the Type A behaviour pattern: results from a spouse report approach. *Psychological Medicine*, 18(3), 747-755.

Contrada, RJ and Krantz, DS (1988) Stress, reactivity and Type A behaviour pattern: current status and future directions. *Annals of Behavioural Medicine*, 10(2), 64-70.

Cornsweet, TN and Crane, AD (1973) Training the visual accomodative system. *Vision Response*, 13, 713-715.

Cox, T Mackay, CJ and Page, H (1982) Repetitive work, well being and self reported mood. *Journal of Occupational Behaviour*, 3, 247-252.

Cronbach, LJ (1951) Coefficient alpha and the internal structure of tests. *Psychometrika*, 16, 297-334.

Curtis, JD (1974) The effects of educational interventions on the Type A behaviour pattern. Unpublished Doctoral Dissertation, College of Health, University of Utah.

Davison, GC and Neale, JM (1982) Abnormal Psychology: An Experimental, Clinical Approach. John Wiley and Sons Inc (3rd ed).

DeCaria, MD Proctor, S and Malloy, TE (1974) The effects of false heart rate feedback on self reports of anxiety and on actual heart rate. Behaviour Research and Therapy, 12, 251-253.

Dembroski, TM (1978) Reliability and validity of methods used to assess coronary prone behaviour. In Dembroski, TM Weiss, J et al Coronary Prone Behaviour NY Springer-Verlag.

Dembroski, TM Schmidt, TH and Blumcher, G (1983) Biobehavioural Bases for Coronary Heart Disease. Basel: Karger.

Diamond, S and Franklin, M (1976) Biofeedback: choice of treatment in childhood migraine. Biofeedback and Self Regulation, 1, 349.

- Dimsdale, JE (1988) A perspective on Type A behaviour and coronary disease. New England Journal of Medicine, 318(2), 110-112.**
- Duckro, PN and Cantwell-Simmons, E (1989) A review of studies evaluating biofeedback and relaxation training in the management of pediatric headache. Headache, 29(7), 428-433.**
- Dunbar, HF (1943) Psychosomatic Diagnosis. NY Paul B Hoeber Inc.**
- Eaglestone, JR Kirmil-Gray, K Thoresen, CE et al (1986) Physical health correlates of Type A behaviour in children and adolescents. Journal of Behavioural Medicine, 9, 341- 362.**
- Earnshaw, JB (1956) The diode pump integrator. Electronic Engineering, 28, 26.**
- Elmore, AM (1979) A comparison of the psychophysiological and clinical responses biofeedback for temporal pulse amplitude reduction and biofeedback for increases in hand temperature in the treatment of migraine. Unpublished Doctoral Dissertation, State University of New York at Stony Brook.**

**Elmore, AM and Turskey, B (1978) The biofeedback hypothesis:
An idea in search of theory and method. In Sugeran, AA
and Farter, RE (eds) Expanding Dimensions of Conscious-
ness, NY Springer-Verlag.**

**Engel, BT (1971) Operant conditionong of cardiac function:
a status report. Psychophysiology, 9, 161-177.**

**Engel, BT and Hansen, SP (1966) Operant conditioning of
heart rate slowing. Psychophysiology, 3, 176-187.**

**Engel, BT and Chism, RA (1967) Operant conditioning of
heart rate speeding. Psychophysiology, 3, 418-420.**

**Engel, BT and Bleeker, ER (1974) Application of operant
conditioning techniques to the control of cardiac
arrhythmias. In Obriest, PA Black, RH Brener, J and
Dicara, LV (eds) Cardiovascular Psychophysiology,
Chicago Aldine.**

**Engel, BT Nikoomanesh, P and Schuster, MM (1974) Operant
conditioning of rectosphincteric responses in the
treatment of fecal incontinence. New England Journal of
Medicine, 290, 646-649.**

- Engel, GL (1970) Sudden death and the "medical model" in psychiatry. *Canadian Psychiatric Association Journal*, 15, 527-538.
- Engelhardt, P (1976) Application of biofeedback techniques in a public school setting. *Biofeedback and Self Regulation*, 1, 349.
- Evans, PD and Moran, P (1987) The Framingham Type A scale, vigilant coping, and heart rate reactivity. *Journal of Behavioural Medicine*, 10(3), 311-321.
- Eysenck, HJ and Eysenck, SBG (1975) *Manual of the Eysenck Personality Questionnaire*. London, Hodden and Stroughton.
- Eysenck, HJ and Fulker, D (1983) The components of Type A behaviour and it's genetic determinants. *Personality and Individual Differences*, 4(5), 499-505.
- Feldman, GM (1976) The effect of biofeedback training on respiratory resistance of asthmatic children. *Psychosomatic Medicine*, 38, 27-34.
- Feuerstein, M Labbe, EE and Kuczmierczyk, AR (1986) *Health Psychology: A Psychobiological Perspective*. NY and London Plenum Press, 317-381.

Finley, WW (1977) Operant conditioning of EEG in two patients with epilepsy: methodological and clinical implications. *Pavlovian Journal of Biological Sciences*, 12, 93-111.

Fontana, AF Rosenberg, RL Marcus, JL and Kerns, RD (1986) Type A behaviour pattern, inhibited power motivation and activity inhibition. *Journal of Personality and Social Psychology*, 52(1), 177-183.

Frazier, TW (1966) Avoidance conditioning of heart rate in humans. *Psychophysiology*, 3, 188-202.

Friedberg, CK (1966) *Diseases of the Heart*. Philadelphia, Saunders.

Friedman, M and Rosenman, RH (1959) Association and specific overt behaviour pattern with blood and cardiovascular findings. *Journal of the American Medical Association* 169, 1286-1296.

Friedman, M and Rosenman, RH (1960) Overt behaviour pattern in coronary disease: detection of overt pattern A behaviour in patients with coronary disease by a new psychophysiological procedure. *Journal of the American Medical Association*, 173, 1320-1325.

Friedman, M and Rosenman, RH (1964) Serum lipids and conjunc-
-tival circulation after fat ingestion in men
exhibiting Type A behaviour pattern. *Circulation*, 29,
874-886

Friedman, M and Rosenman, RH (1974) *Type A Behaviour and
Your Heart*. NY Knopf.

Friedman, M Byers, SO Diamond, J and Rosenman, RH (1975)
Plasma catecholamine response of coronary prone
(Type A) subjects to a specific challenge.
Metabolism, 24(2), 205-210.

Friedman, M Thoresen, CE and Gill, JJ (1981) Type A
behaviour: it's possible role, detection and alter-
-ation in patients with ischemic heart disease. In
Hurst, JW (ed) *Update V: The Heart*, NY McGraw-Hill.

Friedman, M thoresen, CE Gill, JJ Ulmer, DK and Price, VA
(1982) feasibility of altering the Type A behaviour
pattern after myocardial infarction. *Circulation*,
66(1), 83-92.

Fruhling, M Basmajian, JV and Simard, TB (1969) A note on
the conscious control of motor units by children
under six. *Journal of Motor Behaviour*, 1, 65-68.

Furnham, AF Hillard, A and Brewin, C (1985) Type A behaviour pattern and the attribution of responsibility. *Motivation and Emotion*, 9, 39-51.

Furnham, AF Borovay, A and Henley, S (1986) Type A behaviour pattern and the recall of positive personality information. *British Journal of Medical Psychology*, 59, 365-374.

Gallacher, JE Yarnell, JW and Butland, BK (1988) Type A behaviour and prevalent heart disease in the Caerphilly study: increase in risk or symptom reporting. *Journal of Epidemiology and Community Health* 42(3), 226-231.

Gasturf, JW Suls, J and Saunders, GS (1980) Type A coronary prone behaviour pattern and social facilitation. *Journal of Personality and Social Psychology*, 48(2), 299.

Gatchel, RJ (1974) Frequency of feedback and learned heart rate control. *Journal of Experimental Psychology*, 103, 274-283.

Glass, DC (1977) *Behaviour Patterns, Stress and Coronary Disease*. Hillsdale, NJ Lawrence Erlbaum Associates.

Glass, CD and Karkoff, LR (1980) Effects of harrassment and competition upon cardiovascular and plasma catecholamine responses in Type A and Type B individuals. *Psychophysiology*, 17(5), 453-463.

Glaros, AG (1975) Expectation effects in the subjective reports of subjects undergoing EEG alpha and beta feedback training. Unpublished Doctoral Dissertation, University of New York.

Grazzi, L, Frediani, F, Rappocosta, B et al (1988) Psychological assessment in tension headache before and after biofeedback treatment. *Headache*, 28(5), 337-338.

Greenhouse, SW and Geisser, S (1959) On methods in the analysis of profile data. *Psychometrika*, 24, 95-112.

Guralnick, S and Mott, M (1976) Biofeedback training with a learning disabled child. *Perceptual and Motor Skills*, 42, 27-30.

Haaga, DA (1987) Treatment of the Type A behaviour pattern. *Clinical Psychology Review*, 7, 557-574.

- Hart, KE (1984) Anxiety management training and anger control for Type A individuals. *Journal of Behaviour Therapy and Experimental Psychiatry*, 15, 133-139.
- Haynes, SG and Feinleib, M (1980) Women, work and coronary heart disease: prospective findings from the Framingham Heart Study. *American Journal of Public Health*, 70, 133-141.
- Haynes, SG Feinleib, M Levins, S Scotch, N and Kannell, WB (1978) The relationship of psychosocial factors to coronary heart disease in the Framingham Study II: prevalence of coronary heart disease. *American Journal of Epidemiology*, 107(5), 384-402.
- Heller, RF (1979) Type A behaviour and coronary heart disease. *British Medical Journal*, 11, 368.
- Herman, S Blumenthal, JA Haney, T Williams, RB and Barefoot, J (1986) Type As who think they are Type Bs: discrepancies between self ratings and interview ratings of Type A (coronary prone) behaviour pattern. *British Journal of Medical Psychology*, 59, 83-88.
- Hilgard, ER and Bower, GH (1975) *Theories of Learning*. NJ Englewood Cliffs Princeton Hall.

- Hofner, KJ (1982) Ursachen von verkehrsvorstößen arbeiten aus dem verkehr. Psychologischen Institut, 19(6), 47-56.
- Hubert, HB Eaker, ED Garrison, RJ and Castelli, WP (1987) Life style correlates of risk factor change in young adults: an eight year study of coronary heart disease in the Framingham offspring. American Journal of Epidemiology, 125, 812-831.
- Hunter, S Russell, H Russell, E et al (1976) Control of finger tip temperature increases via biofeedback in learning disabled children. Perceptual and Motor Skills 43, 743-755.
- James, W (1890) Principles of Psychology. NY Henry Holt.
- Jenkins, CD (1976) Recent evidence supporting psychologic and social risk factors for coronary heart disease: part II. New England Journal of Medicine, 294, 1033-1038.
- Jenkins, CD (1978) Acomparative review of the interview and questionnaire methods in the assessment of the coronary prone behaviour pattern. In Dembroski, TM Weir, J Shields, JL et al (eds) Coronary Prone Behaviour, NY Spriger-Verlag.

Jenkins, CD Friedma, M and Rosenman, RH (1965) The Jenkins Activity Survey for Health Prediction. NC Chapel Hill.

