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Joseph Kemp Murphy
Louisiana State University and Agricultural & Mechanical College

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STRESS MANAGEMENT AND THE AMELIORATION OF TYPE A BEHAVIOR IN CARDIAC REHABILITATION

The Louisiana State University and Agricultural and Mechanical Col. PH.D. 1985

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STRESS MANAGEMENT AND THE AMELIORATION OF TYPE A BEHAVIOR IN CARDIAC REHABILITATION

A Dissertation
Submitted to the Graduate Faculty of the Louisiana State University and Agricultural and Mechanical College in partial fulfillment of the requirements for the degree of Doctor of Philosophy in The Department of Psychology

by
Joseph K. Murphy
B.A., McGill University, 1976
M.A., Louisiana State University, 1980
December, 1985
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Abstract

The effects of a stress management program upon the Type A behavior pattern were examined in a sample of patients with coronary heart disease who were enrolled in an on-going cardiac rehabilitation program. Based upon exhibition of Type A behavior (defined by the Structured Interview), patients were randomly assigned to three experimental conditions: (a) behavioral stress management, (b) supportive (attention placebo control group), and (c) no treatment (waiting list control group). Patients receiving treatment attended 10 sessions of 1-1½ hours duration, as well as pretreatment and posttreatment assessment sessions. Patients in the no treatment condition also completed the assessments.

The evaluation of treatment outcome was based upon pre- to post-treatment change. Hypotheses that patients in the behavioral stress management condition would demonstrate greater reductions than the supportive and no treatment conditions on measures of the behavior pattern and cardiovascular reactivity during the Quiz Electrocardiogram (QE) and cold pressor were not supported. Changes during treatment were, however, significant for some of the Type A measures (Structured Interview, Framingham Scale, and Bortner Scale) and the QE but did not distinguish between experimental conditions. Cold pressor results demonstrated neither a significant time (pre- to post-treatment) by group (experimental conditions) interaction nor a significant time effect. Secondary variables (lipids, anxiety, and marital satisfaction) demonstrated a similar pattern of results. Results did offer a slight suggestion that the assessment procedures may have had a sensitizing effect which motivated patients to act more appropriately with respect
to the stressors in their lives and that participation in treatment may have assisted patients' daily coping with stress.

As a whole, this intervention program was not effective. The lack of positive outcome is discussed in terms of possible mitigating factors, i.e., a negative association between Type A behavior and severity of coronary artery disease, patients' ages, patients' medication, and patients' prolonged participation in an exercise oriented rehabilitation program. The present results suggest that specific Type A interventions are not necessary for all coronary patients and that the parameters of matching coronary patients with psychological interventions need refinement.
INTRODUCTION

Coronary heart disease (CHD) has been and continues to be the leading cause of death in the United States (Krantz, Glass, Schaeffer, & Davia, 1982; World Health Organization, 1970). The social and economic costs are staggering. Annual mortality estimates range from 600,000 due to CHD (Fishman, 1982) to 1,000,000 due to heart and blood vessel disease (Russell, 1980). As far back as 1963, Felton and Cole estimated that the economic loss from CHD was over four billion dollars annually, excluding financial losses due to early death. Further, approximately 28 million people with cardiovascular disease require continuous medical care or rehabilitation, which has attendant social and financial costs. Considering the magnitude of this problem, research has focused upon identifying factors associated with CHD, e.g., the Framingham Heart Disease Epidemiology Study (Kannel, McGee, & Gordon, 1976) and the Western Collaborative Group Study (Rosenman et al., 1964; Rosenman, Brand, Sholtz, & Friedman, 1976). These and other investigations have led to the identification of CHD risk factors such as sex, age, elevated levels of low-density lipoproteins, cholesterol, and triglycerides, low levels of high-density lipoproteins, diabetes mellitus, hypertension, obesity, smoking, lack of exercise, and a familial history of heart disease (Krantz et al., 1982, Suinn, 1982). However, because reliance

---

1 The terms coronary heart disease (CHD) and coronary artery disease (CAD) are distinguishable, i.e., heart versus arterial disease, but are frequently used interchangeably. CHD is the more inclusive term and the term more frequently used in the literature. This paper will, therefore, use the term CHD unless the respective author makes the distinction or the discussion is focused upon arterial disease.
these factors predicts less than half the incidence of new heart attacks (Jenkins, 1971; Keys et al., 1972), additional risk factors have been sought.

