Effects of Aerobic Exercise on Type A Physiological Hyperreactivity

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Abstract

EFFECTS OF AEROBIC EXERCISE ON TYPE A PHYSIOLOGICAL HYPERREACTIVITY

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Recent studies have suggested that Type A individuals may be more susceptible to coronary heart disease because of their exagerrated physiological reactivity to challenge. The present study evaluated the efficacy of aerobic exercise, which improves cardiorespiratory fitness, in modifying physiological hyperreactivity to mental stress. Twenty-six healthy, male, Type A hyperreactors were randomly assigned to aerobic exercise or stress management programs which met for 1 hour, 3 times weekly, for 6 weeks. Pre and post test sessions, in a controlled laboratory setting, assessed: cardiorespiratory fitness by the estimated VO2 max level derived from the mean heart rate obtained during the last two minutes of a submaximal bicycle ergometer test; and physiological reactivity by measures of heart rate, blood pressure, epinephrine and norepinephrine sampled prior to, during, and following termination of two counterbalanced mental stress tasks. After training, Group Exercise subjects demonstrated a significant improvement in cardiorespiratory fitness, and

a significant decline in heart rate reactivity during recovery from mental stress, whereas, the non-exercising control group remained unchanged. These findings suggest that aerobic exercise has potential as an intervention technique in modifying physiological hyperreactivity manifested by Type A individuals, which may lead to a reduced risk for coronary heart disease.

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The Type A behavior pattern, which encompasses such characteristics as excessive achievement striving, impatience, time urgency and aggressiveness, has recently been established as an independent risk factor for coronary heart disease (CHD) (Review Panel on Coronary-Prone Behavior and Coronary Heart Disease, 1981). An increasing body of evidence suggests that the excessive sympathetic reactivity to challenge displayed by Type A individuals may contribute to this increased risk. Type As, when compared to their more relaxed Type B counterparts, have been found to exhibit a greater rise of heart rate, systolic and/or diastolic blood pressure, and catecholamine secretion during exposure to laboratory stressors (Dembroski, MacDougall, & Shields, 1977; Dembroski, MacDougall, Shields, Pettito, & Lushene, 1978; Pittner & Houston, 1980). Although the exact physiological mechanisms mediating CHD are presently a matter of speculation, results from animal studies suggest that sympathetic reactivity may be implicated (Herd, 1978; Schneiderman, 1978). However, given what is known, it is possible to explore intervention strategies that may influence sympathetic reactivity. One possibility is aerobic exercise, which over time produces physiological changes that improve cardiorespiratory fitness (Clausen, 1977). These include the following: a reduction in heart

rate and blood pressure at rest and during exercise (Clausen, 1977); and an increased oxygen transport capacity of the circulatory system (Clausen, 1977). Studies have shown that these changes also modify sympathetic reactivity to both physical and psychosocial stress (Clausen, 1977; Cox, Evans, & Jamieson, 1979; Keller, 1981). If it can be shown that the physiological changes produced by aerobic exercise also modify sympathetic reactivity in Type A individuals, this may have important implications for future prospective research.

Type A Behavior Pattern

Origin of concept. An association between distinguishing behavioral characteristics and CHD has been noted in the literature for nearly one hundred years. Osler (1892) first described the coronary patient as a "keen and ambitious man, the indicator of whose engines are set at full speed ahead". Other researchers added attributes of aggressive tendencies, compulsive striving, and hard-driving, goal-directed behavior to this description (Arlow, 1945; Dunbar, 1943; Gildea, 1949; Menninger & Menninger, 1936). However, it was not until the 1950s that two cardiologists, Meyer Friedman and Ray Rosenman, combined such attributes to form a behavioral concept (Friedman & Rosenman, 1974). Concerned that

traditional heart disease risk factors (e.g., smoking, diet, exercise habits) could account for only half of the incidence of CHD, Friedman and Rosenman began to systematically observe their patients (Friedman & Rosenman, 1974). They found that most of their coronary patients under the age of 60-65 years exhibited a certain constellation of behavioral characteristics now known as the Type A behavior pattern (TABP) (Friedman, 1978).

The TABP is defined as "an action-emotion complex that can be observed in any person who is aggressively involved in a chronic, incessant struggle to achieve more and more in less and less time, and if required to do so, against the opposing efforts of other things or other persons" (Friedman & Rosenman, 1974). The major facets of TABP are a chronic sense of time urgency, a striving for greater achievement in both vocational and avocational endeavours, and an enhanced aggressiveness which at times can evolve into competitive hostility (Jenkins, 1979). Overt characteristics of these facets include: explosive, loud, terse, accelerated speech patterns; a hard-driving life style; a tendency to be self-centered; polyphasic thoughts and actions; an impatience with slow-moving events; a general appearance of hyperalertness and restlessness; an obsession to continually prove self-worth by achieving the maximum number of goals in a minimal amount of time;

and a compulsive attraction to competition and challenge, even in avocational pursuits (Friedman & Rosenman, 1974). An intense exhibition of most of these characteristics is called Type A-1 behavior, whereas, a less overt, less exaggerated display is called Type A-2 behavior (Rosenman, 1978). A relative absence of Type A characteristics is called Type B behavior. This is not a unique pattern, rather, it is a lack of the extreme, intense type of responding seen in the Type A. This is characterized by a more relaxed, easy-going life style with few signs of time urgency, competitiveness, impatience and aggressive drive (Rosenman, 1978). An equal distribution of Type A and Type B characteristics is called Type X behavior. This category exists to accomodate those individuals who cannot easily be distinguished as true Type As or Type Bs (Rosenman, 1978).

Finally, it must be emphasized that TABP is not considered to be a trait, stress situation, or distressed response. Rather , it is a style of overt behavior elicited in susceptible people by situations in their environment that they perceive as challenging (Rosenman, 1978).

Assessment of Type A Behavior Pattern. The two most common measures used to assess TABP are the Structured Interview (SI) (Rosenman, Friedman, Straus, Wurm,

Kositchek, Hahn & Werthessen, 1964), and the Jenkins Activity Survey (JAS) ((Jenkins, Rosenman, & Friedman, 1967).

The SI is a challenge situation consisting of approximately 22 quesions which are verbally administered and subsequently evaluated by trained personnel. Several questions are asked for the specific content of their answers (e.g. "How do you feel about waiting in lines, bank lines, supermarket lines? When you play games with people your own age, do you play for the fun of it, or are you really in there to win?"). Others are designed, by their manner of presentation, to elicit the characteristics of TABP. For example, "Most people who work have to get up fairly early in the morning - in your particular case, uh-what-time-uh-do-you-uh, ordinarily uh-uh-to-uh-uh-get-up?" A typical Type A will interrupt and answer prior to the completion of this long, drawn-out question. Other techniques used during the interview are: interrupting while the subject is answering; giving a series of questions rapidly; and challenging the accuracy of a response (Rosenman, 1978). During the interview, several nonverbal, overt behavioral characteristics are also noted. These include: voice quality, speech hurrying, attitude, hyperalertness, gestures and movements, facial expressions, and sighing (Rosenman,

1978). Although the content of the responses is considered, assessment of TABP is primarily based upon the foregoing specific psychomotor mannerisms observed during the interview (Rosenman, 1978).

The JAS is a self-administered, computer-scored questionnaire of approximately 50 multiple-choice questions, similar to those used in the SI. It was developed to provide a convenient and inexpensive psychometric method of assessing TABP in large-scale studies (Jenkins et al., 1967). The JAS scale is standardized to have a mean of 0.0 and a standard deviation of 10.0. Positive scores indicate TABP, whereas negative scores indicate Type B behavior (Jenkins et al., 1967).

Both measures appear to be reliable. For example, the interview method was found to have a test-retest reliability of 80% over a period of 12 to 20 months (Rosenman, 1978). Similarly, interrater agreement on several occasions has ranged from 76 to 88% (Rosenman, 1978). Test-retest correlations for the JAS ranged from .60 to .70 over a four year interval, and in one study, correlations between raters were approximately .70 (Jenkins, 1978).

The main criticism of both measures has been their subjective nature (Rosenman, 1978). Type A individuals

are often not aware of their style of behavior, which leads to inaccurate responses. However, the SI partially controls for this subjectivity by basing assessment mainly on observed interview behavior, which is more difficult to disguise than self-report (Rosenman, 1978).

To summarize, the SI and JAS are currently the two most utilized methods for assessing TABP. Although both methods are reliable, they are also, by virtue of their self-report format, both vulnerable to deception. However, because the SI takes into account hard to disguise psychomotor characteristics, it is felt to be the the more valid measure.

Type A Behavior and Coronary Heart Disease

Epidemiological evidence. Numerous studies have verified that the TABP is an independent risk factor for CHD (see Cooper, Detre, & Weiss, 1981; Dembroski, Weiss, Shields, Haynes, & Feinleib, 1978 for reviews). The most significant support for this association comes from the Western Collaborative Group Study (WCGS) conducted by Rosenman et al. (1964). In this prospective study, 3,154 initially well, middle-aged American men were classified as Type A or B at intake, and then were followed for the development of CHD over a 8.5 year period. Results showed that Type As had an estimated risk of developing CHD 2.37 times that of Type Bs (Rosenman, Brand, Jenkins, Friedman,

Straus, & Wurm, 1975). Even more important was the finding that when the major traditional risk factors of age, systolic blood pressure, cholesterol level, and smoking were statistically controlled, the independent Type A risk factor of CHD only decreased to 1.97 (Rosenman, Brand, Sholtz, & Friedman, 1976). Further analyses, among men surviving their first coronary episode, showed an association between TABP and recurrence of myocardial infarction. The JAS score was found to be a stronger predictor of reinfarction than either smoking or serum cholesterol level (Jenkins, Zyzanski, & Rosenman, 1976).

Framingham, another large-scale prospective study, has recently confirmed these findings (Haynes, Feinleib, & Kannel, 1980). In both men and women, TABP was found to be associated with a twofold risk of CHD. Again, this association was independent of other standard coronary risk factors.

Relation to Coronary Atherosclerosis. The association between TABP and CHD has been further strengthened by evidence suggesting that the behavior pattern contributes to the increased development of atherosclerosis. Postmortem findings from the WCGS revealed that Type A subjects, regardless of cause of death, exhibited significantly greater degrees of atherosclerosis than did

Type B (Friedman, Rosenman, Straus, Wurm, & Kositchek, 1968). In addition, coronary angiography studies have also demonstrated a strong relation between TABP and extent of atherosclerosis in the coronary arteries (Blumenthal, Williams, Kong, Thompson, Jenkins, & Rosenman, 1978; Zyzanski, Jenkins, Ryan, Flessas, & Everist, 1976), and a faster progression in the development of atherosclerotic plaques (Krantz, Sanmarco, Selvester, & Matthews, 1979). Although these correlational studies are subject to selection bias, and one recent failure to replicate has appeared (Dimsdale, Hackett, Hutter, & Block, 1980), there still remains some circumstantial evidence that TABP influences the atherosclerotic process.

Prevalence rate. The prevalence rate of TABP, in the general population, is as yet unknown. The WCGS, which was comprised of middle class, employed males over age 39 years, classified 50% of its sample as exhibiting TABP (Rosenman, et al., 1964). Other studies using subpopulations have found: 1)a higher prevalence in white than in blue-collar workers (Howard, Cunningham, & Rechnitzer, 1977); 2)no difference in prevalence between middle aged men and women when equated for education and race, but a higher prevalence in women working full-time compared to women working part-time or to housewives

(Waldron, Zyzanski, Shekelle, Jenkins, & Tannebaum, 1977); 3) a higher prevalence in mainland U.S. than in Europe or Hawaii (Zyzanski, 1978); 4) a positive correlation between prevalence and occupational status (Zyzanski, 1978); and 5) a negative correlation between prevalence and younger adults aged 20-25 years (Zyzanski, 1978). Thus, at present, the highest concentration of TABP can be found in middle-aged Americans working in white-collar occupations.

In summary, TABP is now recognized as a widespread, independent risk factor for CHD in an important subpopulation of our society. Recently, the National Heart, Lung, and Blood Institute organized a distinguished review panel to evaluate the accumulating evidence on the association between TABP and CHD. Their conclusion was: "The review panel accepts the available body of scientific evidence as demonstrating that type A behavior...is associated with an increased risk of clinically apparent CHD in employed, middle-aged U.S. citizens. This risk is greater than that imposed by age, elevated values of systolic blood pressure and serum cholesterol, and smoking and appears to be of the same order of magnitude as the relative risk associated with the latter three of these other factors." (Review Panel on Coronary-Prone Behavior and Coronary Heart Disease, 1981).

Type A Behavior and Physiological Reactivity

Physiological studies. In response to biochemical and environmental challenge, Type As display enhanced reactivity on a number of physiological dimensions suspected of playing a role in the pathogenesis of CHD. Initial investigations found differences in biochemical and neuroendocrine functioning between middle-aged, male Type A-1 (extreme Type A) and Type B subjects. In contrast to their Type B counterparts, Type A-1 subjects exhibited: 1)higher levels of norepinephrine during working hours, and in response to a competitive contest (Friedman, Byers, Diamant, & Rosenman, 1975; Friedman, St. George, & Byers, 1960); 2)accelerated blood coagulation (Friedman & Rosenman, 1959); 3)chronic elevation of plasma cholesterol (Friedman, Byers, Rosenman, & Elevitch, 1970; Friedman & Rosenman, 1959); 4)increased serum triglycerides before and after fat ingestion, with pronounced sludging of erythocytes during the postprandial cycle (Friedman, Rosenman, & Byers, 1964); 5)greater insulenemia response to glucose challenge (Friedman et al., 1970); 6)higher serum levels of corticotropin (Friedman, Byers, & Rosenman, 1972); and 7)decreased growth hormone response to arginine (Friedman, Byers, Rosenman, & Nueman, 1971).