Jenkins, CD Rosenman, RH and Friedman, M (1968) Replicability of rating the coronary prone behaviour pattern. British Journal of Preventive and Social Medicine, 22(1), 16-22.

Jennings, JR and Matthews, KA (1984) The impatience of youth: phasic cardiovascular responses in Type A and Type B elementary school aged boys. Psychosomatic Medicine, 46(6), 498-511.

Jonah, BA (1986) Accident risk and risk taking behaviour among young drivers. Accident Analysis and Prevention, 18(4), 255-271.

Jones, KV (1985) The thrill of victory: blood pressure variability and the Type A behaviour patter. Journal of Behavioural Medicine, 8(3), 277-285.

Johnson, DW (1984) Biofeedback, relaxation and related procedures in the treatment of psychophysiological disorders. In Johnson, DW (ed) Health and Human Behaviour. London, Academic Press.

Johnson, DW and Shaper, AG (1983) Type A behaviour pattern in British men: reliability and intercorrelation of two measures. *Journal of Chronic Diseases*, 36, 203-207

Kahn, JP Kornfeld, DS Frank, KA Heller, SS and Hoar, PF (1980) Type A behaviour and blood pressure during coronary artery bypass surgery. *Psychosomatic Medicine*, 42(2) 407-414.

Kamya, J (1968) Conscious control of brain waves. *Psychology Today*, 1(1), 57-60.

Kaplan, BJ (1975) Biofeedback in epileptics: equivocal equivocal relationship of reinforced EEG frequency to seizure reduction. *Epilepsia*, 16, 477-485.

Kemple, C (1945) Rorchach method and psychosomatic diagnosis personality traits of patients with rheumatic disease, hypertensive cardiovascular disease, coronry occlusion and fracture. *Psychosomatic Medicine*, 7, 85-89.

Kimble, GA (1961) Hilgand & Marquis conditioning & learning N.Y. Appleton Century Croft.

Kimmel, HD (1967) Instrumental conditioning of autonomically mediated behaviour. *Psychological Bulletin*, 337-45.

Kimmel, HD and Hill, FA (1960) Operant conitioning of the GSR. Psychological Reports 7, 555-62.

Koskenvou, M Shapiro, J Langinvainio, H and Romo M (1983). Mortality in relation to coronary prone behaviour: a six year follow up of the Bortner scale in middle aged Finnish men. *Activita Nervosa Superior*, 25(2) 107-109.

Kotses, H.Glaus, KD and Crawford, PR (1976). Operant reduction of frontalis EMG activity in the treatment of asthma in children. *Journal of Psychosomatic Research*, 20, 453-460.

Krantz, DS Arabian, JM Davia, JE and Parker, JS (1982) Type A behaviour and coronary artery bypass surgery: intra-operative blood pressure and pre-operative complications. *Psychosomatic Medicine*, 44, 273-284.

Kratsky, I (1988) Contribution to the questions & possibilities of psychological modification of the TABP as one of the possible ways to the prevention of CHD risks. *Studia Psychologica*, 30(1) 65-77.

Kuhlman, DM & Wimberly DI (1976). Expectations of choice choice behaviour held by cooperators, competitors and individualists across four classes of experimental game. *Journal of Personality and Social Psychology*, 34, 69-81.

Lekarczik, DT and Hill, KT (1969) Self esteem, test anxiety, stress, and verbal learning. *Developmental Psychology*, 1, 147-154.

Lang, PJ (1974) Learned control of human heart rate in a computer directed environment. In Obriest, Pa Black, HA Brener, J and Dicara, LV (eds) *Cardiovascular Psychophysiology*, NY Aldine.

Lang, PJ and Lazovik, AD (1963) Experimental desensitization of a phobia. *Journal of Abnormal Social Psychology*, 66, 519-525.

Lang, PJ and Twentyman, CT (1976) Learning to control heart rate: effects of varying incentive and criterion of success on past performance. *Psychophysiology*, 13, 378-385.

Lang, PJ Stronfe, LA and Hastings, JE (1967) Effects of feedback and instructional set on the control of cardiac variability. *Journal of Experimental Psychology*, 75, 425-431.

- Lawler, KA and Schmied, L Mitchel, VP and Rixse, D (1984)
Type A behaviour pattern and physiological
responsivity in young women. *Journal of Psychosomatic
Research*, 28, 197-204.
- Leon, GE Finn, SE Murray, D and Bailey, JM (1988) Inability
to predict cardiovascular disease from hostility scores
or MMPI items related to Type A behaviour. *Journal of
Consulting and Clinical Psychology*, 56(4), 597-600.
- Levenson, RW (1974) Automated system for direct measurement
and feedback of total respiratory resistance by a
forced oscillation technique. *Psychophysiology*, 11,
86-90.
- Lundberg, U Hedman, M Melin, B and Frankenhaeser, M (1987)
Type A behaviour pattern in males and females as
related to physiological reactivity and blood lipids.
Reports from Dept Psychology, University of Stockholm
number 671, p12.
- Lynch, WG Hama, H Kohn, S and Miller, NE (1976) Instrumental
control of peripheral vasomotor responses in children.
Psychophysiology, 13, 219-221.

- Macdougall, JM Dembroski, TM and Musante, L (1979) The structured interview and questionnaire methods of assessing coronary prone behaviour in male and female college students. *Journal of Behavioural Medicine*, 2, 71-83.
- Mackay, CJ Cox, T Burrows, G and Lazzerini, T (1978) An inventory for the measurement of self reported stress and arousal. *British Journal of Social and Clinical Psychology*, 17(3), 283-284.
- Manuk, SB Craft, SA and Gold, KJ (1978) coronary prone behaviour pattern and cardiovascular response. *Psychophysiology*, 15, 403-411.
- Marsh, P and Collett, P (1987) The car as a weapon. Etc, 44(2), 146-151.
- Matthews, KA (1977) Caregiver-child interactions and the Type A coronary prone behaviour pattern. *Child Development*, 48, 1752-1756.
- Matthews, KA (1978) Assessment and development antecedents of pattern A behaviour in children. In Dembroski, TM Weiss, SM Shields, SG etal (eds) *Coronary Prone Behaviour*, NY Springer Verlag.

- Matthews, KA (1979) efforts to control by children and adults with the Type A coronary prone behaviour pattern. *Child Development*, 50, 842-847.
- Matthews, KA and Krantz, DS (1976) Resemblance of twins and their parents in pattern A behaviour. *Psychosomatic Medicine*, 28, 140-144.
- Matthews, KA and Angulo, J (1980) Measurement of the Type A behaviour pattern in children: assessment of children's competitiveness, impatience anger and aggression. *Child Development*, 51, 466-475.
- Matthews, KA and Siegel, T (1983) Type A behaviour by children, social comparison and standards for self evaluation. *Developmental Psychology*, 19, 135-140.
- Matthews, KA and Haynes, SG (1986) Type A behaviour and coronary risk: update and critical evaluation. *American Journal of Epidemiology*, 123, 923-960.
- Matthews, KA and Woodall, KL (1988) Childhood origins of overt Type A behaviour and cardiovascular reactivity to behavioural stressors. *Annals of Behavioural Medicine*, 10(2), 71-77.

Matthews, KA Rosenman, RH Dembroski, TM McDougall, JM and Harris, E (1984) Familial resemblance in components of the Type A behaviour pattern: reanalysis of the California Type A twin study. *Psychosomatic Medicine*, 46, 512-522.

Matthews, KA Weiss, SM Detra, T etal (1986) *Handbook of Sress, Reactivity and Cardiovascular Disease*. NY Wiley and Sons.

McCoy, GC Blanchard, EB Wiltrock, DA Morrison, S etal (1988) Biochemical changes associated with thermal biofeedback treatment of hypertension. *Biofeedback and Self Regulation*, 13(2), 139-150.

McGill, HC (1984) Persistent problems in the pathogenesis of atherosclerosis. *Arteriosclerosis*, 4, 443-451.

Miller, NE (1969) Learning of visceral and glandular responses. *Science*, 163, 434-453.

Miller, NE and Dwarin, BR (1974) Visceral learning: recent difficulties with curarized rats and significant problems for human research. In Obriest, PA etal *Cardiovascular Psychophysiology*, Chicago Aldine.

Miller, NE DiCara, LV Solomon, VH Weiss, JM and Dworkin, BR
(1970) Learned modifications of autonomic functions: a
review and some new data. *Circulation*, 36(1), 3-11.

Mischel, W (1968) *Personality and Assessment*. NY Wiley and
Sons.

Mischel, W (1973) Towards a cognitive social learning
reconceptualization of personality. *Psychological
Reviews*, 80, 252-283.

Mookherjee, HN (1986) Comparison of some personality charac-
-teristics of male problem drinkers in rural
Tennessee. *Journal of Alcohol and Drug Education*,
31(2),
22-28.

Murphy, PJ Darwin, J and Murphy, DA (1977) EEG feedback
training for cerebral dysfunction: a research
programme with learning disabled adolescents.
Biofeedback and Self Regulation, 2, 228.

Muskatel, N Woolfolk, RL Carrington, P Lehrer, PM and
McCann, BS (1984) Effects of meditation training on
aspects of coronary behaviour. *Perceptual and Motor
Skills*, 58, 515-518.

Nisbett, RE and Schachter, S (1966) Cognitive manipulation of pain. *Journal of Experimental and Social Psychology* 2, 227-236.

Obriest, PA Black, RH Brener, J and Dicara, LV (1974) (eds) *Cardiovascular Psychophysiology*. Chicago, Aldine

Obriest, PA Light, KC McCubbin, JA Hutchinson, JS and Hoffer JL (1978) Pulse transit time: relationship to blood pressure. *Behavioural Research Methods and Instrumentation*, 10, 623-626.

O'looney, BA (1984) The assessment of Type A behaviour and the prediction of CHD. *Current Psychological Research and Reviews*, Winter 84, 63-84.

Osler, W (1892) *Lecture on Angina Pectoris and Allied States* NY Appleton.

Panek, PE and Wagner, EE (1986) Hand held personality variables related to automative moving violation in female drivers. *Journal of Personality assessment*, 50(2), 208-211.

Parkinson, B and Colgan, L (1988) False autonomic feedback: effects of attention to feedback on ratings of

- pleasant and unpleasant target stimuli. *Motivation and Emotion*, 12(1), 87-98.
- Paskewitz, DA and Orne, MT (1973) Visual effects of alpha feedback training. *Science*, 181, 360-363.
- Pedersen, NL Lichtenstein, P Plomin, R DeFaire, U McClearn, GE and Matthews, KA (1989) Genetic and environmental influences for Type A like measures and related traits: a study of twins reared apart and twins reared together. *Psychosomatic medicine*, 51, 428-440.
- Perkins, KA (1986) Change in Type A and B males as a function of response cost and task difficulty. *Psychophysiology*, 21(1), 14-21.
- Perry, AR (1986) Motor vehicle drivers' behaviour. *Perceptual and Motor Skills*, 63(2), 875-878.
- Pitariu, H (1985) The Holtzman Inkblot Technique: a tentative study with professional drivers. XXIII International Congress of Psychology (Acapulco, Mexico) *Revue Roumaine des Science Sociales Serie de Psychologie*, 29(1), 75-81.
- Powell, GE (1981) *Brain Function Therapy*, Academic Press.