Chief among the potential risk factors that have been examined are psychosocial stress and certain behavior patterns (Krantz et al., 1982). Of the two factors, the behavior pattern, Type A (Friedman & Rosenman, 1959) has received the most consistent support (Friedman et al., 1982; Newlin, 1981). A recent report by Blumenthal (1982) summarizes the contributions of various risk factors to CHD (see Table 1). While the risk factors in Table 1 have been significantly correlated with CHD, their respective contributions are sometimes confounded by associations with other risk factors, e.g., obesity with cholesterol and an inactive lifestyle, or the particular substrate of a risk factor may be in dispute, e.g., high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and very-low-density lipoprotein cholesterol (Abbott, Garrison, Wilson, & Castelli, 1982). Finally, the hypothesis has been offered that all of the risk factors may be secondary to the Type A behavior pattern (TABP) (Friedman, 1979; Friedman et al., 1982). As implied, the evidence for psychosocial stress and, in particular, psychological stress, is not as consistent as the evidence for TABP (Krantz et al., 1982). However, of particular relevance to the present proposal is the implication of acutely stressful life events beyond the individual's control in coronary disease (Glass, 1977; Jenkins, 1971; 1976). In addition, there seems to be an association between the TABP and stress, i.e., the appearance, or elicitation of the Type A pattern
Table 1

Cardiovascular Risk Profile

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Risk Profile</th>
<th>Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;65</td>
<td>++</td>
<td></td>
</tr>
<tr>
<td>45-65</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>&lt;45</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>++</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Family History</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MI &lt;55</td>
<td>+++</td>
<td></td>
</tr>
<tr>
<td>MI &lt;65</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>CVD &lt;65</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>absent</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Cholesterol level (mg %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥270</td>
<td>+++</td>
<td></td>
</tr>
<tr>
<td>240-269</td>
<td>++</td>
<td></td>
</tr>
<tr>
<td>221-239</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>≤220</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Triglyceride level (mg %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥200</td>
<td>++</td>
<td></td>
</tr>
<tr>
<td>151-199</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>≤150</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Blood pressure (mm Hg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;160 (systolic) and/or</td>
<td>+++</td>
<td></td>
</tr>
<tr>
<td>&gt;100 (diastolic)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>140-159 (systolic) and/or</td>
<td>+++</td>
<td></td>
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<tr>
<td>90-99 (diastolic)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>100-139 (systolic) and/or</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>69-89 (diastolic)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;one pack per day</td>
<td>+++</td>
<td></td>
</tr>
<tr>
<td>&lt;one pack per day</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Stopped at least 5 years</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Nonsmoker</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Behavior patterns</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type A (symptomatic)</td>
<td>+++</td>
<td></td>
</tr>
<tr>
<td>Type A (asymptomatic)</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Type B (asymptomatic)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Exercise</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None or some irregular</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>regular aerobic exercise</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td>++</td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>-</td>
<td></td>
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</tbody>
</table>

+++ = severe: ++ = moderate: + mild: - not significant.

from Blumenthal (1982)
under stressful conditions (Glass, 1977). A discussion of these three topics, psychological stress, the Type A behavior pattern (TABP), and their interaction, follows.

Psychosocial stress and coronary heart disease

Psychosocial stress has been implicated as an etiological factor in physical disorders, e.g., cancer (Sklar & Anisman, 1981) and musculoskeletal disorders (Stovya, 1979), and psychiatric disorders (American Psychiatric Association, 1980). Coronary heart disease is not an exception to this conclusion and reviews have concluded that a relationship does exist between stress and CHD. In a recent paper, Razin (1982) concluded that "there are positive associations of some kind between psychosocial factors and CAD development" (p. 363). As might be expected, different psychosocial factors show different degrees of association. The influence of stress appears to be due to the adaptation required to cope with the stressor. Further, the stress is considered to exert a strain upon the individual which may eventually result in organ damage (Blumenthal, 1982). While a thorough review of psychological factors is beyond the scope of this paper, pertinent research will be briefly reviewed. For a more extensive summary, the interested reader is referred to reviews by Glass (1977) and Jenkins (1971; 1976).