Investigations using contrived laboratory challenges

have also reported differences in physiological functioning between the behavior types. Although no A-B differences were observed during resting baseline periods, when confronted by appropriately challenging or stressful tasks, Type As typically exhibited significantly greater increases in plasma epinephrine or norepinephrine, heart rate, and blood pressure than their Type B counterparts (Dembroski et al, 1977; Dembroski et al, 1978; Dembroski, MacDougall, Herd, & Shields, 1979; Friedman et al., 1975; Glass, Krakoff, Contrada, Hilton, Kehoe, Mannucci, Collins, Snow, & Elting, 1980; Manuck, Craft, & Gold, 1978; Pittner & Houston, 1980; Van Egeren, 1979).

The use of controlled laboratory tasks, in this type of study, has provided insight into the circumstances which evoke the differential responding observed in Type As. These events can be described as frustrating (Glass et al., 1980), difficult (Dembroski et al., 1978; Dembroski et al., 1979; Friedman et al., 1975), moderately competitive (Dembroski et al., 1979; Glass et al., 1980), or requiring patience (Lundberg & Forsman, 1979). High incentive or highly competitive situations (Manuck & Garland, 1979; Glass et al., 1980) did not yield A-B differences. Under these conditions the Type Bs were found to be as responsive as the Type As. It is also noteworthy that two recent studies found TABP to be

significantly correlated with greater blood pressure responses in anethesized patients undergoing coronary bypass surgery (Kahn, Kornfeld, Frank, Heller, & Hoar, 1980; Krantz, Arabian, Davia, & Parker, 1982). These results suggest that the overt behavioral characteristics observed in Type As may predict rather than elicit a heightened physiological responsiveness.

A few laboratory studies have failed to find any difference in physiological reactvity between the behavior types. In a choice-reaction time task, during which each subject maintained control over the pace, no A-B differences were found in urinary catecholamines (Frankenhauser, Lundberg, & Forsman, 1980). Lovallo and Pishkin (1980) found no A-B differences in heart rate and blood pressure following exposure to failure or uncontrollable noise. Scherwitz, Berton and Leventhal (1978) reported no A-B differences in heart rate and blood pressure reactivity across a battery of tasks until subjects were subdivided according to the number of self-references (I, me, my, mine) they made during the Structured Interview. Then, the highest levels of systolic blood pressure during baseline and task performance were found to occur in Type A individuals who made many self-references. These results suggest that Type As are not uniformly more physiologically responsive

than Type Bs, which might explain the fact why only some Type A individuals develop CHD. They also underline the importance of continued research into the environmental variables which evoke A-B differences.

Nevertheless, there is a considerable body of research that offers some support for the idea that Type A and B subjects, under specific sets of environmental circumstances, differ in physiological arousal. When challenged, Type A individuals display excessive and sustained levels of defensive physiological arousal inappropriate to the demands of the situation. At present, it is not clear whether the enhanced aggressive, impatient, hard-driving characteristics of the TABP result in a lower threshold for perceiving challenge, or whether the TABP reflects an underlying excessive sympathetic response to environmental stress (Review Panel on Coronary Prone Behavior and Coronary Heart Disease, 1981).

Defensive physiological arousal. Defensive physiological arousal is an adaptive response, which prepares the body for vigorous muscular activity in order to stand and fight, or to run from physical danger (Cannon, 1953). The ultimate source of energy for muscular activity comes from the oxidation of one or more of the foodstuffs (e.g. carbohydrates, fats, and proteins (Guyton, 1981). Thus, a threatening situation creates an

increased demand by the muscles for oxygen and fuel to provide energy for anticipated strenuous activity.

The purpose of defensive physiological arousal is to mobilize the body's energy resources for massive and quick motor activity (Eliot, 1976). Initially, there is a "mass discharge" of the sympathetic nervous system throughout the body. This is a simultaneous release of the neurotransmitter norephinephrine from sympathetic nerve endings that terminate on various organs (Guyton, 1981). The overall effect of "mass action" is a rapid change in end-organ activity, which results in alterations in metabolic processes and cardiovascular functioning (Guyton, 1981). Oxygen levels are increased by more rapid, and deeper breathing and by dilation of the bronchial tubes. Fuel levels are also elevated by increases in the release of glucose from the liver, blood glucose concentration, and glycogenolysis in muscle. To speed up the conveyance of these resources, the circulation of the blood is increased by an acceleration of heart rate, and a rise in cardiac output and blood pressure. Further, the blood supply is redistributed from less vital areas, such as the gastrointestinal tract, to the active muscles, myocardium and brain (Guyton, 1981).

Although the effects of sympathetic neural activation on end-organ activity are immediate, they are not long-

term. This is because the sympathetic nerves have a limited ability to constantly release norepinephrine (Guyton, 1981). Therefore, in order to prolong defensive arousal, the sympathetic nervous system also stimulates the adrenal medulla to release epinephrine and norepinephrine into the bloodstream. The effects of these two catecholamines are functionally identical to direct sympathetic innervation, except for a 20 to 30 sec delay in onset, and a tenfold increase in duration (Guyton, 1981). The sum of these effects allows a person to perform vigorous physical activity for extended periods of time.

Potential pathophysiological processes. Research has generated abundant evidence that defensive arousal can also be triggered by psychosocial situations that are perceived as threatening but do not require vigorous muscular activity (Froberg, Karlsson, Levi, & Lidberg, 1971; Levi, 1972; Mason, 1968). As a result, the mobilized resources intended to support physical exertion are not utilized, which in turn prolongs the sympathoadrenomedullary mediated changes in blood chemistry and cardiovascular activity (Eliot, 1976). The question then arises whether these unresolved, prolonged changes contribute to the development of CHD.

Thus far, there is only indirect evidence to support such a hypothesis. Animal studies have demonstrated that

dominant animals allowed to actively cope with predictable aversive emotional or physical stressors respond with sustained sympathetic arousal (Schneiderman, 1978). Such stimulation, if prolonged, was found to produce various cardiovascular pathologies. These include; elevated serum cholesterol and decreased clotting time (Friedman & Rosenman, 1959), increased arterial pressure (Herd, Morse, Kelleher, & Jones, 1969), myofibrilar degeneration (Corley, Mauck, & Shiel, 1975), and atherosclerosis (Henry, Ely, Stephens, Ratcliffe, Santisteban, & Shapiro, 1971).

Type A individuals, when actively coping with challenges in their environment, also have been found to respond with sustained sympathetic arousal. If one assumes that over time these individuals would be prone to develop CHD, it becomes necessary to try to identify the pathways whereby such arousal might lead to pathophysiological processes. One possibility is the sympathetic catecholamines, which have been associated with various cardiovascular pathologies. For example, catecholamines have been demonstrated to cause anatomical damage to the heart. Myocardial lesions have been produced in animals by the administration of exogenous sympathetic catecholamines, with the severity and extent of the lesion varying directly with the dose and rate of

administration (Haft, 1974). Similar myocardial lesions were found in patients dying after prolonged infusion of norepinephrine for shock, or of pheochromocytoma (an adrenal medulla tumour which secretes large amounts of epinephrine and norephinephrine) (Haft, 1974).

Catecholamines may also play an indirect role in the development of atherosclerosis. This disease has been found to be associated with sustained elevations of blood cholesterol (Rosenman et al., 1964). Catecholamine release promotes the formation of lipids (fatty acids), which are subsequently transported to active cells where they are oxidized to give energy (Guyton, 1981). Surplus circulating lipids, not utilized in the production of energy, are eventually taken up by the liver for degradation (Guyton, 1981). One product of this breakdown is cholesterol-rich low density lipoproteins (Guyton, 1981). Although cholesterol is an essential ingredient to every organ of the body, it also infiltrates arterial lesions to form atherosclerotic plaques (Guyton, 1981). Hypercholesterolemia has been found to enhance the severity of these lesions (Guyton, 1981). A chronic lack of utilization of mobilized lipids might eventually lead to increased cholesterol deposits in the walls of arteries.

Although a strong relation between TABP and severity

of atherosclerosis has been found, there is no direct evidence of the possible mechanisms which might mediate such an association. However, there is considerable circumstantial evidence that a number of sympathetically influenced reactivity differences, potentially related to increased development of atherosclerotic plaques, exist between Type A and Type B individuals. These include the following: elevated levels of free fatty acids, serum triglycerides, and plasma cholesterol during normal working hours (Friedman & Rosenman, 1959; Friedman et al., 1970); and enhanced rates of blood coagulation (Friedman & Rosenman, 1959), aggregation of blood platelets (Simpson, Olewine, Jenkins, Ramsey, Zyzanski, Thomas, & Hams, 1974), and sludging of erythrocytes (Friedman et al., 1964).

Intervention

The Review Panel on Coronary-Prone Behavior and Coronary Heart Disease (1981) has stated that "the goal of intervention should be a change in level (intensity) of the type A pattern or a specific subset of its components and/or changes in level of relevant physiologic mediators". The prospect then arises that an intervention which modifies the physiological hyperreactivity manifested by Type As might further our knowledge of the behavior-physiology relationship, and thus ultimately contribute to the prevention of CHD. One potential

intervention is aerobic exercise, which over time produces physiological changes that improve cardiorespiratory fitness.

Exercise prescription. The American College of Sports Medicine (1975) has suggested that the prescription of exercise follow certain basic guidelines. These include; type of exercise, intensity, duration, and frequency.

Cardiorespiratory fitness is enhanced and maintained by exercises which: 1) involve large muscle groups in low tension; 2) are high repetition contractions of a rythmic nature; and 3) employ the aerobic pathway as the primary source of energy (American College of Sports Medicine, 1975). Aerobic exercises such as walking, jogging, and cycling all fulfill these criteria.

The intensity of the exercise is the most important factor in the exercise prescription (Mathews & Fox, 1976). A minimal level of exercise intensity is required to ensure training effects, whereas a maximal safe intensity should not be exceeded. Because the initial fitness level determines this range, this parameter should be individually prescribed (Mathews & Fox, 1976). For example, depending upon initial fitness level, exercise intensity should be great enough to raise the heart rate by at least 60% from the resting value to the maximal value (Mathews & Fox, 1976). Therefore, a young low fit

male with a resting heart rate of 70 beats per minute and a maximal rate of 195 beats per minute would aim to have his heart rate reach 195-70 = 125 X 0.6 = 75+70=145 beats per minute during exercise. On the other hand, if this subject were highly fit, his target rate would be an increase of 80% from the resting value to the maximal value. Similarly, a maximal safe intensity can be established for each individual. Tables to calculate individual exercise heart rate ranges can be found in de Vries (1980).

The duration of exercise is dependent upon two factors: the energy source being developed; and the intensity of the exercise (American College of Sports Medicine, 1975; Mathews & Fox, 1976). During exercise, instant energy is produced in the muscle cells via anaerobic glycolysis (Guyton, 1981). Anaerobic output declines after 2-5 min and is replaced by aerobic energy derived from reactions requiring oxygen (Guyton, 1981). This lag between the start of exercise and the response of the aerobic system is the time needed for the heart and lungs to begin delivering the extra oxygen required for aerobic reactions (Mathews & Fox, 1976). Development of the aerobic pathway therefore requires that the exercise be maintained for at least 5 minutes to allow the aerobic system to become the primary source of muscular energy.

There is also an inverse relationship between duration of exercise and its intensity (American College of Sports Medicine, 1975). Although cardiorespiratory improvements have been obtained with high intensity-short duration sessions, these are not recommended for the average subject entering an exercise program (American College of Sports Medicine, 1975). Instead, sessions of moderate durations (20 to 30 minutes) and moderate intensities (60 to 70% of functional capacity) are advised for the first few weeks of a program. As cardiorespiratory fitness improves, the duration-intensity level can be gradually increased to 45 minutes at 80% of functional capacity (American College of Sports Medicine, 1975). Regardless of the duration-intensity level, each exercise session should be preceded by warm-up exercises of increasing intensity to prepare the body for vigorous activity. Similarly, each session should be followed by cooling down exercises of decreasing intensity to avoid muscle problems such as shin splints (de Vries, 1980).

Although there is a positive relationship between frequency of exercise and improvement in cardiorespiratory fitness, between three to five sessions a week are recommended. Research has shown that only slightly higher gains in improvement are realized with six or seven sessions per week, whereas injury rates increase (Pollock,

Gettman, Milesis, Bah, Durstine, & Juohnson, 1977).

Improvement in cardiorespiratory fitness is most rapid at the beginning of an exercise program when fitness is poorest. The greatest rate of change can be seen between 6 and 10 weeks, after which progress becomes slower as the fitness level improves (de Vries, 1980). However, generally the longer the program, the greater will be the improvement in fitness (Mathews & Fox, 1976).

Measurement of cardiorespiratory fitness. The maximal rate at which oxygen can be consumed per minute (VO2 max) is considered to be the single most accurate measure of cardiorespiratory fitness (Mathews & Fox, 1976). This is because VO2 max is limited by the ability of the lungs, heart, blood, and circulatory system to capture and transport oxygen to active muscles (Clausen, 1977). Thus, higher rates of VO2 max reflect higher levels of cardiorespiratory fitness as more energy can produced with less demand on the system (Clausen, 1977).