Powell, LH Friedman, M Thoresen, CE Gill, JJ and Ulmer, DK
(1984) Can the Type A behaviour pattern be altered after
myocardial infarction? a second year report from the
CCPP. *Psychosomatic Medicine*, 46, 293-313.

Price, VA (1983) *Type A Behaviour Pattern: A Model for
Research and Therapy*. NY Academic Press.

Price, VA (1988) Research and clinical issues in treating
Type A behaviour. In Houston, BK and Snyder, CR (eds)
*Type A Behaviour Pattern: Research, Theory and Inter-
vention*, NY John Wiley.

Prigatano, GP and Johnson, HJ (1972) Biofeedback control of
heart rate variability to phobic stimuli: a new
approach to treating spider phobia. In *Proceedings
of the Annual Convention, American Psychological
Association, Washington*, 403-404.

Prior, DW Goodyear, RK and Holen, MC (1983) EMG biofeedback
training of Type A and B behaviour pattern subjects.
Journal of Consulting Psychology, 30(3), 316-322.

Radnitz, CL and Blanchard, EB (1988) Bowel sound biofeedback
as a treatment for irritable bowel syndrome.
Biofeedback and Self Regulation, 13(2), 169-179.

- Ragland, DR and Brand, RJ (1988) Type A behaviour and mortality from CHD. *New England Journal of Medicine*, 318(2), 65-69.
- Rahe, RH, Havig, L and Rosenman, RH (1978) The heritability of Type A behaviour. *Psychosomatic Medicine*, 40, 478-486.
- Reinking, RH and Kohl, ML (1975) Effects of various forms of relaxation training on physiological and self report measures of relaxation. *Journal of Consulting and Clinical Psychology*, 43, 596-600.
- Rhodewalt, F (1984) Self attribution, self involvement and Type A coronary prone behaviour pattern. *Journal of Personality and Social Psychology*, 47, 662-670.
- Rioux, SC and Wapner, S (1986) Commitment to use of automobile seat belts: an experimental analysis. *Journal of Environmental Psychology*, 6(3), 189-204.
- Robinson, N and Heller, RF (1980) Experience with the Bortner questionnaire as a measure of Type A behaviour in a sample of UK families. *Psychological Medicine*, 10, 567-571.

Rosenman, RH (1978) The interview method of assessment of the coronary prone behaviour pattern. In Dembroski, TM Weiss, SM etal (eds) Coronary Prone Behaviour, NY Springer Verlag.

Rosenman, RH and Friedman, M (1977) Modifying the Type A behaviour pattern. Journal of Psychosomatic Research, 21, 323-331.

Roskies, E (1987) Stress Management for the Healthy Type A. NY Guilford Press.

Roskies, E Seraganian, P Osensohn, R Hanley, JA etal (1986) The Montreal Type A intervention project: major findings. Health Psychology, 5, 45-69.

Ruberman, W Weinblatt, E Goldberg, DD and Chandbury, B (1984) Psychosocial influence on mortality after myocardial infarction. New England Journal of Medicine, 311, 552-559.

Rustin, RM Dramaiz, M Kittel, F etal (1976) Validation de techniques d'évaluation du profil comportemental "A" utilisées dans le project Belge de Prevention de affections cardiovasculaires. Revue Epidemiologie et Sante Publique, 24, 497-507.

Schachter, S (1964) The interaction of cognitive and physiological determinants of emotional state. In Berkowitz, L (ed) *Advances in Experimental Social Psychology*, Academic Press.

Schneiderman, N Dauth, GW and Van Dercar, DH (1974) *Electrocardiogram: techniques and analysis*. In Thompson, RF and Patterson, MM (eds) *Bioelectric Recording Techniques (Part C: Reception & Effector Processes)* Academic Press.

Schucker, B and Jacobs, DR (1977) assessment of behavioural risk for coronary disease by voice characteristics. *Psychosomatic Medicine*, 39(4), 219-228.

Schwartz, GE (1973) Biofeedback as therapy: some theoretical and practical issues. *American Psychologist*, 28, 666-673.

Schwartz, DP Burish, TG O'rourke, DF and Holmes, DS (1986) Influence of personal and universal failure on the subsequent performance of persons with Type A and Type B patterns. *Journal of Personality and Social Psychology*, 51(2), 459-462.

Seligman, MEP (1975) *Helplessness: On Depression, Development and Death*. WH Freeman.

Seraganian, P Roskies, E Hanley, JA Oseason, R and Collu, R
(1987) Failure to alter psychophysiological reactivity in Type A men with physical exercise or stress management programmes. *Psychology and Health* 1(3), 195-213.

Shahidi, S (1986) Effects of false heart rate biofeedback on subjective and physiological measures of relaxation in high and low suggestible subjects. Unpublished Masters Dissertation, University of Surrey.

Shahidi, S and Powell, GE (1988) Biofeedback training of relaxation: the effects of false feedback and the influence of suggestibility. *Personality and Individual Differences*, 9(6), 990-995.

Shapiro, D and Surwit, RS (1979) Biofeedback. In Pomerlean, OF and Brady, JP (eds) *Behavioural Medicine: Theory & Practice*, Baltimore, William & Wilkins.

Shekelle, RB Gale, M Ostfeld, AM and Paul, O (1983) Hostility, risk of CHD and mortality. *Psychosomatic Medicine*, 45(2), 109-114.

Shern, DW (1962) Operant conditioning of heart rate. *Science* 137, 530-531.

- Siegel, JM and Leitch, CJ (1981) Assessment of the Type A behaviour pattern in adolescents. *Psychosomatic Medicine*, 43, 45-56.
- Simard, TG (1969) Fine sensorimotor control in healthy children: an electromyographic study. *Pediatrics*, 43, 1035-1041.
- Smith, TW and Anderson, NB (1986) Models of personality and disease: an interactional approach to Type A behaviour Pattern and cardiovascular risk. *Journal of Personality and Social Psychology*, 50, 1166-1177.
- Spiga, R (1986) Social interaction and cardiovascular responses in boys exhibiting the coronary prone behaviour pattern. *Journal of Pediatric Psychology*, 11(1), 59-69.
- Steinberg, L (1986) Stability and instability of Type A behaviour from childhood to young adulthood. *Developmental Psychology*, 22, 393-402.
- Sterman, MB (1973) Neurophysiological and clinical studies of sensorimotor EEG biofeedback training: some effects on epilepsy. In Birk, L (ed) *Biofeedback: Behavioural Medicine*, NY Grune & Stratton.

Sterman, MB and Friar, L (1972) Suppression of seizures in an epileptic following EEG feedback training. *Electroencephalographic Clinical Neurophysiology*, 33, 89-95.

Sterman, MB Lopresti, RW and Fairchild, MD (1969) EEG and behavioural studies of monomethyl hydrazine toxicity in the cat. Technical Report AMRL-TR-69-3, Air Systems Command Wright Patterson Air Force Base, Ohio.

Stern, GS and Elder, RD (1982) The role of challenging incentives in feedback assisted heart rate reduction for coronary prone adult males. *Biofeedback and Self Regulation*, 7(1), 53-69.

Stern, RM Botto, RW and Herrick, CD (1972) Behavioural and physiological effects of false heart rate feedback: a replication and extension. *Psychophysiology*, 9(1), 21-29

Sternbach, RA (1964) The effects of instructional sets on autonomic responsivity. *Psychophysiology*, 1(1), 67.

Stroebe, CF and Glueck, BC (1973) Biofeedback treatment in medicine and psychiatry: an ultimate placebo? In Birk, L (ed) *Biofeedback: Behavioural Medicine*, NY Grune & Stratton.

Suinn, RM (1974) Behaviour therapy for cardiac patients.

Behaviour Therapy, 5, 569-571.

Suinn, RM (1976) How to break the vicious circle of stress.

Psychology Today, 10, 59-60.

Thayer, RE (1967) Measurement of activation through self

report. Psychological Reports, 20, 663-678.

**Thompson, BP (1976) Effectiveness of relaxation techniques
in reducing anxiety and stress factors in Type A post
myocardial infarction patients. Unpublished Doctoral
Dissertation, University of Massachusetts.**

**Thoresen, CE and Pattillo, JR (1988) Exploring the Type A
behaviour pattern in children and adolescents. In
Houston, BK and Snyder, CR (eds) Type A Behaviour
Pattern: Research, Theory and Intervention, NY
John Wiley.**

**Thoresen, CE Friedman, M Powell, LH Gill, JJ and Ulmer, D
(1985) Altering the Type A behaviour pattern in post
infarction patients. Journal of Cardiopulmonary
Rehabilitation, 5, 258-266.**

Thurman, CW (1983) effects of a rational emotive treatment programme on Type A behaviour among college students. *Journal of College Student Personnel*, 24, 417-423.

Tove, PA and Czekajewski, J (1964) Pulse period meter with short response time applied to cardiometry. *Electronic Engineering*, 36, 290-295.

Tramill, JL Kleinhammer-Tramill, PJ Davis, SF and Parks, CS (1985) The relationship between Type A and Type B behaviour patterns and level of self esteem. *Psychological Records*, 35(3), 323-327.

Valins, S (1966) Cognitive effects of false heart rate feedback. *Journal of Personality and Social Psychology*, 4, 400-408.

Van Egeren, LF (1979) Cardiovascular change during social competition in a mixed motive game. *Journal of Personality and Social Psychology*, 37(6), 858-864.

Vasina, B (1983) A/B behaviour pattern and psychophysiological reactions to laboratory challenges: a preliminary study. *Activitas Nervosa Superior*, 25(2), 104-105.

Van Schijndel, M De May, H and Naring, G (1984) Effects of behavioural control & Type A behaviour on cardiovascular responses. *Psychophysiology*, 21(5), 501-509.

Visintainer, PF and Matthews, KA (in press) Stability of overt Type A behaviour in children: results from a two and five year longitudinal study. Child Development (Quoted in Matthews and Woodall, 1988).

Volpe, R (1977) Feedback facilitated relaxation training in primary prevention of drug abuse in early adolescence. Journal of Drug Education, 7, 179-194.

Ward, MM (1986) Cardiovascular responses in Type A and Type B men to a series of stressors. Behavioural Medicine, 9(1), 43-49.

Weidner, G Sexton, G Mattarazzo, JD etal (1988) Type A behaviour in children, adolescents and their parents. Developmental Psychology, 24(1), 118-121.

Weiner, N (1948) Cybernetics. NY John Wiley.

Weiss, T and Engel, BT (1970) Voluntary control of premature ventricular contractions in patients. American Journal Cardiology, 26, 666.

Welgan, PR (1974) Learned control of gastric acid secretion in ulcer patients. Psychosomatic Medicine, 36, 411-419.

Wells, DT Feather, BW and Headrick, MW (1973) The effects of immediate feedback upon voluntary control of salivation rate. *Psychophysiology*, 10, 501-509.

Whalen, CK and Henker, B (1986) Type A behaviour pattern in normal and hyperactive children: multisource evidence overlapping constructs. *Child Development*, 57, 688-699.

Wickramasekara, I (1973) The application of verbal instructions and EMG feedback training to the management of tension headache: preliminary observations. *Headache*, 13, 74-76.

Williams, RB (1975) Physiologic mechanisms underlying the association between psychosocial factors and CHD. In Gentry, WD and Williams, RB (eds) *Psychosocial Aspects of Myocardial Infarction and Coronary Care*, St Louise, CV Mosby.