For the most part, the examination of the relationship between psychosocial stress and CHD has not proven fruitful except in fairly well delineated areas. Many of the earlier studies examined various demographic variables and, in general, the relationships have not been consistent (Jenkins, 1976; Razin, 1982). A notable exception is the
consistent data concerning national mortality rates. Keys (1970) obtained marked differences in CHD incidence, prevalence, and mortality according to nationality. The seven country survey reported rates of angina, infarction, and death to be three to four times higher in Finland and the United States than in Japan, Greece, and Yugoslavia (Keys, 1970). Clearly though, international comparisons are confounded by genetic and cultural factors. Similarly, reports which have examined socioeconomic factors have obtained positive associations but are frequently flawed by inattention to covarying factors, e.g., financial and occupational promotions usually occur with advancing age which also raises the risk of CHD. In general, results indicate that major changes/differences in residence, occupation, status, mobility, and cultural congruity may affect the rate of CHD. However, the unifying theme of changing CHD rates seems to be that as individuals move into unfamiliar social circumstances or environments for which they have not been prepared, CHD rates increase (Russek & Russek, 1976).

Another area of investigation has been neuroticism and, in particular, anxiety and depression. Several reports have demonstrated differences on the MMPI between patients with coronary disease and healthy individuals (e.g., Bakker & Levenson, 1967; Bruhn, Chandler, & Wolf, 1969; Lebovits, Shekelle, Ostfeld, & Paul, 1967; Ostfeld, Lebovits, Shekelle, & Paul, 1964). The most prominent differences have been on the "neurotic triad," Hs, D, and Hy. Scale means for angina patients were higher than normals and myocardial infarction (MI) patients were lower than normals. An association between anginal pain and hypochondriasis (MMPI) has also been reported by Jenkins (1971). Studies using the 16PF have yielded similar results. Bakker (Bakker,
1967; Bakker & Levenson, 1967) and others have reported increased emotional instability in angina patients compared with MI patients while all of the Coronary disease patients exhibited some emotional problems (Jenkins, 1971; 1976). The experimental evidence, thus, suggests that "neurotic" problems are associated with coronary disease and that, in particular, neuroticism may be linked with morbidity while anxiety and/or depression may be linked with angina (Jenkins, 1976; Razin, 1982). While the preceding studies have been both prospective and retrospective and, thereby, suggestive of coronary risk, results could possibly indicate a subclinical disease process (Friedman, Ury, Klatsky, & Siegelaub, 1974). Consequently, the predictive significance of psychological distress remains an investigative question.

Another area of psychosocial stress which has generated experimental investigation is life changes. Much of this work was prompted by Holmes and Rahe and their examinations of the concomitants of life changes, both positive and negative (e.g., Holmes & Rahe, 1967). A number of retrospective studies have demonstrated an increase in life changes during the six months preceding symptom onset in MI patients (Theorell & Rahe, 1971; Jenkins, 1976). Further, patients perceive stressful events as a major cause of their illness. Blumenthal (1982) reported that 59% of 44 consecutive MI patients viewed stress as significant in their condition. The difficulty with most of these studies is the lack of an adequate control group (Jenkins, 1976). That is, patients serve as their own controls in recalling events in the last six months, as well as, earlier time periods. Thus, life change may be a function of better recent memory than distant memory. The MI may also serve as a sensitizing event with patients searching for reasons for
their MI.

In prospective studies of life change and coronary events, the evidence is mixed. For example, Parkes, Benjamin, and Fitzgerald (1969) followed over 4,000 widowers for nine years after the death of their wives. The mortality due to CHD was 67% above the expected rate for married men of the same age. Opposing results were reported by Theorell, Lind, and Floderus (1975) who followed 6,500 construction workers for 12 to 15 months. Elevated life change scores and MI incidence showed no relationship. In sum, the evidence is promising but mixed. Based upon his review of the literature, Razin (1982) concluded that stressful life events seem linked to general morbidity rather than coronary morbidity. This conclusion echoes Jenkins (1971; 1976) who stated that the evidence is provocative but unconvincing due to inconsistencies between studies.