Direct measurement of VO2 max involves an individual breathing into a gasometer while working to exhaustion on a treadmill. This method requires sophisticated equipment, trained staff, and can be hazardous to the subject (Mathews & Fox, 1976). For these reasons, safer, less expensive tests have been developed that estimate VO2 max from submaximal exercise data. These tests are based

upon two facts: 1)that heart rate is linearly related to oxygen consumption during work requiring 50 to 100% of VO2 max (work is defined as force X distance, thus 100 kilograms lifted 3 metres = 300 kilogram metres of work); and 2)that there is a constant maximal heart rate for any given population (Mathews & Fox, 1976). Astrand and Rhyhming (1954) used these facts to develop a nomogram that estimates VO2 max from heart rate attained during submaximal exercise tests. For example, while pedalling a stationary bicycle (ergometer), an individual is given a series of increasing workloads at 2 min intervals. The workload that elicits a preselected heart rate of between 120 and 150 beats per minute is then maintained a further 4 min after which the test is terminated. The mean heart rate for the last 2 min of the test is then applied to the nomogram to derive an estimated VO2 max (Astrand & Rhyming, 1954).

Although this test is widely used, it has been criticized for its level of error which varies according to the workload used during the last six minutes of the submaximal test. Greater workloads have smaller standard errors of prediction. Thus, the errors of prediction are larger with unfit individuals who require a smaller workload to attain the criterion heart rate (de Vries, 1980). However, de Vries (1980) has argued that in order

to avoid undue risk to the subject, a known standard error is acceptable. Overall, this technique has a correlation of 0.74 with tests using the direct method of obtaining VO2 max. The advantages of the Astrand-Rhyming test are that only about fifteen minutes of a subject's time is required, little equipment is needed, and minimal risk to the subject is encountered (de Vries, 1971).

Physiological effects of aerobic exercise. A regular program of aerobic exercise causes physiological changes that reduce the relative stress placed upon the cardiorespiratory system during physical work. Skeletal muscle adaptations include: 1)an increase in concentration of myoglobin which releases stored oxygen to the mitochondria during muscle contraction (Pattengale & Holloszy, 1967); 2)an increase in both the size and number of mitochondria associated with an increase in their capacity to oxidize glycogen and fatty acids aerobically (Oscai, Mole, & Holloszy, 1971); and 3)a greater utilization of fat as an energy source during exercise (Mole, Oscai, & Holloszy, 1971). The result of these skeletal muscle changes is an increase in the capacity to produce energy aerobically.

Three major circulatory effects of training are apparent during both rest and exercise. These are: a decreased heart rate; an increased cardiac stroke volume;

and decreased blood pressure (Clausen, 1977). The first two result in maintaining the same cardiac output at fewer beats per minute (Mathews & Fox, 1976). Two different mechanisms are felt to contribute to the reduction in heart rate. The lowering of the resting heart rate is likely a direct effect of training on the heart, with the the amount of decrease dependent upon the VO2 max, cardiac output and stroke volume attained during training (Clausen, 1977). During exercise, the reduction may result from reduced sympathetic stimulation of the heart. This is suggested because the fall in exercise heart rate is closely related to a fall in sympathetic vasoconstriction activity in the splanchnic-hepatic vascular bed. Further, the extent of both these reductions is related to a concomitant increase in VO2 max (Clausen, 1977). Although not proven, it is felt that unknown receptors exist which respond to the demand imposed on the muscle metabolic system in proportion to its maximal capacity. If the system cannot meet the demand, the sympathetic system is activated. The enhanced oxidative metabolic capacity, due to training, therefore, might lessen this degree of activation (Clausen, 1977). The fact that training also reduces plasma catecholamine levels, which are considered an index of sympathetic nervous system activity, gives some support to this idea

(Winder, Hickson, Hagberg, Ehsani, & McClane, 1979).

In summary, aerobic exercise training improves the oxidative metabolic capacity of the skeletal muscle cells, and the oxygen transport capacity of the circulatory system. As a result the body's VO2 max is enhanced, thus demanding less change in metabolic pattern and circulatory regulation during exercise.

Effects of aerobic exercise on emotional reactivity. Recent research suggests that the physiological effects of aerobic exercise also modify sympathetic reactivity to psychosocial stress. Cox et al. (1979) found that cardiorespiratory fitness was unrelated to heart rate response during a series of psychosocial stressors, but was positively correlated with quicker heart rate recovery to baseline during a 5 min period immediately following the stress session. Similarly, Keller (1980) found no relationship between fitness level and skin conductance response during a psychosocial stressor, but a positive relationship during recovery. The fitter recovered more quickly. Hollander (Note 1) further explored the relationship between fitness level and sympathetic reactivity by monitoring both heart rate and skin conductance during exposure to a psychosocial stressor. Again fitness level was unrelated to either physiological response during stress, but was positively correlated in

both with the speed of recovery to baseline.

One recent study examined the effects of aerobic exercise on TABP (Blumenthal, Williams, Williams Jr., & Wallace, 1980). Significant reductions of the JAS type A scale following completion of a ten week aerobic exercise program were found only for Type A subjects. Type Bs did not show any change. Although physiological reactivity was not examined, improved physiological functioning (increased HDL, decreased cholesterol and blood pressure) was observed in both groups.

These findings of the beneficial effects of aerobic exercise suggest that it has potential as an intervention technique in modifying the physiological hyperreactivity manifested by Type A individuals.

Present Study

The present experiment was undertaken to explore the effects of a six week aerobic exercise program on physiological reactivity to controlled laboratory stressors in Type A subjects. Volunteers, who had previously been identified as healthy Type As, were further screened for physiological hyperreactivity during two challenging mental tasks, and for a level of cardiorespiratory fitness below 90% for their age group. Only individuals meeting the above criteria were included in the study. Subjects, initially matched on fitness

level, were randomly assigned to either Group Exercise, which received aerobic exercise training, or Group Stress Management, which received training in stress management techniques. A previous study by Brochocka (Note 2) found that stress management techniques had little effect in reducing hyperreactivity in Type As. Thus, it was felt to be not only a suitable control for subject expectancy and group participation, but would also serve as a check for any habituation of physiological reactivity due to repeated exposure to psychosocial stimuli.

All subjects were individually tested pre and post treatment. In each test session the subject first received two of four counterbalanced mental stress tasks, and then a submaximal ergometer test for cardiorespiratory fitness. Physiological reactivity was assessed by measures of heart rate, systolic and diastolic blood pressure, norepinephrine and epinephrine sampled prior to, during, and following termination of the stress tasks. Cardiorespiratory fitness was assessed by the estimated VO2 max level derived from the mean heart rate obtained during the last two minutes of the ergometer test (Astrand & Rhyming, 1954).

Three major questions were explored. First, would Type A hyperreactors randomly assigned to an aerobic exercise program improve in cardiorespiratory fitness. A

second question was derived from the observation that the physiological effects of aerobic exercise appear to modify heart rate reactivity to psychosocial stress. Post treatment, after exposure to mental stress, would Group Exercise show a decline in heart rate reactivity? A third and similar aim of the present study was to determine whether post treatment Group Exercise would also show declines in blood pressure and catecholamine reactivity during recovery from mental stress.

Method

Subjects

Subjects were recruited from 750 male, English speaking Bell Canada managers in central Montreal. During a three month recruitment period, each manager received two mailings of a letter (see Appendix A) that invited him to participate in a research stress management program. Of the 70 volunteers, 56 were invited to an interview at the Bell Medical Department, while 14 were asked to join a waiting list. The interview consisted of a brief description of the study, and an initial screening to identify candidates who exhibited: 1) no history of heart disease, hypertension and diabetes, as indicated on a personal data questionnaire, and the London School of Hygiene questionnaire (see Rose, 1962); 2) an asymptomatic resting 12 lead electrocardiogram (ECG); 3) a resting blood pressure below 145/90 mm Hg; and 4) a presence of TABP as assessed by the Structured Interview (Rosenman, 1978). Of the 56 individuals initially screened, 25 were disqualified for the following reasons: nine reported a history of chest pain or high blood pressure; one had an ECG which was not acceptable; thirteen did not exhibit TABP; and two were not available during the treatment program period. The 31 remaining candidates were then further screened at the Department of Psychology,

Concordia University to identify those who: 1) exhibited physiological hyperreactivity in response to challenging mental arithmetic and anagram tasks (at least a 20% increase over baseline, in at least three of the following measures; heart rate, systolic and diastolic blood pressure, plasma epinephrine and norepinephrine); and 2) an absence of a high level of cardiorespiratory fitness (top 10% of the population as measured by the Standardized Test of Fitness (Fitness & Amateur Sport Branch of the Department of National Health & Welfare, 1979). Three candidates did not meet the above criteria; one for reactivity, and two for cardiorespiratory fitness. The remaining 28 subjects (mean age, 37.7 years), after being matched on fitness level, were randomly assigned to either an aerobic exercise or stress management program. Prior to the start of the programs, one subject assigned to the exercise group withdrew because of a broken leg. Before participating in the study, all subjects were required to read and sign a Type A Project consent form (see Appendix B), and to make a $200.00 deposit as a guarantee that at least 75% of the treatment sessions would be attended. The deposits were placed in a special interest bearing account and were refunded with interest if attendance obligations were met. Any forfeited deposits were to be donated anonymously to a charitable organization. All

subjects agreed to the deposit, and all fulfilled attendance requirements.

Apparatus

Experimental testing took place in a 3 X 3.5 m temperature and humidity regulated electrically shielded chamber (Spectrashield). During the physiological reactivity test, a Vita-Stat microprocessor based blood pressure monitor (model 900-S) displayed and printed out heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP). A 19 gauge butterfly needle (Abbot-4550) and Becton-Dickinson evacuated tubes (Vacutainer) containing anticoagulant and antioxidant were used to obtain blood samples. During the cardiorespiratory test, HR was monitored via a Beckman dynograph (model R511A). The HR signal was recorded using Andover Medical Inc. Dyna-Trace ECG monitoring electrodes and processed through a Beckman cardiotachometer coupler (model 9857), which gave beat-to-beat interval tracings linearly proportional to HR (20 bpm/cm). A Bodyguard ergometer (model 990) and a Franz electric metronome (model LM-FB-5) were used for fitness testing. Additional equipment included a Sony (model TC630) reel to reel tape recorder and headphones, a Kodak Carousel (model 750A) slide projector connected to an automatic timer, and a Llyod's (model V182) cassette recorder.

Psychosocial Stimuli

A battery of four, three minute tasks were used to elcit physiological reactivity. Previous studies have shown that Type A individuals respond to these tasks with excessive and sustained levels of physiological arousal. To minimize order effect, the tasks were identically counterbalanced for each group.

Stroop Color-Word. A series of 80 color-word slides were flashed at one sec intervals onto a screen facing the subject. Each color-word ("red", "blue", "green", "yellow") was printed in an incongruous color (red, blue, green, yellow). The task was to identify the color of the print and not the word itself. For example, if the word "green" appeared printed in red, the correct answer was "red". Random color-words were simultaneously heard order over the headphones.

General Knowledge Quiz. This task consisted of 23 tape recorded questions and answers drawn from a 35 item test designed by Schiffer, Heartley, Schulman and Abelman (1976). The series of questions varied in difficulty, with some appearing deceptively simple. Subjects were given 5 sec to reply, after which the correct response was heard via the headphones.

Digit Span. A random series of seven, eight or nine digit numbers were presented via the headphones. The

subject was required to correctly repeat each sequence of numbers within five seconds. Subjects were told that the task was a subscale of the Wexler Aptitude and Intelligence Scale, and that they would be tested until ten consecutive correct repetitions occurred or three minutes had elapsed.

Reaction Time. In this task, a red or green panel light was flashed at varying intervals. The subject was instructed to depress a telegraph key near his left hand only in response to the green panel light. The sequence and timing of the lights was random, with interstimulus intervals ranging from 5 to 15 sec.

Treatments

Aerobic exercise. Supervised sessions were held at the Ville Marie Squash Club, in central Montreal, four times weekly for six consecutive weeks. Each session consisted of the following protocol: 1)a 5 to 10 min warm-up period of stretching and calisthenics, 2)at least 20 min of walking or jogging, depending on fitness level, and 3)a 5 to 10 min cooling down period of stretching and calisthenics. Individual exercise intensities were prescribed to produce elevations of HR equal to 60-80% from the resting value to the maximal value. Subjects were taught and encouraged to measure their own HR by carotid or radial artery palpation. For the first week

about 50% of the subjects were walking only, but by the sixth and final week, almost all subjects were jogging continuously for more than 30 min.

Stress Management. Two trained behavior therapists led these sessions, which were held in a seminar room at Bell Canada for one hour, two times weekly for six consecutive weeks. A make-up session was offered weekly for subjects unable to attend any of the scheduled weekly sessions. The aim of the program was to develop skills in handling everyday stress. This involved learning to: 1)identify cognitive, behavioral and physiological stress cues; 2)substitute relaxation for physical tension; 3)prepare for and confront a stress situation; and 4)evaluate and reinforce the successful use of the new skills. Homework consisting of a stress diary and relaxation practice was assigned at the end of each session. This was considered an essential part of the program in developing the new skills.

Procedure

Subjects were individually tested at two identical evaluation sessions: before treatment (pre) and end of treatment (post). To reduce variability in physiological reactivity, subjects were asked to abstain from the following for at least one hour before each test session: smoking, caffeine, alcohol, drugs, and strenuous activity.

To allow for the diurnal variation of catecholamines, each subject was tested at the same time of day in both sessions. In each session the subject first received two of four counterbalanced psychosocial stress tasks, and then a submaximal ergometer test for cardiorespiratory fitness.

Upon arrival at the laboratory, compliance with the test restrictions was noted. Non-compliance contraindicated testing and the appointment was then re-scheduled. No subject failed to comply. After changing into loose clothing and runnning shoes suitable for pedalling an ergometer, weight was recorded, and an Informed Consent Form (see Appendix C) was read and signed. Following preparation of the skin with alcohol, HR electrodes were placed on either side of the back, just above the shoulder blade, with a ground electrode on the lower part of the sternum.