Williams, RB (1978) Psychophysiological processes, the coronary prone behaviour pattern and CHD. In Dem-broski, TM Weiss, SM et al (eds) *Coronary Prone Behaviour*, NY Springer Verlag.

Williams, RB Barefoot, JC and Shekelle, RB (1985) The health consequences of hostility. In Chesney, MA and Rosen-

-man, RH (eds) Anger, Hostility and Behavioural
Medicine, NY Hemisphere/ McGraw Hill.

Winer, BJ (1971) Statistical Principles in Experimental
Design. NY McGraw Hill.

Wolf, TM Sklov, MC Wenzl, TA Hunter, SM and Berenson, GS
(1982) Validation of a measure of Type A behaviour
pattern in children: Bogalusa heart study. Child Deve-
-lopment, 53, 126-135.

Wolpe, J (1959) Psychotherapy by Reciprocal Inhibition.
Stanford, California, Stanford University Press.

Woods, PJ (1987) Reductions in Type A behaviour pattern
anxiety, anger and physical illness as related to
changes in irrational beliefs: results of a demonstra-
-tion project in industry. Journal of Rational Emotive
Therapy, 5(4), 213-237.

Yarnold, PR and Mueser, KT (1988) Student version of the
Jenkins Activity Survey. In Hersen, M and Bellak, AS
(eds) Dictionary of Behavioural Assessment Techniques,
Beverley Hills, CA, Pergamon Press.

Yarnold, PR and Grimm, LG (1988) Interpersonal dominance of Type As and Type Bs during involved group discussion. *Journal of Applied Social Psychology*, 18, 787-795.

Yarnold, PR Mueser, KT and Grau, BW (1988) Fully faceted Type A survey for students. In Hersen, M and Bellak, AS (eds) *Dictionary of Behavioural Assessment Techniques*, Beverley Hills, CA Pergamon Press.

Yarnold, PR Mueser, KT Grau, BW and Grimm, LG (1986) The reliability of the student version of the Jenkins Activity Survey. *Journal of Behavioural Medicine*, 9(4) 401-413.

Zajonc, RB (1965) Social Facilitation. *Science*, 149, 269-274

Zyzanski, SJ and Jenkins, CD (1970) Basic dimensions within the coronary prone behaviour pattern. *Journal of Chronic Diseases*, 22, 781-795.

APPENDIX 1: THE EYSENCK TYPE A QUESTIONNAIRE

Please answer each of the following questions by putting a circle around either Yes or No. There are no right or wrong answers. Please answer the questions as honestly as you can.

1. Are you an ambitious, forceful person ? Yes No
2. Do you take things as they come without getting too irritated ? Yes No
3. Are you very keen that people should know about when you have done a good a job ? Yes No
4. Do you refuse to be rushed ? Yes No
5. Do you strongly need to do well at school ? Yes No
6. Can you wait patiently without getting upset ? Yes No
7. Are you ambitious to get on socially ? Yes No
8. Do you do things as they come rather than trying to do many things at once ? Yes No
9. Are you aware of time pressures and deadlines ? Yes No
10. Are you a slow and calm talker ? Yes No
11. Do you get involved in a lot of different activities ? Yes No
12. Are you slow at eating and other activities ? Yes No
13. Do things and people often make you angry ? Yes No
14. Are you quite satisfied with your school work without too much ambition? Yes No
15. Do you get things done quickly ? Yes No
16. Would you call yourself easy-going ? Yes No
17. Would you call yourself a person of action ? Yes No
18. Do you express your feelings freely ? Yes No
19. When under pressure of stress, do you do something about it immediately ? Yes No
20. Do you have many interests outside school ? Yes No

- | | | |
|--|-----|----|
| 21. Do you go "all out" whatever you are doing ? | Yes | No |
| 22. When you are getting tired while working do you slow down until you revive ? | Yes | No |
| 23. Does it irritate you a lot to be interrupted in your work ? | Yes | No |
| 24. Do you have less energy than most people ? | Yes | No |
| 25. Do you hate queuing or waiting in line ? | Yes | No |
| 26. Have you always been rather even-tempered ? | Yes | No |
| 27. Are you always in a hurry to get somewhere ? | Yes | No |
| 28. Do you prefer not to compete with others ? | Yes | No |
| 29. Do you usually find you make much greater efforts than others to get something finished? | Yes | No |
| 30. Are you usually slow in your movement ? | Yes | No |
| 31. Do you enjoy competing and try hard to win ? | Yes | No |
| 32. Do people consider you relaxed and easy-going ? | Yes | No |
| 33. Are you usually impatient and interrupt people who are slow at coming to the point ? | Yes | No |
| 34. Are you casual about appointments ? | Yes | No |
-
-

please provide the following information ;

Name:

Age:

Sex: male female

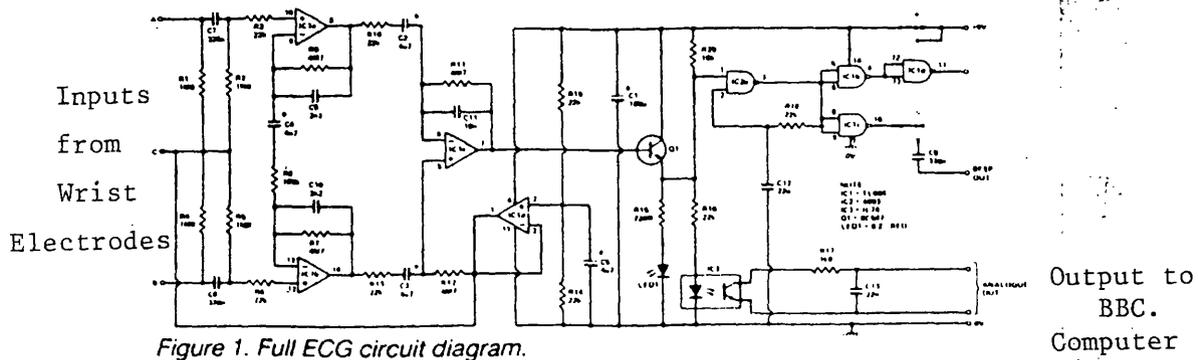


Figure 1. Full ECG circuit diagram.

IC1c is a differential amplifier with a high frequency roll-off at 4Hz, set by R11/C11. This stage provides further common mode rejection to eliminate interference (mainly 50Hz hum), and also has a gain of 50, making a total gain through the system of 2500.

The fourth op-amp in the TL084 package is simply used as a unity gain amp to bias up the other op-amps to half-supply. Q1 is connected as an emitter-follower and it drives the LED indicator (LED1) plus the opto-coupler LED in IC3. LED1 gives a visual indication of the heart rate, while the isolated output from IC3 can be coupled to the analogue port of the Beeb. IC2a is a gated oscillator; normally its pin 1 input is held low; its output will be high and C12 will charge up through R18. When it does, pin 2 goes high and the output goes low. C12 then discharges into pin 3, and the cycle starts again. When Q1 is switched on, pin 1 is pulled up to nearly 9V, and the output will be held low for as long as this condition is maintained. The other gates of the 4093 are used as buffers; the main output is available through the switch SW1.

Output to
BBC.
Computer

E&CM


```

6260IFFU%=3 PROCD
6270IFFU%=4 G%=0:PROCT
6280IFFU%=5 PROCX
6290IFFU%=6 PROCV
6400UNTILFALSE:ENDPROC
7000DEFFPROCd:REPEAT:PROCh("Patient Details",8):PRINT""Press RETURN to leave th
e same.":*FX15,1
7100REPEAT:PRINTTAB(0,7)"Name :":INPUT""I#:UNTILFN1
7120IFI#=""PRINTTAB(8,7)N#ELSEN#=#I#
7130REPEAT:PRINTTAB(0,10)"Date :":INPUT""I#:UNTILFN1
7140IFI#=""PRINTTAB(8,10)D#ELSED#=#I#
7150REPEAT:PRINTTAB(0,13)" Age :":INPUT""I#:UNTILFN1
7170IFI#=""PRINTTAB(8,13)A#ELSEA#=#I#
7200REPEAT:PRINTTAB(0,16)" Sex :":INPUT""I#:UNTILFN1
7220IFI#=""PRINTTAB(8,16)S#ELSE#=#I#
7250REPEAT:PRINTTAB(0,19)"Details :":INPUT""I#:UNTILFN1
7270IFI#=""PRINTTAB(11,19)E#ELSEE#=#I#
7500PRINTTAB(5,23)"Are details correct (Y/N) ? ":REPEAT:I#=#GET#:UNTILINSTR("Yy
Nn",I#):UNTILINSTR("Yy",I#):ENDPROC
7900DEFFN1:IFLENI#>30 PRINT"SPC10" Too Long":FORI=1TO5000:NEXT:VDU11,11,11,11:P
RINTSPC160:=0 ELSE =-1
8000DEFFPROCf:GCOL0,2:REM VDU19,1,6,0,0,0
8140MOVE132,132:DRAW132,700:DRAW1204,700:DRAW1204,132:DRAW132,132:FORY%=136TO70
OSTEP56.4:MOVE124,Y%:DRAW132,Y%:MOVE1212,Y%:DRAW1204,Y%:NEXT
8160FORX=136TO1200STEP53.2:MOVEX,132:DRAWX,124:MOVEX,700:DRAWX,708:NEXT
8170FORX=136TO1200STEP266:MOVEX,132:DRAWX,116:MOVEX,700:DRAWX,716:NEXT
8180IFG%VDU5:MOVE0,710:PRINT;U%:MOVE0,428:PRINT;(L%+U%)/2:MOVE32,148:PRINT;L%:M
OVE 120,108:PRINT"0":MOVE652,108:PRINT;gst/2;" Minutes":MOVE1190,108:PRINT;gst;
8200VDU4:COLOUR2:PRINTTAB(9,0)"Heart and Pulse Monitor"TAB(9,2)"Heart Rate :
Bts/min."
8300PRINTTAB(0,4)" Name :"" Date :"" Age :"" Sex :""Details :":CO
LOUR3:PRINTTAB(10,4)N#TAB(10,5)D#TAB(10,6)A#TAB(10,7)S#TAB(10,8)E#
8900COLOUR2:PRINTTAB(6,30)"Press SPACE BAR, COPY or 'P'":GCOL0,3:GCOL0,128:COLO
UR3:PROCu:IFQ%VDU2,21:PRINT"" Time"SPC13"Rate":VDU6,3
8950ENDPROC
9000DEFFPROCy:*FX15,1
9100PROCh("Keys for Graphical Options",2):PRINT""Use cursor keys for the follo
wing:"
9250PRINT"" Up _Increase trigger level,""" Down _Decrease trigger level.
"""" 'P' _ Pause screen temporarily"""" COPY _ Dump screen to a printer""
"" SPACE _ Return to main menu"
9300PRINT"" Press SPACE BAR to return"
9400REPEAT:UNTILGET=32:ENDPROC
10000DEFFPROCe:PROCC:*FX15,1
10110VDU23;11,255;0;0;0:PRINTTAB(7,30)"Is Printer ready (Y/N) ? ":REPEAT:I#=#GET
#:UNTILINSTR("YyNn",I#):PROCC
10150IFINSTR("Yy",I#) VDU26:CALL&900:VDU3:VDU24,136;136;1200;696;:COLOUR2:PRINTT
AB(6,30)"Press SPACE BAR, COPY or 'P'":COLOUR3
10500VDU23;11,0;0;0;0
10600P%=0:ENDPROC
11000DEFFPROCp:IX=TIME:*FX4,1
11020PROCC:*FX15,1
11030PRINTTAB(9,30)"Press 'C' to continue ":REPEATI#=#GET#:UNTILINSTR("Cc",I#):P
ROCC:COLOUR2:PRINTTAB(6,30)"Press SPACE BAR, COPY or 'P'":COLOUR3:*FX4
11060TIME=IX:ENDPROC
12000DEFFPROCb:*FX15,1
12050PROCh("Battery Test",10):PRINT"" Connect up the interface and
switch it to the TEST position then press the SPACE BAR."
12200REPEAT:UNTILGET=32
12210VDU23;11,0;0;0;0
12300REPEAT:*FX15,1
12310J=0:FORI=1TO250:J=J+ADVAL1:NEXT:B%=J/250:V=B%/&10000*1.8*112/12:PRINTTAB(8,
13);:IV=INT(V*10)/10
12340IFIV=4.7ANDV<=5.3PRINT"Battery O.K.(";IV;"V)"SPC9"""" Press the SPACE B
AR then switch the interface to the ON position."
12350IFV<4.7PRINT"Battery Low(";IV;"V)"SPC9"""" Please check connections

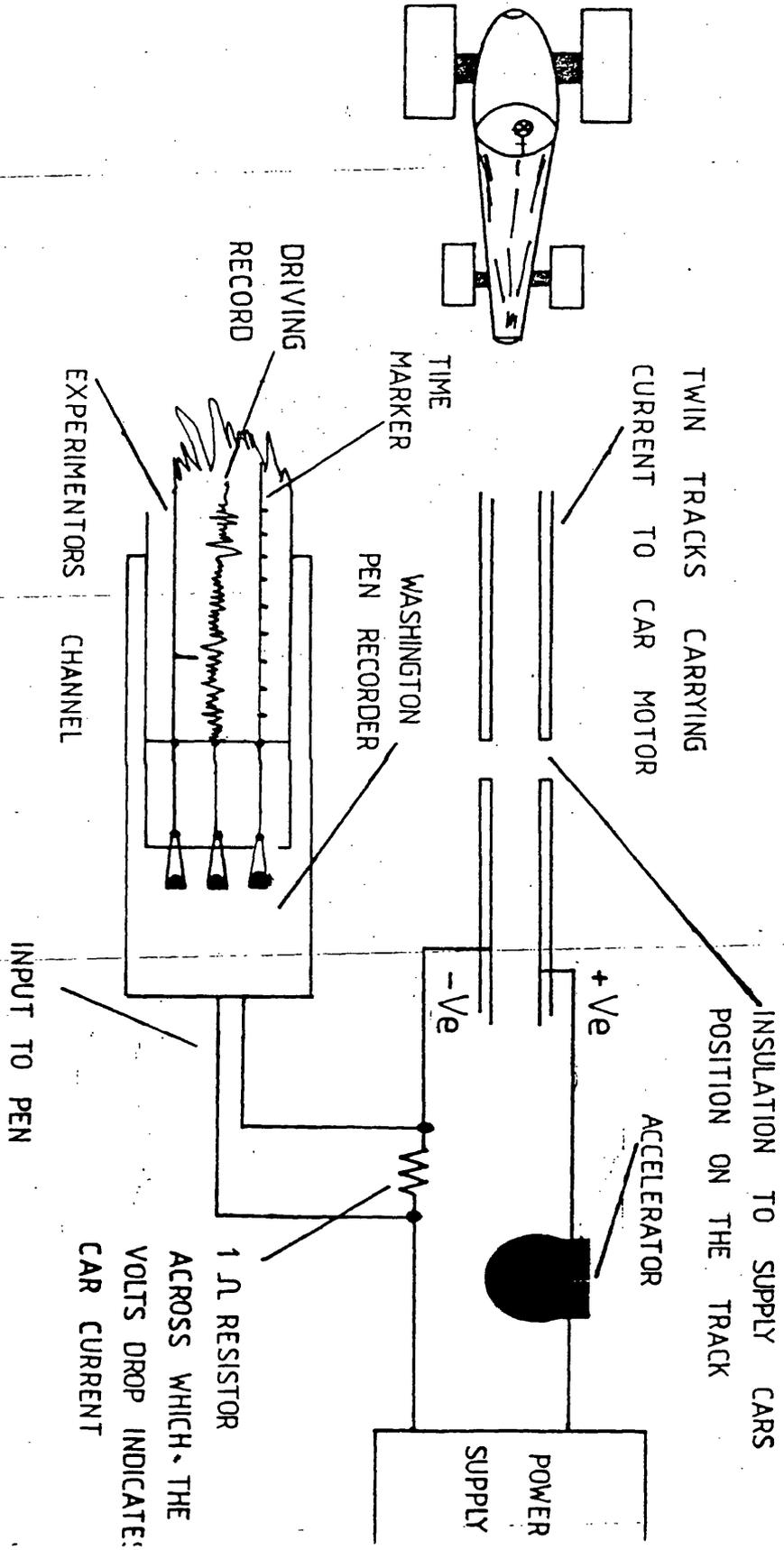
```

```

OR THEN SWITCH THE INTERFACE TO THE ON POSITION.
12350IFV<4.7PRINT"Battery Low(";IV;"V)"SPC9"" Please check connections
and/or replace battery."SPC9
12360IFIV>5.3 PRINT"Voltage High(";IV;"V)"SPC9"" Please check connections
between computer and heart monitor."SPC9
12370UNTILIV>=4.7ANDV<=5.3ANDINKEY(0)=32
12375CLS
12380J=0:FORI=1TO500:J=J+ADVAL1:NEXT:BX=J/500:V=BZ/&10000*1.8*112/12:OZ=BZ/100-4
04:VDU23;11,255;0;0;0:ENDPROC
13000DEFFPROCw:IFTIME>MZR%=0
13050IFTIME>VX+H% V%=TIME-TIME MODH%
13060am%=@%:@%=&908:V%=V%+H%:VDU2,21:K%=TIME DIV100:PRINTRIGHT$("0"+STR$(K%DIV36
00MOD24),2)": "RIGHT$( "0"+STR$(K%DIV60MOD60),2)": "RIGHT$( "0"+STR$(K%MOD60),2)"
-";:PRINTR%:VDU6,3:@%=am%:ENDPROC
14000DEFFPROCv:LOCALH,M
14050PROC("Pulse rate printout",6):PRINT"SPC5"Output to printer(Y/N)?";
14110REPEAT:I#=GET#:UNTILINSTR("YN",I#):OZ=I#="Y"
14120IFNOTO%THENENDPROC
14200IFtok vp=8:GOTO14300 ELSE14210
14205FORI=1TO2000:NEXT:PRINTTAB(0,8)SPC255
14210PRINTTAB(5,8)"Enter time(HH:MM)?";:INPUT""I#:IFMID$(I#,3,1)<>:"ORLENI#<>5P
RINT"SPC10"Incorrect format":GOTO14205
14230H=VAL(MID$(I#,1,2)):M=VAL(MID$(I#,4,2)):IFH>23ORM>59PRINT"SPC10"Invalid ti
me":GOTO14205
14250TIME=(H*3600+M*60)*100:tok=-1:vp=12
14300PRINTTAB(1,vp)"Delay between samples(sec.)?";:INPUTTAB(33,vp)""I:IFI<10RI>3
600PRINTTAB(33,vp)SPC20:GOTO14300
14320H%=I*100:V%=0:ENDPROC
15000DEFFPROCx:G%=TRUE:ss=S:S=rgs:PROC("Heart Rate Graph",7):PRINTTAB(1,4)"Press
RETURNto enter displayed value"
15220PRINTTAB(4,8)"Graph scan time(";gst;" minutes)?";:INPUT""I#:IFI#=""THEN1530
0
15240I=INTVALI#:IFI<10R I>999THEN15220
15250gst=I:rgs=.1773333/gst*1.1:S=rgs
15300PRINTTAB(8,12)"Minimum rate(";L%;" )?";:INPUT""I#:IFI#=""THEN15350
15320I=INTVALI#:IFI<0ORI>200THEN15300
15330L%=I
15350PRINTTAB(8,15)"Maximum rate(";U%;" )?";:INPUT""I#:IFI#=""THEN15400
15370I=INTVALI#:IFI<L%+10ORI>300THEN15350
15380U%=I
15400PRINTTAB(12,18)"Alarm(";a#;" )?";:I#=GET#:IFI#=CHR#13 THEN15500
15420IFI#="Y"ORI#="y" a#=I#:A%=-1:GOTO15500
15430IFI#<>"N"ANDI#<>"n" THEN15400
15440a#=I#:A%=0
15500PROCT:S=ss:ENDPROC
20000DEFFPROCc:PRINTTAB(0,30)SPC79;:COLOUR3:ENDPROC
21000DEFFPROCch(I#,1):VDU22,7:PRINTCHR#129CHR#157CHR#131CHR#141SPCI;I#:PRINTCHR#12
9CHR#157CHR#131CHR#141SPCI;I#:ENDPROC
22000DEFFPROCI
22010*LOAD CODE 900
22020P%=0:L%=50:U%=150:A%=0:a#="N":l=136
22030I#=STRING$(20," "):N#=I#:D#=I#:A#=I#:S#=I#:E#=I#
22040VDU23,224,0,0,0,63,127,63,0,0:@%=&90A
22050S=3:N%=450:M%=300:rgs=.1773333/2:gst=2
22060tok=0:V%=2E9:O%=0
22070*FX16,1
22080*FX190,12
22090*FX4
22100ON ERROR PRINTTAB(0,22);:REPORT:FORI=1TO3000:NEXT:RUN
22110*FX200,0
22120tst=5:S=10*64/tst
22130W3%=500:W2%=500:W1%=500
22200PROCm:ENDPROC
32000REM

```

SCALETRIX CAR SET UP



APPENDIX 5: BBC COMPUTER BIOFEEDBACK PROGRAM (CHAPTER FOUR)