Physiological Reactivity Test. The subject then entered a shielded testing room and was seated comfortably in a reclining chair behind a full-length privacy drape. Following an explanation of the blood sampling technique, a butterfly needle with attached catheter (heparinized between sample collections) was inserted into a right forearm vein. An opening in the drape allowed exposure of the subject's right arm to facilitate blood sampling. At

five predetermined times during the session, blood samples were drawn off into evacuated tubes, which were immediately placed on ice. Next, an occluding blood pressure cuff was wrapped on the upper left arm with its microphone positioned over the brachial artery. At this point, the subject was asked to relax and sit quietly for a few minutes in order that a baseline measurement could be made. Finally, the headphones were secured, and the blood pressure monitor and cassette recorder were activated.

During the baseline period, the subject listened to quiet guitar music while HR and BP were recorded each minute. After 10 min, baseline was established by the first two consecutive readings that showed no variance greater than 5 bpm and 5 mm Hg respectively. At this time, the baseline blood sample was drawn. After 30 min, if baseline was not reached, a blood sample was taken and the session was terminated. No subject failed to reach baseline within 30 min.

Following the baseline period, the tape-recorder was activated and the following general instructions were given:

> You are now going to be presented with a series of tasks that measure your ability to think and move quickly and accurately. These tasks have

been selected because they differentiate well between successful and unsuccessful managers. In this session you will be presented with two challenges, randomly selected from a battery of six. For some of these tasks, we shall tell you what the expected level of performance is for successful managers, while in others you will simply be asked to do as well as you can.

Two, three minute stress tasks, separated by a three minute rest period were then administered. At the end of the second stress task, there was a ten minute recovery period, after which the headphones, butterfly needle, and BP cuff were removed. During the stress tasks and recovery, HR and BP were recorded each minute. Blood samples were taken at the end of each stress task, and at the second and fifth minutes of recovery. Plasma epinephrine (E) and norepinephrine (NE) were assayed by an independent laboratory using the radioenzymatic method of Peuler and Johnson (1975). This method simultaneously converts E and NE to their corresponding labelled metabolites (metanephrine and normetanephrine) by using a catylzing enzyme in the presence of a radioactive agent. The labelled metabolites are then extracted and separated by thin-layer chromatography. The radioactivity attributable to each catecholamine is determined by

scintillation counting. Catecholamine content in plasma is expressed as the number of picograms/ml.

Cardiorespiratory Fitness Test. The subject was then seated on the ergometer. After adjusting the seat and handlebars, the electrode leads were connected and the polygraph was activated. A pedalling frequency of 50 cycles/min was established by a metronome pulse of 100 beats/min. Following a 1 min warm-up, with no workload, the workload on the ergometer was set at 1 kg, by tightening a frictional band. The test then consisted of progressively increasing, every two min, the workload in 1 kg increments, until HR had reached 120 bpm. The workload which attained this rate was then maintained for a further 4 min. A cool-down period of pedalling with no workload for 1 min, followed. This test, exclusive of the warm-up and cool-down periods could last anywhere from 6 to 14 min depending on level of cardiorespiratory fitness. Cardiorespiratory fitness (CF) levels were obtained by applying work load and HR during the final two min of the test to the Astrand and Rhyming nomogram for est VO2 max in liters/min. This value was then divided by the subject's weight in kilograms to derive a final est VO2 max in ml/kg/min.

Data Analysis

Heart rate, systolic and diastolic blood pressure,

epinephrine and norepinephrine measures were obtained from the following three points in the record: 1)basal (B); 2)the peak response elicited during the two stress tasks (S); and 3)the fifth minute of recovery (R). Due to within subject variability in basal physiological indices, stress and recovery points were expressed as percentage change from basal to better reflect the magnitude of reactivity relative to resting level. Because the variability in the scores within stress versus recovery was so disparate separate analyses of variance were performed for these periods.

Results

Individual subject's data for basal, stress and recovery periods for the two test sessions are contained in the following appendices: Appendix D) est VO2 max; Appendix E) heart rate; Appendix F) systolic blood pressure; Appendix G) diastolic blood pressure; Appendix H) epinephrine; and Appendix I) norepinephrine. Appendix J contains the list of counterbalanced mental stress tasks used by each subject.

Figure 1 is provided to give a graphic description of the level each physiological variable reached during basal, stress and recovery periods. It contains the group means for the absolute values of heart rate, systolic and diastolic blood pressure, epinephrine and norepinephrine.

Examination of the est VO2 max measures revealed that three subjects in Group Stress Management showed a 25% or greater improvement in cardiorespiratory fitness post treatment. Personal communication revealed that during the treatment period each subject had begun some form of aerobic exercise. These subjects' data were therefore excluded from statistical analysis.

Cardiorespiratory Fitness

Figure 2 contains the group means for est VO2 max for each test session. A 2 X 2 (Groups X Sessions) repeated measures analysis of variance unweighted-means solution

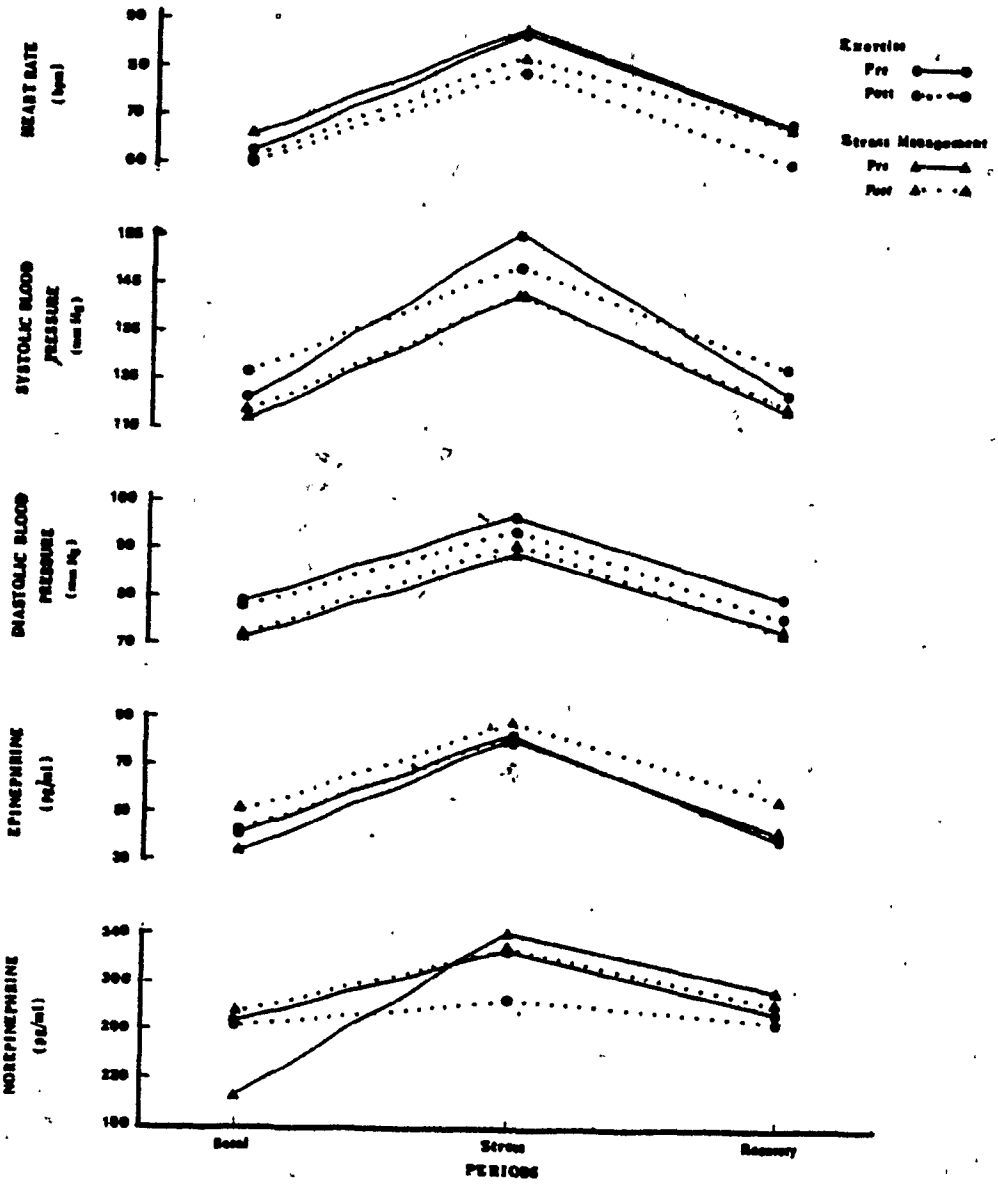

Figure 1. Mean heart rate, systolic blood pressure, diastolic blood pressure, epinephrine, and norepinephrine during Basal, Stress, and Recovery, Pre and Post treatment, for Groups Exercise and Stress Management.

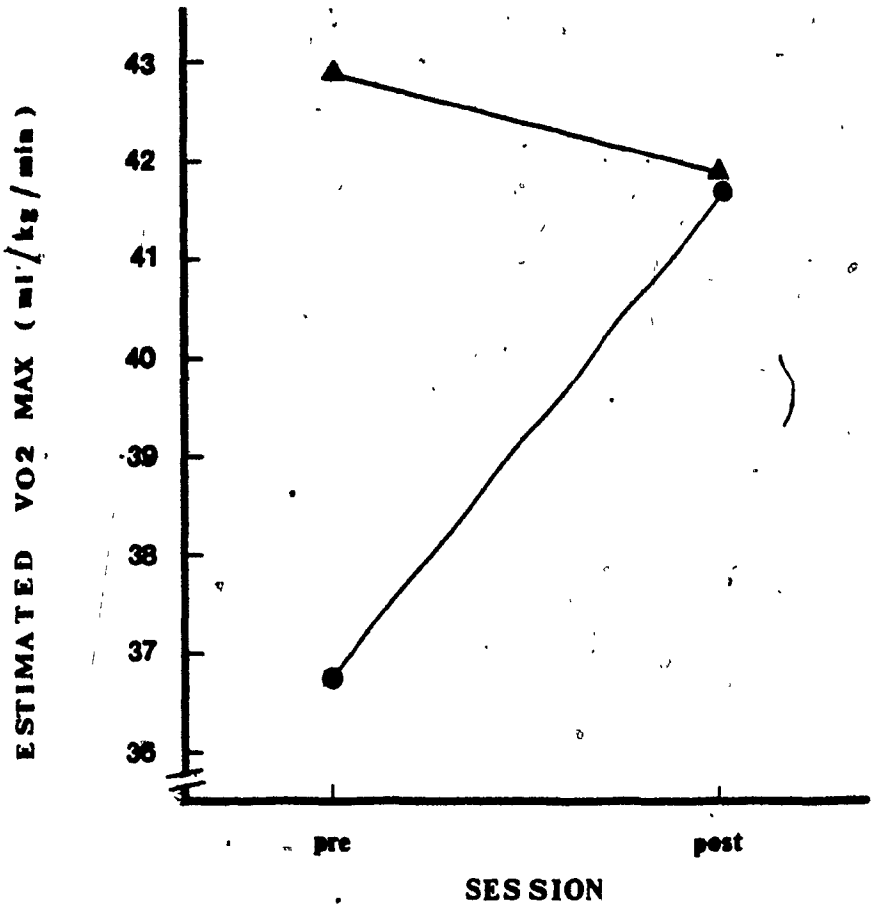

Figure 2. Mean extimated VO2 max in ml/kg/min for Groups Exercise (●) and Stress Management (▲) across the Pre and Post test sessions.

(ANOVA) (Winer, 1971) revealed a significant Sessions effect ($F(1,22)=4.3, p < .05$) and a significant Groups X Sessions interaction ($F(1,22)=10.13$, $p < .01$) (see Table 1). The critical difference (cd) calculated for a Tukey test revealed that Pre scores for Group Stress Management were significantly higher than those of Group Exercise, while Post scores did not differ ($cd=3.74$, $p < .01$). Moreover, while scores for Group Stress Management remained virtually unchanged from Pre to Post, Group Exercise had significantly higher Post scores.

Physiological Reactivity

Heart Rate. ANOVA of Pre and Post basal heart rate (HR) revealed no group differences. Both groups maintained approximately the same basal HR after treatment as before treatment (see Table 2). Figure 3 contains the group means for percent change from basal HR ($\triangle$HR) during stress and recovery for each test session. ANOVA of Pre and Post stress $\triangle$HR found no group differences. Both groups showed a pronounced rise from basal levels in both test sessions (see Table 2). Figure 4 contains the group means for recovery $\triangle$HR for each test session. ANOVA of this period (see Table 3) revealed a significant Groups X Sessions interaction ($F(1,22)=5.15, p < .05$). Tukey tests ($cd=10.15$, $p < .05$) indicated that there were no Pre or Post group differences. However, while scores for Group

Table 1

Summary of Two-Way Repeated Measures Analysis of Variance on Estimated VO2 Max

Source of Variance	SS	df	MS	F	p
Between Subjects					
Group	12416.83	1	12416.83	.94	-
Error (between)	289596.07	22	13163.46		
Within Subjects					
Session	4446.99	1	4446.99	4.30	.05
Group X Session	10504.02	1	10504.02	10.13	.01
Error (within)	22803.27	22	1036.51		

Table 2

Means and Standard Errors for Heart Rate during Pre and Post test sessions for Basal, Stress and Recovery periods for Groups Exercise and Stress Management

	GROUP EXERCISE		GROUP STRESS MANAGEMENT	
Period	Pre	Post	Pre	Post
Basal*	61.92	60.46	65.55	62.27
	±2.54	±3.01	±3.02	±2.74
Stress**	41.31	30.84	35.18	33.00
	±5.31	±5.68	±5.46	±6.59
Recovery**	10.31	0.00	3.36	9.36
	±4.12	±2.64	±2.17	±5.28

* Measures expressed in bpm.
** Measures expressed as percent change from basal.