LIST

```

10REM      Biofeedback
20REM      version 1.06
30REM      copyright (c) Nico Preston 11/87
35REM      for use with pulse meter connected to user port line 1.
40ONERRORGOTO270
50MODE1
60data_store%=&1000:false_rate%=&1900
65port%=&FE60:on%=254:off%=255
70VDU23;10,32;0;0;0;:REM cursor off
80IF FNchoice PROCpulse_monitor(TRUE) ELSE PROCfalse_feedback
90PROCsave_data(outfile%)
100END
110
120DEFFPROCdisplay(data%)
130LOCALy%,c%
140y%=500
160IFdata%=previous% ENDFPROC
170IFdata%<previous% c%=7
180IFdata%>previous% c%=5
190PLOTc%,data%*10,y%
200previous%=data%
210ENDPROC
220
230DEFFPROCdatain
240LOCALi%,file%,ch%
250CLS
260INPUT"Please enter input data filename:"file%
270SCLI("LOAD "+file%+" 1900")
280ENDPROC
290
300DEFFPROCaxes
310CLS
320LOCALi%,oy%,n#:oy%=350
330VDU19,1,6,0,0,0,19,3,5,0,0,0
340MOVE0,oy%:PLOT5,0,oy%+300
350MOVE0,oy%:PLOT5,1250,oy%
360VDU5
370FORi%=200TO1200STEP200
380MOVEi%,oy%:PLOT5,i%,oy%-15
390n%=STR$(i%/10):GCOL0,1
400MOVEi%-LEN(n%)*14,oy%-40:PRINTn%
410GCOL0,3
420NEXT
430GCOL0,1
440MOVE400,250:PRINT"Beats/minute"
450GCOL0,3:VDU4
460ENDPROC
470
480DEFFPROCpulse_monitor(mode%)
490PROCwait
500PROCset_up
510LOCALt1%,t2%,a%,minute%,tally%,count%,bpm%,instant_rate%
520t1%=0:t2%=0:bpm%=data_store%:instant_rate%=data_store%+10
530REPEAT:UNTIL?port%=on%:REM wait for 1st onset
540REPEAT:UNTIL?port%=off%:REM wait for 1st offset
550TIME=0:REM start timing
560count%=1:tally%=0:minute%=0:tf%=TIME
570
580REPEAT
590REPEAT:IFTIME-t2%>=6000 bpm%?minute%=count%:t2%=TIME:count%=0:minute%=minut
e%+1
600IFNOTmode% PROCfalse_present

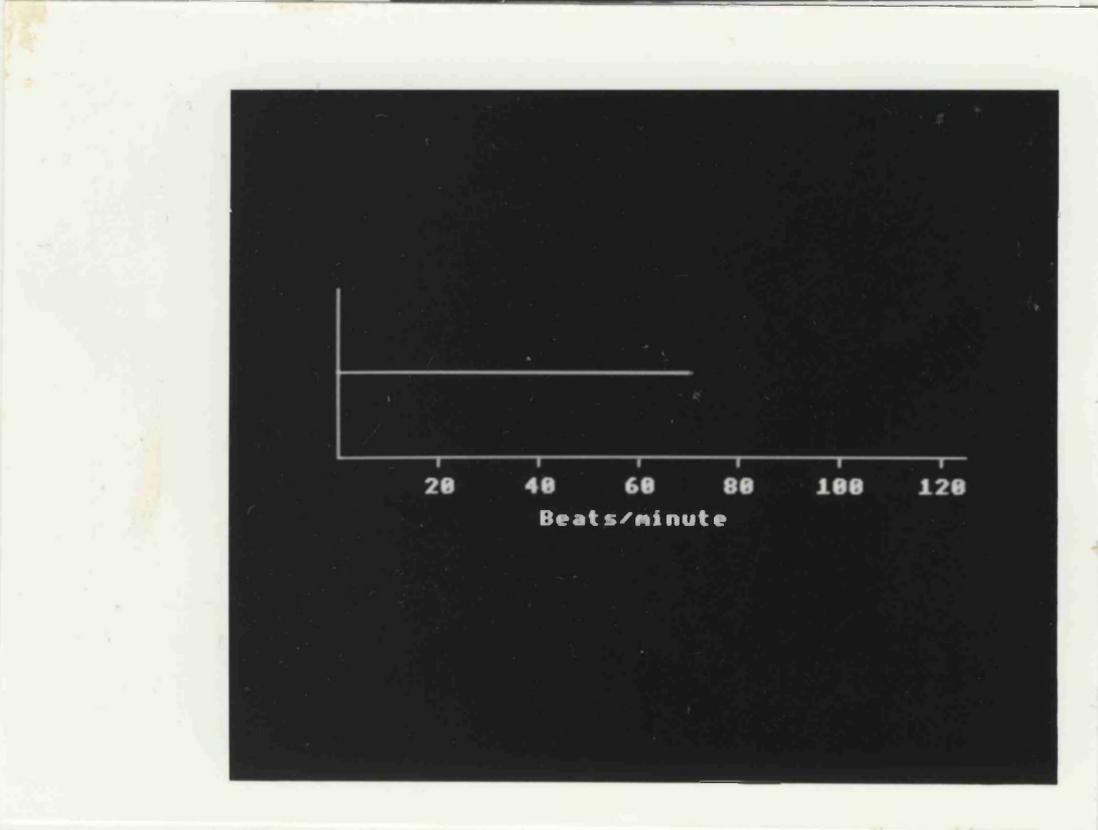
```

```

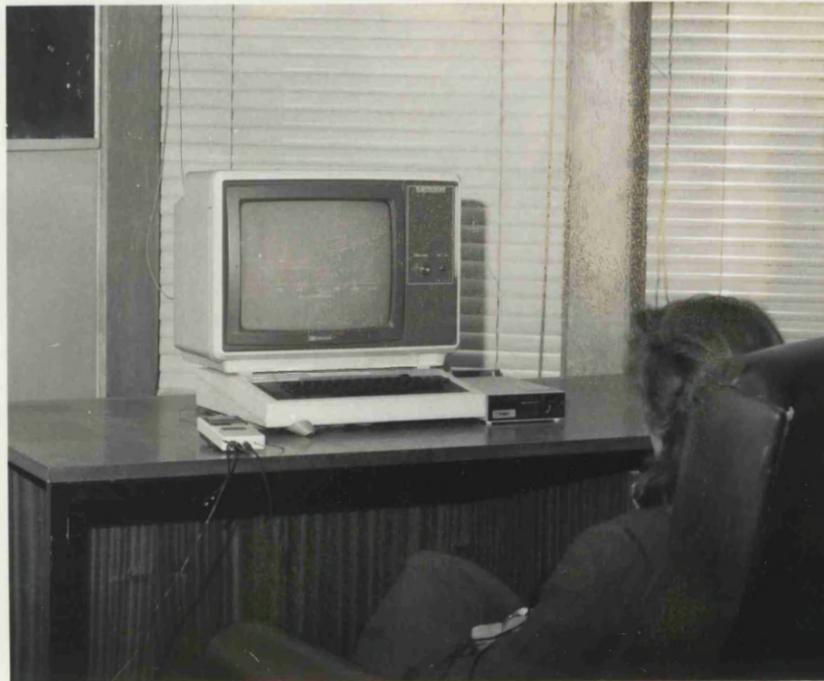
e%+1
600IFNOTmode% PROCfalse_present
610UNTIL?port%=on%:REM wait for onset
620count%=count%+1:tally%=tally%+1
630REPEAT:IFTIME-t2%>=6000 bpm%?minute%=count%:t2%=TIME:count%=1:minute%=minut
e%+1
640IFNOTmode% PROCfalse_present
650UNTIL?port%=off%:REM wait for offset
660ibi%=TIME-t1%:REM record ibi
670:t1%=TIME:REM start time
680instant_rate%?tally%=60/ibi%*100:REM calculate & record rate
690IFmode% PROCdisplay(instant_rate%?tally%)
700UNTILminute%=10
710instant_rate%?(tally%+1)=0
720ENDPROC
730
740DEFPROCset_up
750PROCaxes
760GCLO,2
770MOVE0,500
780previous%=0
790ENDPROC
800
810DEFPROCfalse_feedback
820PROCdatain
830ft%=0:fibi%=60/?false_rate%*100
840PROCpulse_monitor(FALSE)
850ENDPROC
860
870REM Error handler
880MODE7
890REPORT:PRINTERR,ERL:PRINT
900END
910
920DEFNchoice
930LOCALa#,a%
940CLS:PRINTTAB(0,5)"Which mode (T or F) ? "
950REPEAT:a#=GET#:a%=INSTR("TtFf",a%):UNTILa%
960CLS:PRINT "Please enter results filename:":REPEAT:INPUToutfile#:UNTILLEN(ou
tfile#)<9ANDLEN(outfile#)>0
970IFa%<2.=TRUE.ELSE=FALSE.
980
990DEFPROCsave_data(filename#)
1000QSCLI("SAVE r."+filename#+ " 1000 2000")
1010PRINT"Data saved OK"
1020ENDPROC
1030
1040DEFPROCfalse_present
1050IFTIME-tf%<fibi% ENDPROC
1060PROCdisplay(false_rate%?ft%)
1070ft%=ft%+1:fibi%=60/(false_rate%?ft%)*100:tf%=TIME
1080ENDPROC
1090
1100DEFPROCwait
1110CLS:PRINTTAB(3,10)"Press any space bar to start trial"
1120REPEAT:UNTILINKEY(-99)
1130ENDPROC

```

APPENDIX SIX: SET UP OF LABORATORY EQUIPMENT
(CHAPTER FOUR)



1. GREY DATA DAY 13, 17



Var
 Sou
 SUB
 TYP
 COM
 TYP
 Res
 SUB
 DAYS

CONDITIO	4	644.421	314.807	1.16	0.735
DAYS TYPE	3	514.884	171.561	1.16	0.736
DAYS COMP	3	70.148	23.382	0.16	0.824
TYPE.CONDITIO	3	302.134	67.278	0.46	0.713
COMP.CONDITIO	3	201.394	67.128	0.46	0.714
DAYS TYPE COMP	3	510.859	170.820	1.15	0.734
TYPE COMP CONDITIO	3	701.996	233.399	1.39	0.300
Residual	3	32.371	37.457	0.19	7.905
	72	10614.832	147.423		
SUBJECT MINUTES					
MINUTES					
Lin	9	429.413	47.498	8.22	<.001
Quad	1	393.124	393.124	68.92	<.001
Deviations	1	1.875	2.026	0.35	0.554
MINUTES TYPE	9	32.163	4.608	0.80	0.590
Lin TYPE	9	171.482	19.098	3.30	<.001
Quad TYPE	1	118.894	118.894	20.57	<.001
Deviations	1	9.317	9.317	1.63	0.201
MINUTES COMP	9	43.471	6.210	1.07	0.380
Lin COMP	9	26.938	2.893	0.52	0.861
Quad COMP	1	1.533	1.533	0.27	0.606
Deviations	1	1.891	1.891	0.30	0.480
MINUTES TYPE COMP	9	22.508	1.213	0.36	0.791
Lin TYPE COMP	9	67.770	7.530	1.38	0.236
Quad TYPE COMP	1	23.333	23.333	4.07	0.043
Deviations	1	4.882	4.882	1.19	0.278
	7	37.355	3.338	0.92	0.489

APPENDIX SEVEN: DETAILED ANOVA TABLE (CHAPTER
(FOUR)

```

1 OPEN 'DATA.DAT';2;IN
2
3 UNIT [1280]
4 READ [CH=2] HR

6 FACT [1280;32] SUBJECT;!(40(1...32))
7 FACT [1280;10] MINUTES;!(1...10)128)
8 FACT [1280;4;LAB=!T(CONTFB,NOFB,DOWNFB,UPFB)] CONDITION;!(10(1...4))32
9 FACT [1280;4;LAB=!T(ACOMP,ANOCOMP,BCOMP,BNOCOMP)] GROUP;!(320(1...4))
10 FACT [1280;2;LAB=!T(A,B)] TYPE;!(320(1,1,2,2))
11 FACT [1280;2;LAB=!T(COMP,NGCOMP)] COMP;!(320(1,2,1,2))
12 FACT [1280;8] DAYS;!(10(1...4)),(10(2,3,4,1)),(10(3,4,1,2)),(10(4,1,2
13
14 BLOC SUBJECT*DAYS*MINUTES
15
16 TREA TYPE*COMP*(DAYS+CONDITION)*POL(MINUTES;2)
17 ANOV[FPROB=Y] HR.

```

***** Analysis of variance *****

Variate: HR

Source of variation	d.f.	s.s.	m.s.	v.r.	F pr.
SUBJECT stratum					
TYPE	1	1.313	1.313	0.00	0.979
COMP	1	1346.851	1346.851	0.71	0.405
TYPE.COMP	1	2805.489	2805.489	1.49	0.233
Residual	28	52763.938	1884.426		
SUBJECT.DAYS stratum					
DAYS	3	644.421	214.807	1.46	0.233
CONDITIO	3	514.684	171.561	1.16	0.330
DAYS.TYPE	3	70.146	23.382	0.16	0.924
DAYS.COMP	3	202.134	67.378	0.46	0.713
TYPE.CONDITIO	3	201.384	67.128	0.46	0.714
COMP.CONDITIO	3	510.059	170.020	1.15	0.334
DAYS.TYPE.COMP	3	701.996	233.999	1.59	0.200
TYPE.COMP.CONDITIO	3	82.371	27.457	0.19	0.905
Residual	72	10614.632	147.425		
SUBJECT.MINUTES stratum					
MINUTES	9	427.413	47.490	8.22	<.001
Lin	1	393.126	393.126	68.02	<.001
Quad	1	2.026	2.026	0.35	0.554
Deviations	7	32.262	4.609	0.80	0.590
MINUTES.TYPE	9	171.832	19.098	3.30	<.001
Lin.TYPE	1	118.894	118.894	20.57	<.001
Quad.TYPE	1	9.517	9.517	1.65	0.201
Deviations	7	43.471	6.210	1.07	0.380
MINUTES.COMP	9	26.938	2.993	0.52	0.861
Lin.COMP	1	1.539	1.539	0.27	0.606
Quad.COMP	1	2.891	2.891	0.50	0.480
Deviations	7	22.508	3.215	0.56	0.791
MINUTES.TYPE.COMP	9	67.770	7.530	1.30	0.236
Lin.TYPE.COMP	1	23.532	23.532	4.07	0.045
Quad.TYPE.COMP	1	6.882	6.882	1.19	0.276
Deviations	7	37.355	5.336	0.92	0.489

Residual	252	1456.472	5.780		
SUBJECT.DAYS.MINUTES stratum					
DAYS.MINUTES	27	84.696	3.137	0.54	0.972
DAYS.Lin	3	31.567	10.522	1.33	0.141
DAYS.Quad	3	2.873	0.958	0.17	0.919
Deviations	21	50.257	2.393	0.42	0.991
MINUTES.CONDITIO	27	251.059	9.298	1.62	0.026
Lin.CONDITIO	3	141.076	47.025	8.17	<.001
Quad.CONDITIO	3	31.432	10.477	1.82	0.142
Deviations	21	78.551	3.741	0.65	0.882
DAYS.MINUTES.TYPE	27	140.877	5.218	0.91	0.603
DAYS.Lin.TYPE	3	13.769	4.590	0.80	0.496
DAYS.Quad.TYPE	3	49.735	16.578	2.88	0.035
Deviations	21	77.374	3.684	0.64	0.890
DAYS.MINUTES.COMP	27	188.796	6.992	1.21	0.210
DAYS.Lin.COMP	3	11.610	3.870	0.67	0.569
DAYS.Quad.COMP	3	88.216	29.405	5.11	0.002
Deviations	21	88.970	4.237	0.74	0.797
MINUTES.TYPE.CONDITIO	27	146.765	5.436	0.94	0.547
Lin.TYPE.CONDITIO	3	15.636	5.212	0.91	0.438
Quad.TYPE.CONDITIO	3	29.269	9.756	1.69	0.167
Deviations	21	101.860	4.850	0.84	0.667
MINUTES.COMP.CONDITIO	27	124.246	4.602	0.80	0.756
Lin.COMP.CONDITIO	3	12.120	4.040	0.70	0.551
Quad.COMP.CONDITIO	3	9.819	3.273	0.57	0.636
Deviations	21	102.307	4.872	0.85	0.662
Residual	702	4040.986	5.756		
Total	1279	77587.313			

APPENDIX 8: THE BORTNER ADJECTIVE
RATING SCALE

Below are 14 pairs of adjectives or phrases separated by a 7 point rating scale. Each pair represents two kinds of behaviour and each of us belongs somewhere along the line. For example, most of us are neither the most competitive person nor the least competitive person we know. Please indicate on the scales below where you think you belong .

1. Never late for appointments [] [] [] [] [] [] [] Casual
about
appointments

2. Not competitive [] [] [] [] [] [] [] Very competitive

3. Anticipates what others
are going to say (nods,
interrupts, finishes
for them) [] [] [] [] [] [] [] Good listener
hears others
out

4. Always rushed [] [] [] [] [] [] [] Never feels rushed
even under pressure

5. Can wait patiently [] [] [] [] [] [] [] Impatient
when waiting

6. Goes all out [] [] [] [] [] [] [] Casual

7. Takes things one
at a time [] [] [] [] [] [] [] Tries to do
many things
at once

8. Emphatic in speech
(may pound desk) [] [] [] [] [] [] [] Slow, deliberate
talker

9. Wants good job
recognised by others [] [] [] [] [] [] [] Only cares
about
satisfying
oneself

10. Fast (eating,
walking, etc) [] [] [] [] [] [] [] Slow doing things

11. Easy going [] [] [] [] [] [] [] Hard driving

12. Sits on feelings [] [] [] [] [] [] [] Expresses
feeling

10. How often do you put words in peoples mouths to speed things up ?

- a. Frequently b. Occasionally c. Almost never

11. If you tell your friend that you will meet him or her somewhere at a definite time, how often do you arrive late ?

- a. Once in a while b. Rarely c. I am never late

12. Do you find yourself hurrying to get places even when there is plenty of time ?

- a. Often b. Occasionally c. Rarely or never

13. Suppose you are to meet someone at a public place (street corner, building lobby) and the other person is already 10 minutes late. Will you

- a. Sit and wait b. Walk about while waiting
c. Usually carry some reading matter or writing paper so you can get something done while waiting

14. When you have to wait in line such as at the post office the cinema or a shop, do you

- a. accept it calmly b. Feel impatient but do not show it
c. Feel so impatient that someone watching could tell you were restless d. Refuse to wait in line

15. When you play games with much younger children how often do you let them win on purpose ?

- a. Most of the time b. Half of the time
c. Only occasionally d. Never

16. Do most people consider you to be

- a. Definitely hard-driving and competitive
b. Probably hard-driving and competitive
c. Probably more relaxed and easy-going
d. Definitely more relaxed and easy-going

17. Nowadays, do you consider yourself to be

- a. Definitely hard-driving and competitive
b. Probably hard-driving and competitive
c. Probably more relaxed and easy-going
d. Definitely more relaxed and easy-going

18. How would your closest friend rate you ?

- a. Definitely hard-driving and competitive
b. Probably hard-driving and competitive
c. Probably more relaxed and easy-going
d. Definitely more relaxed and easy-going

19. How would your best friend rate your general level of activity ?

- a. Too slow; should be more active
b. About average; is busy much of the time
c. Too active; needs too slow down

20. Would people who know you well agree that you take your work too seriously ?

- a. Definitely yes
- b. Probably yes
- c. Probably no
- d. Definitely no

21. Would people who know you well agree that you have less energy than most people ?

- a. Definitely yes
- b. Probably yes
- c. Probably no
- d. Definitely no

22. Would people who know you well agree that you get irritated easily ?

- a. Definitely yes
- b. Probably yes
- c. Probably no
- d. Definitely no

23. Would people who know you well agree that you tend to do most things in a hurry ?

- a. Definitely yes
- b. Probably yes
- c. Probably no
- d. Definitely no

24. Would people who know you well agree that you enjoy a contest (competition) and try hard to win ?

- a. Definitely yes
- b. Probably yes
- c. Probably no
- d. Definitely no

25. Would people who know you well agree that you get a lot of fun out of your life ?

- a. Definitely yes
- b. Probably yes
- c. Probably no
- d. Definitely no

26. How was your temper when you were much younger ?

- a. Fiery and hard to control
- b. Strong but controllable
- c. No problem
- d. I almost never got angry

27. How is your temper nowadays ?

- a. Fiery and hard to control
- b. Strong but controllable
- c. No problem
- d. I almost never get angry

28. When you are in the middle of studying and someone interrupts you, how do you usually feel inside ?

- a. I feel OK because I study better after an occasional break
- b. I feel a little bit annoyed
- c. I really feel irritated because most interruptions are unnecessary

29. How often are there deadlines in your studies ?

- a. Every day
- b. Every week
- c. Every month
- d. never

30. Do these deadlines usually

- a. Carry little pressure because they are only routine ?
- b. Carry a lot of pressure since delay would upset things

31. Do you ever set deadlines for yourself in studying or other things ?

- a. No b. Yes, but only occasionally c. Yes, Once a week or more

32. When you have to work against a deadline is the quality of work

- a. Better b. Worse c. The same

33. In school, do you ever start two projects at the same time and then shift back and forth from one to the other ?

- a. No, never b. Yes, but only in emergencies c. yes, regularly

34. Do you usually study regularly during holidays (Christmas or Easter) ?

- a. Yes b. No c. sometimes

35. How often do you study during weekends or late at night?

- a. Rarely or never b. Sometimes c. Regularly

36. When you find yourself getting tired while studying, do you usually

- a. Slow down for a while until your strength comes back
b. Keep pushing yourself at the same pace

37. When you are in a group do other people look to you for leadership ?

- a. Rarely b. About as often as they look to others
c. More often than they look to others

38. Do you write a list of things to be done to help you remember important things ?

- a. Never b. Occasionally c. Frequently

IN EACH OF THE FOLLOWING QUESTIONS, PLEASE COMPARE YOURSELF WITH AN AVERAGE STUDENT AT YOUR SCHOOL. PLEASE CIRCLE THE MOST ACCURATE DESCRIPTION.

39. In amount of effort put forth, I give

- a. Much more effort b. A little more effort
c. A little less effort d. Much less effort

40. In sense of responsibility I am

- a. Much more responsible b. A little more responsible
c. A little less responsible d. Much less responsible

41. I find it necessary to hurry

- a. Much more of the time b. A little more of the time
c. A little less of the time d. Much less of the time

APPENDIX TEN: THE BELIEFS AND FEARS QUESTIONNAIRE

Please indicate how true each of the statements below are for you. The statements are all about beliefs and feelings that people have. If you think a certain statement is "very true" for example make a circle around number 1. If you think it is "somewhat true" circle 3 and so on. There are no right or wrong answers. Your personal opinion is what we want. All your responses will be treated as confidential.