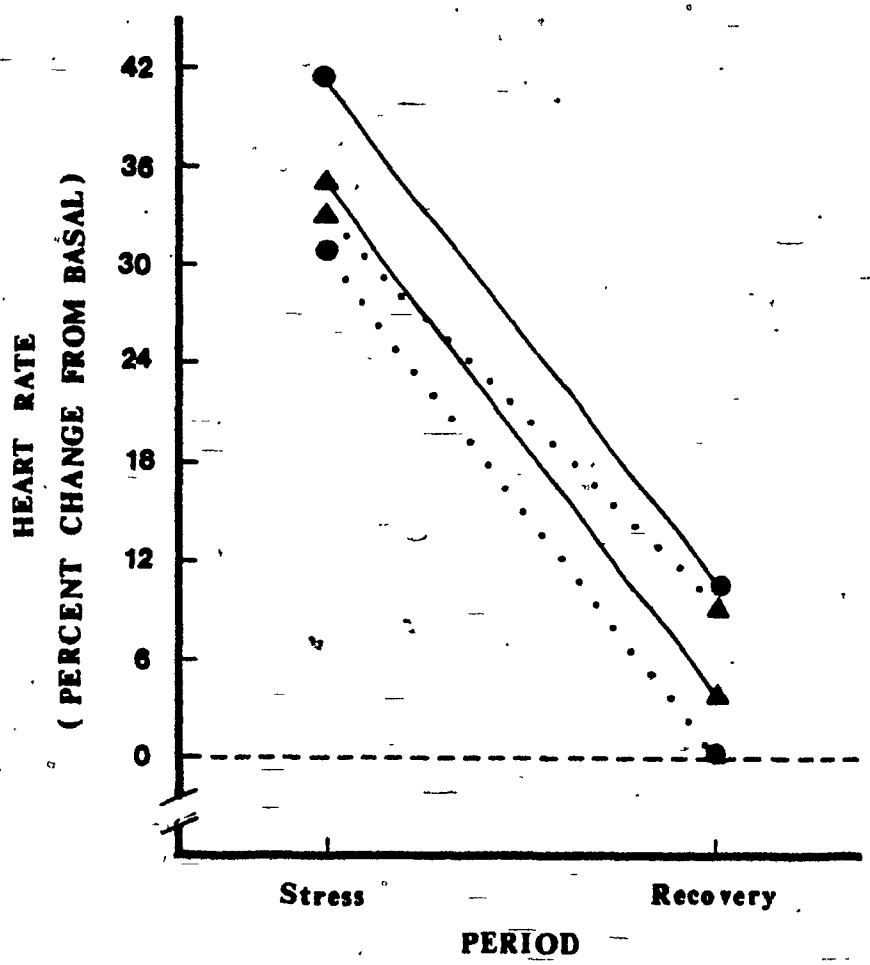

Figure 3. Mean percent change from basal in heart rate during stress and recovery, Pre (—) and Post (···) treatment, for Groups Exercise (●) and Stress Management (▲).

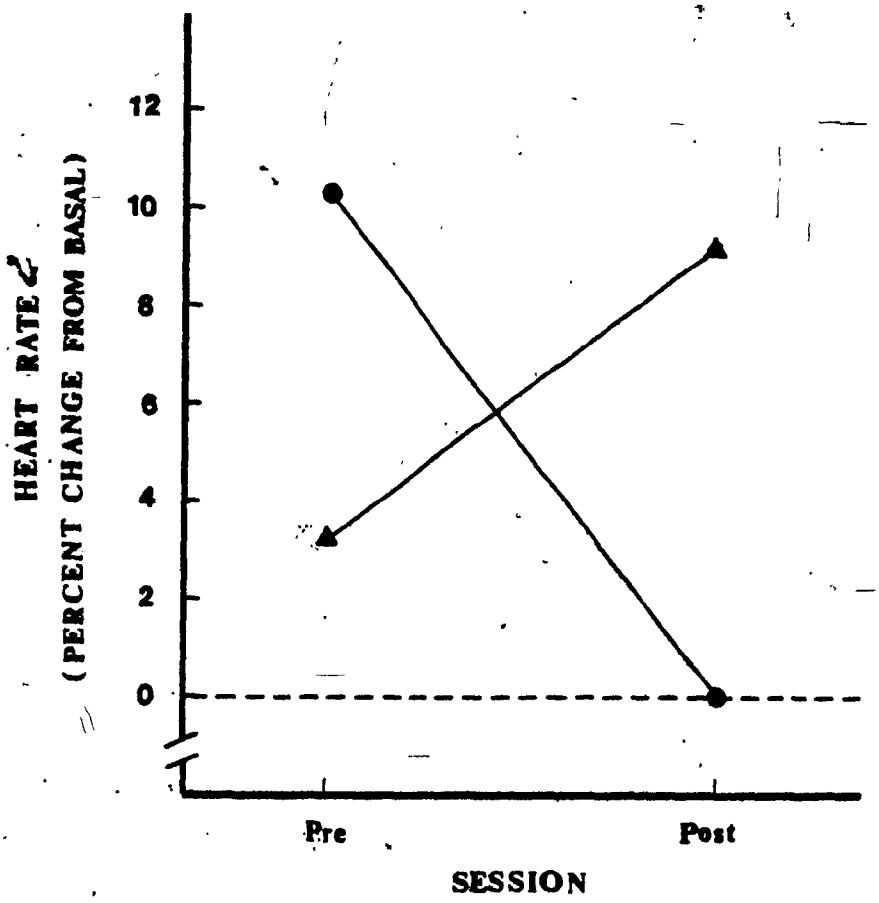

Figure 4. Mean percent change from basal in heart rate during recovery, Pre and Post treatment, for Groups Exercise (●) and Stress Management (▲).

Table 3

Summary of Two-Way Repeated Measures Analysis of Variance on Recovery Heart Rate

Source of Variance	SS	df	MS	F	p
Between Subjects					
Group	17.28	1	17.28	.10	-
Error (between)	3960.48	22	180.02		
Within Subjects					
Session	55.31	1	55.31	.36	-
Group X Session	792.80	1	792.80	5.16	.05
Error (within)	3383.38	22	153.79		

Stress Management did not change from Pre to Post, Group Exercise had significantly lower post scores (see Table 2).

Systolic Blood Pressure. Figure 5 contains the group means for basal systolic blood pressure (SBP) for each test session. ANOVA of this period revealed a significant Sessions effect ($F(1,22)=7.19$, $p<.05$) (see Table 4). Subsequent Tukey tests ($cd=3.85$, $p<.01$) indicated that scores for Group Stress Management remained unchanged from Pre to Post, while Group Exercise had significantly higher post scores (see Table 5). Figure 6 contains the group means for percent change from basal in SBP ($\triangle$SBP) during stress and recovery for each test session. ANOVA of Pre and Post stress $\triangle$SBP revealed no group differences. Both groups exhibited a marked increase from basal levels in both test sessions (see Table 5). ANOVA of Pre and Post recovery $\triangle$SBP found no group differences. Both groups had essentially returned to basal levels in both test sessions (see Table 5).

Diastolic Blood Pressure. ANOVA of Pre and Post basal diastolic blood pressure (DBP) revealed no group differences. Both groups achieved approximately the same scores in both test sessions (see Table 6). Figure 7 contains the group means for percent change from basal in DBP ($\triangle$DBP) during stress and recovery for each test

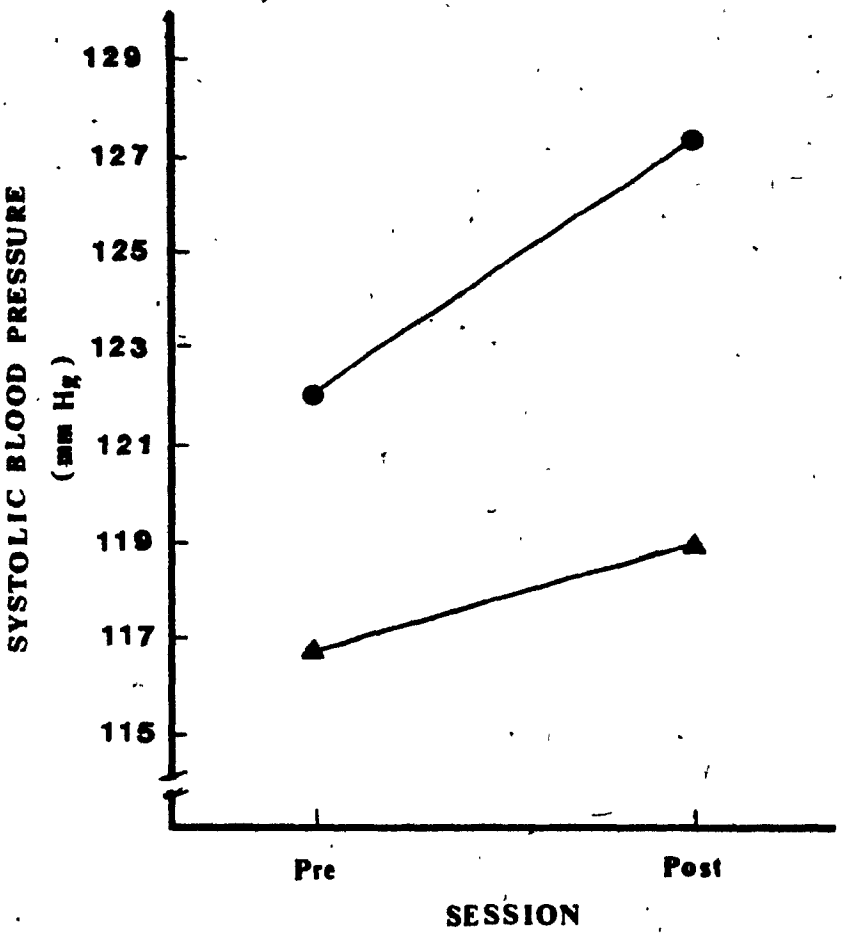

Figure 5. Mean basal systolic blood pressure in mm Hg for Groups Exercise (●) and Stress Management (▲) across the Pre and Post test sessions.

Table 4

Summary of Two-Way Repeated Measures Analysis of Variance on Basal Systolic Blood Pressure

Source of Variance	SS	df	MS	F	p
Between Subjects					
Group	546.29	1	546.29	2.80	-
Error (between)	4294.83	22	195.22		
Within Subjects					
Session	161.40	1	161.40	7.19	.05
Group X Session	23.36	1	23.36	1.04	-
Error (within)	493.54	22	22.43		

Table 5

Means and Standard Errors for Systolic Blood Pressure during Pre and Post test sessions for Basal, Stress and Recovery periods for Groups Exercise and Stress Management

	GROUP EXERCISE		GROUP STRESS MANAGEMENT	
Period	Pre	Post	Pre	Post
Basal*	122.00	127.08	116.63	118.91
	±2.21	±3.20	±3.29	±3.34
Stress**	24.00	16.38	22.64	20.64
	±1.95	±2.61	±3.03	±3.86
Recovery**	-0.54	1.62	2.45	-0.10
	±1.73	±1.52	±1.46	±1.98

* Measures expressed in mm Hg.
** Measures expressed as percent change from basal.

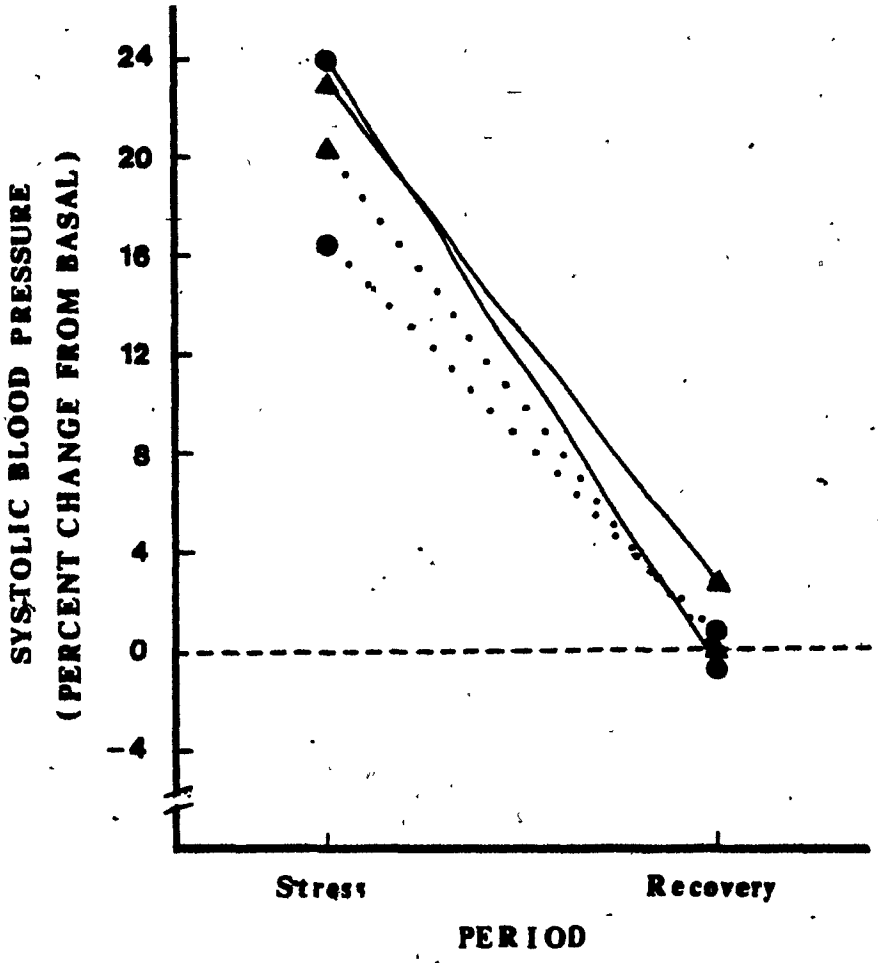

Figure 6. Mean percent change from basal in systolic blood pressure during stress and recovery, Pre (—) and Post (···) treatment, for Groups Exercise (●) and Stress Management (▲).

Table 6

Means and Standard Errors for Diastolic Blood Pressure during Pre and Post sessions for Basal, Stress and Recovery periods for Groups Exercise and Stress Management

	GROUP EXERCISE		GROUP STRESS MANAGEMENT	
Period	Pre	Post	Pre	Post
Basal*	78.31	76.53	71.00	72.64
	±3.09	±3.50	±3.17	±3.54
Stress**	24.77	23.08	24.45	25.45
	±3.98	±4.41	±2.41	±6.91
Recovery**	1.85	1.00	3.18	-1.00
	±1.72	±3.40	±2.05	±1.66

* Measures expressed in mm Hg.
** Measures expressed as percent change from basal.

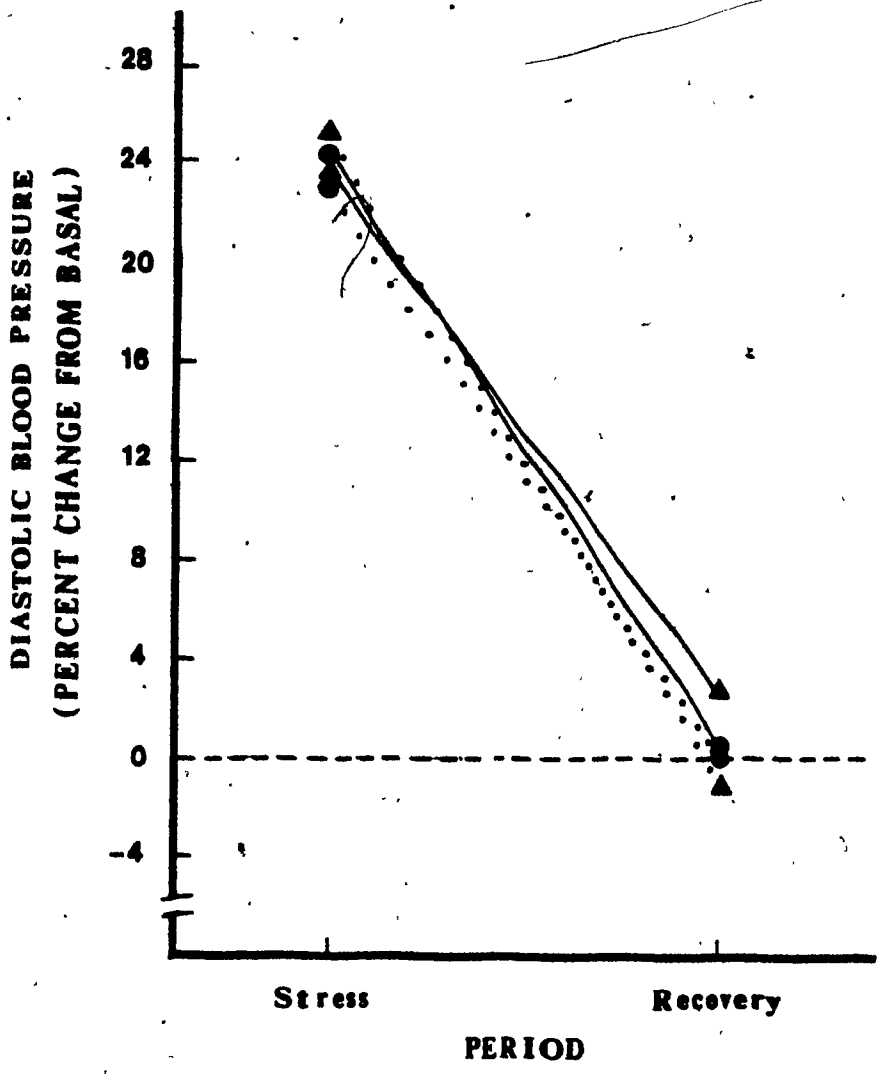

Figure 7. Mean percent change from basal in diastolic blood pressure during stress and recovery, Pre (—) and Post (···) treatment, for Groups Exercise (●) and Stress Management (▲).

session. ANOVA of Pre and Post stress △ DBP found no group differences. Both groups showed a marked rise during stress in both test sessions (see Table 6). Similarly, ANOVA of Pre and Post recovery △DBP found no group differences. Both groups were approaching basal levels in both test sessions (see Table 6).

Epinephrine. ANOVA of Pre and Post basal epinephrine (E) revealed no group differences. Group Exercise remained virtually unchanged from Pre to Post, while Group Stress Management showed slightly higher Post scores (see Table 7). Figure 8 contains the group means for percent change from basal E (△ E) during stress and recovery. ANOVA of Pre and Post stress △ E found no group differences, with both groups showing a very pronounced rise from basal (see Table 7). Due to a technical mishap two samples of recovery E from Group Exercise were lost. ANOVA of Pre and Post recovery △ E revealed no group differences. In both test sessions Group Exercise had returned below basal, while Group Stress Management remained slightly elevated above basal (see Table 7).

Norepinephrine. Figure 9 contains the group means for basal norepinephrine (NE) for each test session. ANOVA of this period revealed a significant Groups X Sessions interaction ($F(1,22)=4.39$, $p<.05$) (see Table 8). Tukey tests ($cd=51.25$, $p<.05$) indicated that Pre scores

Table 7

Means and Standard Errors for Epinephrine during Pre and Post test sessions for Basal, Stress and Recovery periods for Groups Exercise and Stress Management

	GROUP EXERCISE		GROUP STRESS MANAGEMENT	
Period	Pre	Post	Pre	Post
Basal*	41.17	42.91	34.84	52.34
	±5.90	±4.46	±3.17	±7.48
Stress**	102.46	83.23	131.27	70.18
	±19.28	±21.14	±37.44	±23.86
Recovery**	-1.00	-2.10	22.00	8.73
	±19.16	±14.91	±26.00	±16.00

* Measures expressed in pg/ml.
** Measures expressed as percent change from basal.

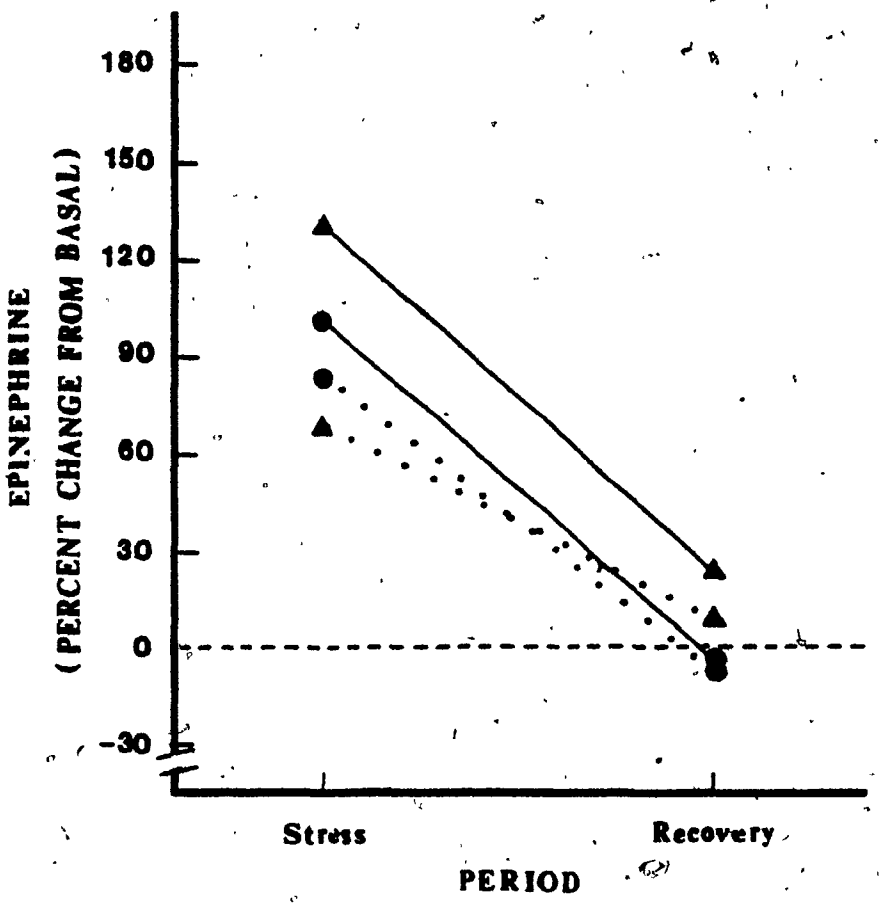

Figure 8. Mean percent change from basal in epinephrine during stress and recovery, Pre (—) and Post (···) treatment, for Groups Exercise (●) and Stress Management (▲).

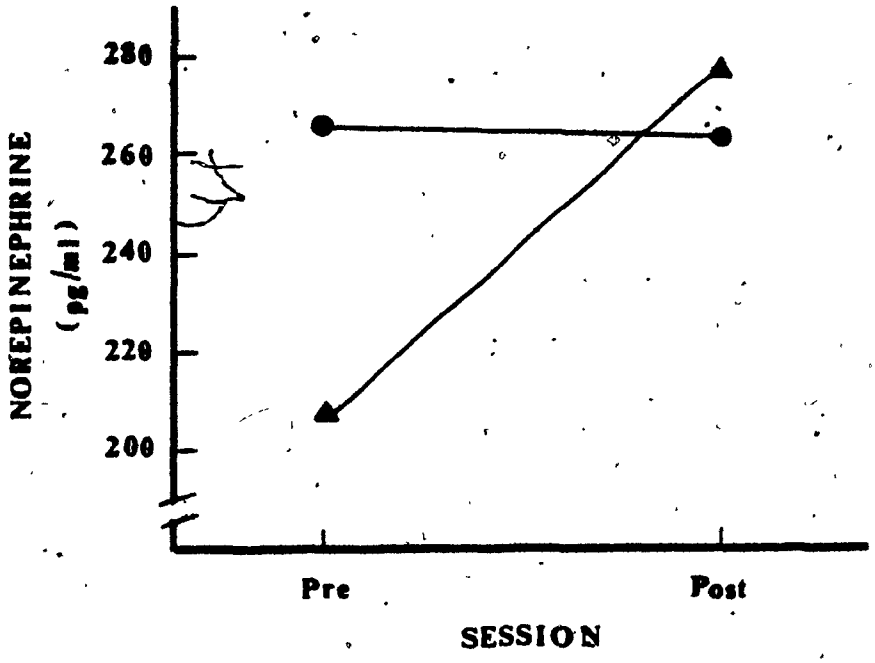

Figure 9. Mean basal norepinephrine in pg/ml for Groups Exercise (●) and Stress Management (▲) across the Pre and Post test sessions.

Table 8

Summary of Two-Way Repeated Measures Analysis of Variance on Basal Norepinephrine

Source of Variance	SS	df	MS	F	p
Between Subjects					
Group	544358.86	1	544358.06	.88	-
Error (between)	1364939.50	22	620426.78		
Within Subjects					
Session	1414586.45	1	1414586.45	3.79	-
Group X Session	1634760.18	1	1634760.18	4.39	.05
Error (within)	8192376.60	22	372380.75		

for Group Exercise were significantly higher than those of Group Stress Management, while Post scores did not differ. In addition, while scores for Group Exercise did not change from Pre to Post, Group Stress Management had significantly higher Post scores (cd=11.05, $p < .01$) (see Table 9). Figure 10 contains the group means for percent change from basal NE (Δ NE) during stress for each test session. ANOVA of this period (see Table 10) revealed a significant Group effect ($F(1,22)=4.84$, $p < .05$), a significant Sessions effect ($F(1,22)=19.75$, $p < .01$), and a significant Groups X Sessions interaction ($F(1,22)=4.83$, $p < .05$). Tukey tests (cd=27.93, p .01) indicated that Pre scores for Group Stress Management were significantly higher than those of Group Exercise, while Post scores did not differ. Scores for Group Exercise remained unchanged from Pre to Post, while Group Stress Management had signficantly lower Post scores (see Table 9). Figure 11 contains the group means for recovery Δ NE for each test session. ANOVA of this period (see Table 11) revealed a significant Sessions effect ($F(1,22)=5.30$, $p < .05$). Tukey tests (cd=35.30, $p < .01$) indicated that scores for Group Exercise remained virtually the same from Pre to Post, while Group Stress Management had significantly lower Post scores (see Table 9).

Table 9

Means and Standard Errors for Norepinephrine during Pre and Post test sessions for Basal, Stress and Recovery periods for Groups Exercise and Stress Management

	GROUP EXERCISE		GROUP STRESS MANAGEMENT	
Period	Pre	Post	Pre	Post
Basal*	266.07	263.49	207.67	279.15
	±25.39	±16.82	±19.49	±16.90
Stress**	23.38	7.77	63.73	17.54
	±8.23	±6.14	±15.29	±6.34
Recovery**	3.62	1.38	39.64	1.45
	±4.87	±7.55	±15.79	±3.98

* Measures expressed in pg/ml.
** Measures expressed as percent change from basal.