	Very true (1)	Fairly true (2)	Somewhat true (3)	A little true (4)	Not at all true (5)
1. People are measured by what they own in life.		1	2	3	4 5
2. Although my friends think I have a lot of confidence I really don't.		1	2	3	4 5
3. No one can tell you what is really good or bad.		1	2	3	4 5
4. I worry that good things won't last in the end.		1	2	3	4 5
5. It is better to get revenge when someone hurts you than to forgive and forget.		1	2	3	4 5
6. There aren't enough goodies for everyone the more you get the less I get.		1	2	3	4 5
7. I worry that I might not be smart enough to make it.		1	2	3	4 5
8. What is really good and valuable in a person's life is his hard work and success.		1	2	3	4 5
9. I worry about what my teachers think of me.		1	2	3	4 5

10. Doing what is good
doesn't always make
you happy. 1 2 3 4 5
11. I worry that real
justice doesn't exist-
it is not certain
that the good guys will win. 1 2 3 4 5
12. I believe that
criminals should
get what they deserve. 1 2 3 4 5
13. I don't think there are
enough goodies in life
for everyone. 1 2 3 4 5
14. I worry that I may not be
able to be really
successful. 1 2 3 4 5
15. A person's value or worth
depends on the things
he owns. 1 2 3 4 5
16. I worry about what
other people
think of me or say about me. 1 2 3 4 5
17. I believe that it
is important
what results you get rather
than how you get them. 1 2 3 4 5
18. I worry about the evidence
I see around me that nice
guys finish last. 1 2 3 4 5
19. I think if someone does
something wrong then he
should really pay for it. 1 2 3 4 5
20. I see a lot of my friends
as rivals or opponents for
the things in life that I
want. 1 2 3 4 5
21. I worry that I may not get
the things I want in life. 1 2 3 4 5
22. People should have more
respect for those who are
successful than those who
are not. 1 2 3 4 5

23. I worry about not making it. 1 2 3 4 5
24. I don't usually do what religion tells me to. 1 2 3 4 5
25. I worry about guilty people being found innocent and innocent people being found guilty. 1 2 3 4 5
26. When someone hurts me or uses me I will get even with him even if it takes a long time. 1 2 3 4 5
27. The only way you can make sure that you will get what you want is to do better than others. 1 2 3 4 5
28. I worry that it will take much too long for me to get the things I really want 1 2 3 4 5
29. I worry that I may not be as worthy as most people. 1 2 3 4 5
30. You have to achieve a lot to be a success in the eyes of others. 1 2 3 4 5
31. I worry that I am not talented enough to be successful at whatever I want to do. 1 2 3 4 5
32. When I try to do something the result is usually different from what I expect. 1 2 3 4 5
33. I believe that revenge is sweet. 1 2 3 4 5
34. My chances of being successful increase as others fail. 1 2 3 4 5
35. You can tell how worthy a person is by the number of things he has achieved (good grades, school prizes) 1 2 3 4 5
36. It is important to me to make other people like me. 1 2 3 4 5

- | | | | | | |
|--|---|---|---|---|---|
| 37. There aren't enough goodies to go around so you end up with winners and losers, rich people and poor people. | 1 | 2 | 3 | 4 | 5 |
| 38. I worry that I may not be as good as most people. | 1 | 2 | 3 | 4 | 5 |
| 39. When others do well in the class I am likely to be judged as less able than them. | 1 | 2 | 3 | 4 | 5 |
| 40. I believe that your gain is my loss. | 1 | 2 | 3 | 4 | 5 |
-
-

Please provide the following information :

Name:

Age:

Sex: male female.

BURKE'S (1983) BELIEFS AND FEARS QUESTIONNAIRE:

The next few pages list various values, beliefs and feelings that might be held by individuals in our society. We are interested in how accurately each of these statements reflects or describes your own values, beliefs and feelings. Please indicate how accurate or true each statement is of your values and beliefs by circling the number on the right that comes closest to representing your own values and beliefs. There are no right answers. We are interested in the extent to which these statements describe you.

Completely accurate or true (1)	Fairly accurate or true (2)	Somewhat accurate or true (3)	A little accurate or true (4)	Not at all accurate or true (5)
--	--------------------------------------	--	--	--

1. A person who has not achieved economic prosperity is often considered a failure as a human being.
2. Although my friends think I may have a lot of self esteem and self confidence, I really don't.
3. There are no universal moral principles that guide the actions of people.
4. I often worry that good may not prevail in the end.
5. It is better to forgive and forget when someone hurts you than to seek revenge.
6. I believe that your gain is my loss.
7. I often worry that I might not be smart enough to make it.
8. Success is best defined in terms of material or tangible achievements and related status and recognition.
9. My sense of self esteem seems to go up and down.
10. Living one's life by the golden rule is no guarantee that you'll be happy.
11. I sometimes worry that an impartial justice does not exist in the world- there's no one guaranteeing that the "good guys" will win.
12. I generally believe that the punishment should fit the crime.
13. There aren't enough "goodies" in this world for everyone the more you get, the less I get.

14. I often worry that I might not be smart enough to be really successful.
15. People are measured by what they achieve and how well off they are economically.
16. I often worry about not being a success or as successful as I would like.
17. I believe that the ends justify the means.
18. I sometimes worry that my own good intentions and actions may produce negative consequences.
19. I believe in the old adage "an eye for an eye, a tooth for a tooth".
20. I don't think there are enough "goodies" in life to meet everyone's needs.
21. I often worry that I won't have the ability to accomplish what I want to achieve.
22. There is little inherently valuable or good about a human life outside of effort and successful accomplishments.
23. I sometimes worry about not making it.
24. Individuals usually make choices on what is best for them based on what is easiest rather than on what is right to do.
25. I worry about how often my own actions and behaviour is guided by expediency rather than standing up for what I believe is right.
26. When someone hurts me or uses me I will get even with him even though it may take a long time.
27. I see a lot of my peers as rivals for those things in life that I want.
28. I often worry that I won't get the recognition and appreciation that I need or would like.
29. An individual's worth is more a function of the things he has than any other factor.
30. I sometimes worry that I may not be as worthy as most people.
31. My own actions are rarely guided by spiritual or religious values.
32. I often worry about the amount of evidence I see around me that "nice guys finish last".

33. I tend to believe in the old adage "revenge s sweet".
34. The only way one can guarantee recognition and rewards is to do better that your peers.
35. I often worry that there isn't enough time for me to accomplish what I would like to do.
36. An individuals possessions are a good indication of his worth and value in our society.
37. I believe that the road to hell is often paved with good intentions.
38. I often worry that others will not find me worthy of their esteem and liking.
39. My chances of being successful increase as others fail.
40. An individual's self worth or self esteem is largely a function of material success.
41. I sometimes fear that I may not be as good as most people.
42. My own actions tend to be more strongly influenced by material than by spiritual values.
43. There aren't enough good things in life to go around- so you end up with have's and have not's, winners and losers.
44. It is important that one achieve a lot to be a success in the eyes of others.
45. I don't see a close link between what my actions are intended to accomplish and what actually happens.
46. I frequently worry that I am not talented enough to be really successful at what I want to do.
47. An individual's self worth or self esteem is largely a function of the number and quality of his achievements.
48. When others do well, I am likely to be judged as less adequate by comparison.
49. An individual's self worth and self esteem is largely a function of his status and prestige.
50. It is very important to me that I be liked by other people.

APPENDIX ELEVEN: THE STRESS AND AROUSAL CHECK LIST

Please put a circle around the most appropriate answer. Indicate for each item how you feel AT THE MOMENT.

(vv) v ? no definitely feel
 vv (v) ? no feel slightly
 vv v (?) no uncertain
 vv v ? (no) definitely do not feel

tense	vv	v	?	no	apprehensive	vv	v	?	no
alert	vv	v	?	no	idle	vv	v	?	no
cheerful	vv	v	?	no	active	vv	v	?	no
dejected	vv	v	?	no	relaxed	vv	v	?	no
comfortable	vv	v	?	no	energetic	vv	v	?	no
drowsy	vv	v	?	no	stimulated	vv	v	?	no
calm	vv	v	?	no	fearful	vv	v	?	no
activated	vv	v	?	no	contented	vv	v	?	no
nervous	vv	v	?	no	sleepy	vv	v	?	no
restful	vv	v	?	no	worried	vv	v	?	no
sluggish	vv	v	?	no	up-tight	vv	v	?	no
passive	vv	v	?	no	pleasant	vv	v	?	no
jittery	vv	v	?	no	aroused	vv	v	?	no
bothered	vv	v	?	no	somnolent	vv	v	?	no
tired	vv	v	?	no	uneasy	vv	v	?	no
lively	vv	v	?	no	distressed	vv	v	?	no
vigorous	vv	v	?	no	peaceful	vv	v	?	no

APPENDIX THIRTEEN: BBC COMPUTER BIOFEEDBACK PROGRAM (CHAPTER SIX)

LIST

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10REM Pulse Measuring Program
15REM Analyse tape via tone decoder.
20REM Version 1.02
30REM copyright (c) N H Preston (UCL) 6/89
35
40MUSIC
50DIMrate(3,50),c%(3)
60IX=6000:REM measuring interval
70session%=0
80PROCfirststart
90REPEAT
100PROCstart
110PROCfirstpulse
120REPEAT
130REPEAT:T2%=TIME:IF T2%-T1%>=IX rate(session%,c%)=B%/(T2%-T1%)*6000:PRINT"rate: ";rate(session%,c%):B%=0:T1%=TIME:c%=c%+1
140IF T2%-T1%>=200 PRINT"End of session.":end=TRUE
150UNTIL?%FE60=255 OR end:REM rising edge
160PRINT" ";A%;" ";TIME-T%
170T%=TIME
180A%=A%+1:B%=B%+1
190REPEAT:UNTIL?%FE60=254:REM falling edge
200UNTILend
205c%(session%)=c%
210session%=session%+1
220UNTILsession%=4
225PROCend
226END
230
240DEFPROCfirststart
250*MOTOR1
260PRINT TAB(3)"Switch on equipment and REWIND tape."
270PRINT TAB(12)"Press space bar when ready.":REPEAT:UNTILGET$=" "
275*MOTOR0
280CLS:PRINT TAB(6)"Press PLAY on tape player."
290PRINT TAB(12)"Press space bar when ready.":REPEAT:UNTILGET$=" "
291PRINT TAB(7)"OK."
300ENDPROC
310
320DEFPROCstart
330REM lose first three pulses
340*MOTOR1
350T%=0:T1%=0:A%=0:B%=0:c%=0:end=FALSE
360J%=0:REPEAT
370REPEAT:UNTIL?%FE60=255:REM rising edge
380IF J%=0 CLS:PRINT"Waiting for stable signal.": ELSE PRINT".":
390REPEAT:UNTIL?%FE60=254:REM FALLING EDGE
400J%=J%+1:UNTILJ%=3
410CLS:PRINT"Starting." beat no. i.b.i."
420ENDPROC
430
440DEFPROCfirstpulse
450REPEAT:UNTIL?%FE60=255:REM rise
460TIME=0
470REPEAT:UNTIL?%FE60=254:REM fall
480ENDPROC
490
500DEFPROCend
510*MOTOR0
520PRINT"End of input."
530PRINT" Press space bar to list results.":REPEAT:UNTILGET$=" "
535PRINT" Pulse rate (beats/min) averaged over 1 minute periods."
540FORs%=0TO3:PRINT"session: ";s%+1
550FORj%=0TO3:s%+1:PRINTTAB(12);INT(rate(s%,j%)*100+.5)/100:NEXT
55 IFs%>0 PRINT"Press space bar for more.":REPEAT:UNTILGET$=" "
55NEXT
560ENDPROC

```