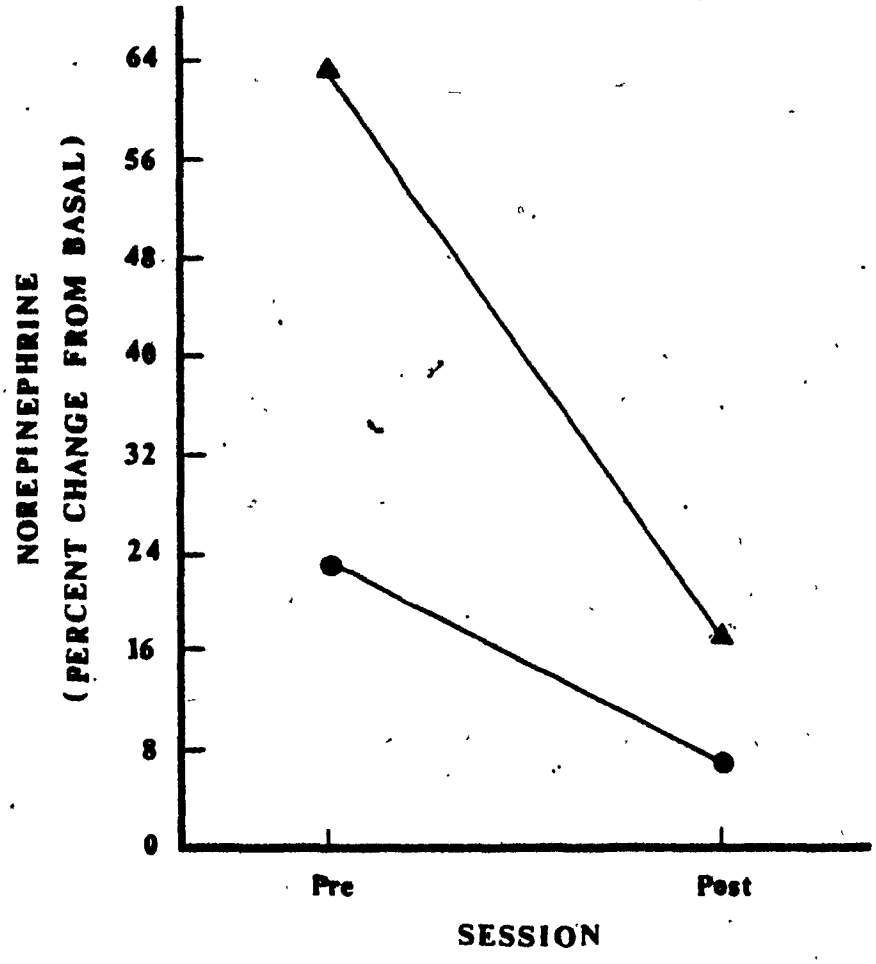

Figure 10. Mean percent change from basal in norepinephrine during stress, for Groups Exercise (●) and Stress Management (▲) across the Pre and Post test sessions.

Table 10

Summary of Two-Way Repeated Measures Analysis of Variance on Stress Norepinephrine

Source of Variance	SS	df	MS	F	p
Between Subjects					
Group	7485.88	1	7485.88	4.84	.05
Error (between)	34015.94	22	1546.18		
Within Subjects					
Session	11381.34	1	11381.34	19.75	.01
Group X Session	2786.66	1	2786.66	4.83	.05
Error (within)	12680.36	22	576.38		

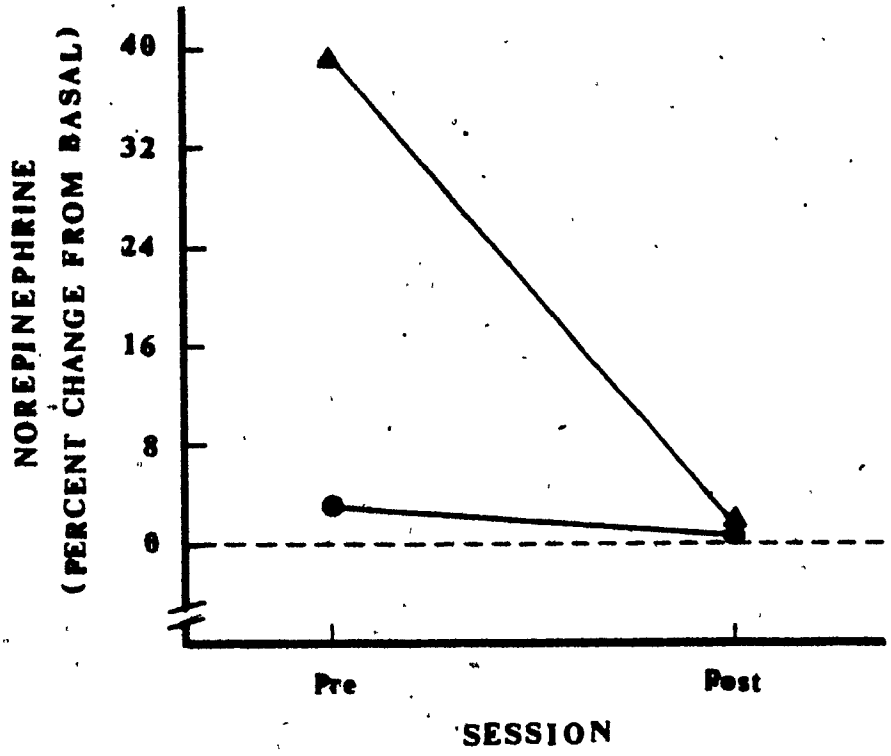

Figure 11. Mean percent change from basal in norepinephrine during recovery, for Groups Exercise (●) and Stress Management (▲) across the Pre and Post test sessions.

Table 11

Summary of Two-Way Repeated Measures
Analysis of Variance on
Recovery Norepinephrine

Source of Variance	SS	df	MS	F	p
Between Subjects					
Group	3881.39	1	3881.39	3.94	-
Error (between)	21636.45	22	983.48		
Within Subjects					
Session	4871.11	1	4871.11	5.30	.05
Group X Session	3851.35	1	3851.35	4.19	-
Error (within)	20214.97	22	918.86		

Discussion

Type A hyperreactors randomly assigned to a six week aerobic exercise program showed a significant improvement in cardiorespiratory fitness. Moreover, after training, these subjects displayed a significant decline in heart rate reactivity during recovery from mental stress. In contrast, these measures remained relatively unchanged in the non-exercising controls. This enhanced heart rate recovery confirms previous findings that cardiorespiratory fitness is associated with quick physiological recovery from mental stress (Cox et al, 1979; Hollander, Note 1; Keller, 1980). Zimmerman and Fulton (1981) have suggested enhanced heart rate recovery following mental stress is not a result of improved cardiorespiratory fitness but is due to lower basal heart rates. The results of the present study do not support this idea. Group Exercise revealed no significant change between Pre and Post basal heart rate. The observed changes in heart rate recovery, therefore, appear independent of baseline differences.

The effects of aerobic exercise training did not appear to modify blood pressure and catecholamine reactivity during recovery from mental stress. No group differences were observed for systolic and diastolic blood pressure or for epinephrine, whereas, contrary to expectations, Group Stress Management showed a significant

decline in norepinephrine reactivity.

One possible explanation for these findings is that some physiological indices may have a shorter recovery time course than others. For example, Hollander (Note 1) found a positive correlation between fitness level and speed of recovery to baseline in heart rate and skin conductance, but a difference in time course between the two measures. As the present study examined only the fifth minute of recovery, earlier group differences may have been missed. Future studies seem justified to investigate this area.

A second possibility is that recovery in some measures may be influenced by factors other than improvement in cardiorespiratory fitness. In general, the blood pressure recovery measures indicated that all subjects had returned to basal levels in both test sessions. It is possible that this recovery was due to a physical mechanism of the circulation called "stress-relaxation" which helps control arterial pressure (Guyton, 1981). A rise in arterial pressure initiates an adaptation process in the smooth muscle fibres of the arteries, which gradually causes a progressive distention of the vessels and a corresponding decrease in pressure. When the stress on the system is removed, blood pressure immediately falls back to basal levels (Guyton, 1981).

Whether this mechanism influenced blood pressure recovery rate in the present study is unknown. Further elucidation must await studies specifically designed to examine this issue.

The catecholamine recovery measures suggest that different factors may be affecting epinephrine and norepinephrine activity during this period. Ursin, Baade and Levine (1978) proposed that epinephrine activity is related to active coping during the performance of a task. Once the task has been completed there is a pronounced fall in activity. The present data tend to support this idea. Epinephrine recovery for both groups was characterized by a rapid return towards baseline after task completion. Norepinephrine recovery, on the other hand, presented a different picture, which is better dealt with by a discussion of overall norepinephrine reactivity. Initially, there was a Pre basal difference between the groups, with Group Exercise having significantly higher levels than Group Stress Management. There is no immediate explanation for this difference as the only factor differentiating the two groups was group assignment. Thus, any interpretation of findings must be tempered with caution. Post treatment, Group Stress Management displayed a significant increase in basal norepinephrine and significant declines in norepinephrine

reactivity during stress and recovery, while Group Exercise remained unchanged. Investigations have shown that tasks involving muscular use increase norepinephrine concentrations (Folkow, Haggendahl, & Lisander, 1967; Hoffman, Benson, Arns, Stainbrook, Landsberg, Young, & Gill, 1982; Ziegler, Lake, & Kopin, 1977). The relaxation technique taught to Group Stress Management consisted of alternately tensing and relaxing groups of muscles. If subjects in this group used this technique during the resting period, it is possible that increased basal norepinephrine post treatment was due to this factor. The reason for the decline in norepinephrine reactivity during stress and recovery is not clear. Ursin et al. (1978) suggested that norepinephrine activity, during cognitive tasks, may be related to "fear of failure". It is possible that stress management training may have provided a means of coping with this fear. However, this conclusion must remain speculative until future studies without the confound of baseline differences can be conducted.

In an extensive review of physiological adaptations to physical training Scheurer and Tipton (1977) found that chronic exercise programs, of ten weeks or longer, resulted in a reduction in resting sympathetic activity, whereas, short periods of training produced mixed results.

Shorter training programs which significantly increased est VO2 max were found to decrease, increase or not change resting sympathetic activity. The present results tend to agree with these findings. After a six week training program, Group Exercise displayed a significant increase in basal systolic blood pressure, while basal levels of heart rate, diastolic blood pressure, epinephrine and norepinephrine remained virtually unchanged. Future studies should therefore consider these facts when planning the length of exercise programs.

The use of contrived laboratory mental stress tasks to elicit physiological arousal has raised the question whether repeated exposure to these stimuli may result in habituation. In the present study, four of the physiological parameters that were followed, post treatment, showed a marked reduction in reactivity during stress. This was seen for heart rate, systolic blood pressure, epinephrine and norepinephrine. These response decrements were not attributed to habituation because only one was significant, and none were uniform for either groups or measures. A high degree of activation was still evident in at least three of the five measures for at least one of the groups. The importance of this finding is that this paradigm offers a relatively controlled, efficient and inexpensive method to explore physiological

reactivity compared to real life stress situations.

It is of interest to note that subjects in the present study displayed a strong commitment to treatment. All subjects met the 75% attendance requirement of their respective groups, with some attending 100% of the sessions. Attendance compliance must be considered an important prerequisite if intervention techniques are to prove beneficial in changing Type A physiological hyperreactivity.

One unexpected problem arose at the beginning of the study. Before being randomly assigned to groups, subjects were matched on cardiorespiratory fitness level based on the results of their first ergometer test, which was done in the Screening Session. The results of the second ergometer test, which was done just as the subjects were entering treatment, revealed that Group Stress Management had significantly higher scores than Group Exercise. This variability may be related to error which occurs on initial exposure to ergometric testing. De Vries (1971) has recommended that at least one previous experience on the ergometer is required when this test is to be used for prediction of est VO2 max.

A second problem was encountered post treatment. Although all subjects had been asked not to change their life-style during treatment, three subjects in Group

Stress Management began some form of aerobic exercise. Although no statistical analyses were performed, a visual scanning of the data found a decline in recovery heart rate reactivity in two of the subjects post treatment. No other changes were apparent. It is suggested that future studies carefully monitor life-style changes during treatment to minimize potential confounds.

In conclusion, the present study demonstrates the effectiveness of aerobic exercise in reducing heart rate reactivity in Type A hyperreactors during recovery from mental stress. Since Type As greater susceptibility to CHD might be due to their sustained physiological hyperreactivity to challenge, these findings imply that the physically fit may be less prone to develop CHD. When actively coping with challenges in their environment physically fit Type As would recover more quickly to basal levels, whereas, the physically unfit Type As would continue to function at elevated levels. Repeated exposure to challenge situations might result in the development of cardiovascular pathologies in the unfit. Aerobic exercise might not only contribute to reduce the Type As hyperreactor's risk for CHD, but it might also reduce the recurrence of myocardial infarction in those individuals who have suffered their first heart attack. As the therapeutic benefits of reducing any aspect of

physiological hyperreactivity are potentially enormous, the short-term improvements produced by aerobic exercise in the present study warrant further exploration.

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Appendix A

Letter of Invitation

TO: All Bell Canada Managers; 1060 University, 800 Place Victoria, 1080 Beaver Hall Hill

One of the most valuable assets to many organizations is the aggressively competitive, hard-driving and ambitious employee. However, although these attributes benefit the company, they may exact a personal toll. For example, recent studies have shown that these individuals are twice as prone to coronary heart disease.

Several major companies, such as Metropolitain Life and A.T. & T., have implemented stress management courses, such as physical fitness and behavior modification, which are designed to lessen the strain and tension on employees while still maintaining their positive work attributes.

Here in Montreal, a government financed research project, along these lines, is being conducted jointly by Concordia, McGill, and the University of Montreal. This study is an extension of two previous projects, done in 1979, using Bell managers.

The present research will evaluate the effectiveness of these two approaches in reducing coronary risk while maintaining or even increasing, occupational productivity.

The programs are designed for male managers who are currently free from heart disease, but are considered to be at high risk because of their hard-driving life styles.

We are therefore asking for volunteers who would be willing to be screened for participation in this project. The programs, which are free of charge, will take place over a 6 week period beginning around mid February 1982. The physical fitness program will take place at the Ville-Marie Squash Club, for one hour (including change and shower) four times a week, with jogging shoes provided, if necessary. The behavior modification course will be held in the Medical Library, 9th flr. Beaver Hall Hill, three times a week for one hour. Time schedules will be discussed with interested parties.

Appendix A (continued)

Those who wish to participate must agree to attend at least 75% of the sessions, accept random assignment to either a physical fitness or behavior program, and participate in some testing before and after the programs.

All information about individual participants will be held in strict confidence by the Universities. Those who take part, however, may have the results of their own tests on request at the end of the study.

If you are interested in knowing more about this research project, you can contact Dr. D. Pomerantz in the Beaver Hall Medical Department at 870-8153,

Signed by D. Pomerantz

Appendix B

Type A Project Consent Form

I agree to participate in the research project aimed at reducing the risk of heart disease associated with being Type A. I understand that the specific goal of treatment is to reduce physiological reactions to emotional stress. I also understand that while the treatments used are reasonable in the light of current knowledge concerning Type A, stress and heart disease, it is not proven that they do, in fact, modify physiological reactivity and/or coronary risk.

My obligations to the project are:

1. To participate in 2 evaluation sessions (pre and post treatment) similar to the screening session I have already experienced.

2. To accept random assignment to either of the two treatments (stress management or exercise).

3. To fulfill attendance requirements of the group to which I am assigned.

   For the exercise group, this means attendance at a minimum of 18 sessions (of 24 offered) over a 6 week period, plus 2 evaluation sessions.

   For the stress management group, this will be attendance at a minimum of 12 sessions (of 18 offered) over a 6 week period, plus 2 evaluation sessions.

   For both groups, nonattendance duration cannot be longer than one week.

4. To deposit $200 as a guarantee of attendance. This money will be deposited in a special interest-bearing account and refunded to me with interest following the final evaluation session, if attendance obligations were met. If not, the money will be donated anonymously to the Montreal Centre Aide Campaign, and no receipt for income tax purposes will be issued.

Appendix B (continued)

The obligations of the investigators are:

1. To conduct evaluation and treatment sessions with professional competence and ethics.

2. To keep all individual results confidential and only release them to the individual involved.

3. To hold a General Meeting, after all results from the project have been analyzed. At this time, the findings from both groups will be presented and any questions arising from the study will be answered.

---------- ----------

Date Signature

---------- Witness

Please print your name here----------

Appendix C

Informed Consent Form

Informed Consent for Physical and Mental Tasks
As a participant in this experiment, you will perform a graded exercise test on a bicycle ergometer and several mental tasks.

Explanation of Graded Exercise Test on Bicycle Ergometer
The work loads will begin at a level you can easily accomplish and will be advanced in stages depending on your work capacity. We may stop the test at any time because of signs of fatigue, or you may stop when you wish to, because of personal feelings of fatigue or discomfort. We do not wish you to exercise at a level which is abnormally uncomfortable for you. Your heart rate will be continuously monitored throughout this test via electrodes attached to your back.

Explanation of Mental Tasks
These tasks will involve presentation of audiovisual materials as well as answering a variety of test questions. These materials have been designed to elicit both subjective and physiological arousal. Although we would appreciate your cooperation, we will terminate this part of the session at any time, upon your request.

Explanation of Periodic Blood Sampling
This will be done by an experienced registered nurse and will require only a single insertion of a tiny needle at the beginning of the session. Although this should cause you little physical discomfort, we will stop the experiment at any time upon your request.

Risks and Discomforts
There exists the pssibility of certain changes occuring during the tests. They include abnormal blood pressure, fainting, disorders of heart beat, and very rare instances of heart attack. Every effort will be made to minimize these by the preliminary screening and by observations during the testing. Personnel trained in Cardio-Pulmonary Resuscitation will be present to deal with unusual situations should they arise.

Inquiries
If you have any doubts or questions about the procedures used in the graded exercise test or the mental tasks, please ask us for further explanations.

Appendix C (continued)

Freedom of Consent

Your performance of the graded exercise test and the mental tasks is voluntary. You are free to deny consent if you desire.

I have read this form and I understand the test procedures and the possible risks and discomfort involved. I consent to participate in these tests. I know of no medical reason preventing me from participating in this research.

_____	_____
Date	Signature of Subject
	_____
	Witness

Appendix D

Estimated VO2 Max

GROUP EXERCISE

Subject	Pre	Post
23	38.5	41.4
24	32.3	46.6
33	29.8	28.2
58	34.8	48.3
60	51.3	59.1
65	28.0	28.9
80	40.4	43.0
84	41.5	46.9
95	38.9	38.3
96	38.5	40.4
261	44.5	48.7
271	28.3	33.9
661	31.3	38.1

GROUP STRESS MANAGEMENT

Subject	Pre	Post
01	36.8	40.1
02	38.8	37.1
16	41.3	44.1
19	43.8	40.7
29	43.0	41.2
31	27.2	29.6
40	52.1	41.2
45	33.6	36.2
50	63.7	59.3
66	39.1	39.1
321	53.3	52.7

Appendix E

Heart Rate

Basal (B), Stress (S), and Recovery (R) Periods

GROUP EXERCISE

Subject	Pre Session			Post Session		
	B	S	R	B	S	R
23	63	98	75	74	111	75
24	52	73	57	62	68	58
33	74	106	78	85	110	84
58	56	97	61	53	63	50
60	52	64	62	45	57	46
65	72	82	69	52	73	52
80	58	90	60	60	95	65
84	61	103	67	55	95	60
95	50	71	43	49	49	44
96	65	71	63	65	72	62
261	57	82	84	56	67	57
271	65	88	78	62	80	52
661	80	107	90	68	92	83

GROUP STRESS MANAGEMENT

Subject	Pre Session			Post Session		
	B	S	R	B	S	R
01	71	98	70	71	93	74
02	70	95	68	56	92	63
16	84	118	83	72	88	77
19	63	88	67	66	82	60
29	68	91	71	58	104	63
31	55	77	58	56	67	89
40	62	69	59	58	64	59
45	78	89	80	79	84	81
50	50	89	60	53	70	58
66	63	74	70	66	88	65
321	57	79	56	50	71	54

Appendix F

Systolic Blood Pressure

Basal (B), Stress (S), and Recovery (R) Periods

GROUP EXERCISE

Subject	Pre Session			Post Session		
	B	S	R	B	S	R
23	125	149	120	122	154	138
24	120	148	124	114	122	113
33	119	153	119	132	157	139
58	109	137	108	125	132	124
60	119	140	109	122	134	124
65	134	177	149	145	187	148
80	124	173	125	124	167	129
84	110	142	112	104	125	109
95	128	152	129	139	153	135
96	129	157	135	140	148	127
261	133	158	135	139	153	140
271	114	139	114	124	147	127
661	122	139	119	122	143	123

GROUP STRESS MANAGEMENT

Subject	Pre Session			Post Session		
	B	S	R	B	S	R
05	134	163	138	134	157	137
02	127	167	134	129	159	125
16	129	145	118	125	147	135
19	109	130	104	114	128	118
29	122	145	124	124	157	128
31	103	123	105	100	155	83
40	104	118	105	103	118	103
45	119	149	119	115	128	118
50	104	135	108	115	127	114
66	120	135	122	132	150	133
321	112	164	124	117	147	124

Appendix G

Diastolic Blood Pressure

Basal (B), Stress (S), and Recovery (R) Periods

GROUP EXERCISE

Subject	Pre Session			Post Session		
	B	S	R	B	S	R
23	89	100	97	80	98	88
24	74	83	72	59	77	69
33	74	89	69	77	95	84
58	75	84	75	84	85	77
60	75	89	79	62	78	64
69	82	102	90	79	133	79
80	88	108	93	78	102	80
84	60	95	58	63	73	67
95	78	94	79	79	89	78
96	98	122	98	99	109	65
261	80	102	90	88	105	95
271	58	88	55	58	72	59
661	87	102	85	89	102	87

GROUP STRESS MANAGEMENT

Subject	Pre Session			Post Session		
	B	S	R	B	S	R
01	90	104	90	92	109	100
02	77	92	79	82	99	74
16	54	69	60	69	80	65
19	73	83	67	59	69	59
29	74	92	79	77	89	80
31	75	89	79	74	143	68
40	54	73	55	65	72	64
45	72	94	79	74	90	74
50	63	87	60	50	63	49
66	72	85	70	84	95	85
321	77	98	87	73	93	75

Appendix H

Epinephrine

Basal (B), Stress (S), and Recovery (R) Periods

GROUP EXERCISE

Subject	Pre Session			Post Session		
	B	S	R	B	S	R
23	36.00	57.00	42.80	40.52	64.28	71.85
24	32.70	69.60	33.00	23.49	32.29	18.98
33	31.10	89.00	62.50	61.86	78.37	33.95
58	73.70	73.80	40.50	50.14	55.88	13.04
60	34.50	87.00	-	52.50	82.93	53.51
65	16.20	39.70	9.50	58.97	106.41	89.93
80	10.90	32.80	25.00	14.58	35.68	21.71
84	68.10	182.80	70.00	49.04	76.10	46.22
95	64.10	96.50	30.20	54.08	114.54	44.74
96	28.30	48.60	32.50	51.10	57.70	-
261	71.70	53.20	20.30	18.60	64.70	22.50
271	25.00	54.10	20.50	28.30	88.80	8.42
661	42.90	84.20	27.90	54.59	64.76	59.79

GROUP STRESS MANAGEMENT

Subject	Pre Session			Post Session		
	B	S	R	B	S	R
01	22.50	56.90	30.00	18.90	16.20	10.50
02	50.10	71.40	128.00	81.30	166.90	105.00
16	20.60	71.80	63.30	32.60	94.39	72.98
19	39.40	18.80	23.40	48.13	58.42	54.02
29	41.00	122.20	46.10	43.44	142.45	59.65
31	49.40	62.50	26.60	41.29	49.32	46.25
40	24.50	47.60	20.10	83.95	90.24	63.77
45	40.70	124.30	32.90	82.20	78.55	50.77
50	26.60	105.30	39.60	51.26	94.26	82.99
66	29.60	17.90	10.20	17.67	32.37	8.11
321	38.80	144.40	30.00	74.85	113.08	60.51

Appendix I

Norephinephrine

Basal (B), Stress (S), and Recovery (R) Periods

GROUP EXERCISE

Subject	Pre Session			Post Session		
	B	S	R	B	S	R
23	175.7	216.2	219.6	233.8	235.2	311.3
24	264.0	399.8	228.4	293.0	308.0	360.1
33	180.2	316.4	185.8	300.0	323.6	375.8
58	242.0	395.0	319.5	279.1	353.5	143.7
60	500.0	405.8	455.4	267.4	281.2	350.1
65	178.4	230.4	247.2	256.6	296.6	323.1
80	226.2	344.1	208.3	228.9	309.5	207.3
84	251.1	223.3	257.9	225.5	243.8	210.3
95	240.2	243.0	219.3	242.9	220.1	274.5
96	321.2	319.9	290.7	306.7	365.9	174.9
261	348.3	348.3	336.6	242.0	122.4	204.2
271	197.5	242.5	209.9	142.1	195.6	151.0
661	336.2	387.5	298.2	407.4	395.2	346.7

GROUP STRESS MANAGEMENT

Subject	Pre Session			Post Session		
	B	S	R	B	S	R
01	170.0	247.3	371.5	240.0	221.0	244.2
02	122.0	261.2	252.8	274.8	317.7	341.8
16	135.1	294.2	205.5	227.2	320.3	241.5
19	136.0	170.5	215.5	360.1	490.9	327.9
29	322.2	312.3	278.2	222.0	329.0	238.7
31	183.3	338.3	348.1	233.8	255.9	210.4
40	288.9	348.0	275.6	271.2	275.2	277.9
45	234.5	274.1	229.4	227.4	212.2	184.8
50	223.1	332.1	227.5	332.0	381.2	401.6
66	235.8	614.5	380.7	217.0	338.4	196.1
321	233.5	401.3	158.3	365.2	481.9	363.4

Appendix J

Counterbalanced Mental Stress Tasks

C=Stroop Color-Word D=Digit Span

E=General Knowledge Quiz F=Reaction Time

GROUP EXERCISE

Subject	Pre	Post
23	DE	FC
24	CD	EF
33	FC	DE
58	CD	EF
60	EF	CD
65	EF	CD
80	FC	DE
84	EF	CD
95	CD	EF
96	CD	EF
261	FC	DE
271	EF	CD
661	DE	FC

GROUP STRESS MANAGEMENT

Subject	Pre	Post
01	FC	DE
02	EF	CD
16	FC	DE
19	CD	EF
29	EF	CD
31	DE	FC
40	FC	DE
45	CD	EF
50	DE	FC
66	EF	CD
321	DE	FC