The preceding review, albeit brief, suggests that psychosocial factors may very well play a role, or serve as risk factors in the genesis of CHD. However, the complexity of factors and the difficulty in separating respective influences has made research difficult. Consequently, a definitive understanding of psychosocial influences is not yet possible (Razin, 1982). Indeed, all of the preceding factors are broadly conceptualized as stressors, e.g., growing up in Western society, being anxious, or having a spouse die. The evidence seems clear that some individuals who experience these stressors do develop heart disease. However, we all experience stress but we do not all develop heart disease. The question would, then, seem to be are some individuals prone to developing CHD in response to stress. The Type A
behavior pattern is the variable, or construct, which has been proposed as mediating the association between stress and CHD.

**Type A behavior pattern and coronary heart disease**

While the TABP was first hypothesized as an etiological factor in the genesis of CHD by Friedman and Rosenman in 1959, they were not the first to recognize the association between behavior and the heart as the following quotations illustrate.

A mental disturbance provoking pain, excessive joy, hope or anxiety extends to the heart, where it affects its temper, and rate, impairing general nutrition and vigor. It is no wonder many serious diseases thus gain access to the body, when it is suffering from faulty nourishment and lack of normal warmth.

William Harvey (1628)

In the worry and strain of modern life, arterial degeneration is not only very common but develops often at a relatively early age. For this I believe that the high pressure at which men live and the habit of working the machine to its maximum capacity are responsible rather than excesses in eating and drinking.

William Osler (1897)

In a group of 20 men (with angina pectoris), every one of whom I knew personally, the outstanding feature was the incessant treadmill of practice; and yet if hard work--the badge of all of our tribe--was alone responsible, would there not be a great many more cases? Every one of these men had an added factor—worry; in not a single case under 50 years of age was this feature absent.

William Osler (1920)

Friedman and Rosenman (1959) first defined the Type A individual as one who exhibits enhanced personality traits of aggressiveness, ambitiousness, competitive drive, is work oriented with preoccupation with deadlines, and exhibits impatience with a strong sense of time urgency. Since 1959, the definition has evolved as a result of
continuing research into the development of the behavior pattern and the pathogenesis of heart disease. Recently, Friedman, Thoresen, and Gill (1981) defined the TABP as follows.

"Type A behavior pattern is an action-emotion complex characterized by a chronic and incessant struggle indulges in by persons who (1) attempt to achieve more and more in less and less time and (2) also frequently harbor a free-floating hostility that is often covert and usually well-rationalized. The sense of time urgency and hostility felt by these persons gives rise to the aggravation, irritation, anger, and impatience (AIAI) so often exhibited by them. Indeed, these latter emotional components form the core of Type A behavior." (p 81)

The behavior pattern is not considered a fixed personality trait which is unalterable. Rather, the behavior pattern is considered to be a learned behavior which is composed of well-established habit patterns. The patterns are often acquired early in life through experience in the home and the individual's cultural experiences (Friedman et al., 1981).

Note should be made that the Type A pattern is neither a stressor nor a response but is, rather, a behavioral style which appears in some people in response to challenging situations (Razin, 1982).

The original formulation of the TABP was based upon Friedman and Rosenman's work as cardiologists at the Harold Bruhn Institute at Mount Zion Hospital in San Francisco. Since 1959, the TABP has been repeatedly shown to be associated with CHD in 12 retrospective studies and seven small scale (relative to the Western Collaborative Group Study and the Framingham Heart Study) prospective studies (Jenkins, 1976). In the Western Collaborative Group Study (WCGS), Rosenman et al. (1964) assessed 3154 healthy (no indications of CHD) middle and upper income men on standard risk factors and the TABP. The men were between the ages of 39 and 59. At two year follow-up, 54 of 1584 Type A men had
developed clinical CHD versus 16 of the 1598 Type B men (Rosenman et al., 1966). The risk ratio for the development of CHD for Type A compared to Type B individuals was 3.4. By the $4\frac{1}{2}$ year follow-up, the risk ratio was 2.7 and the presence of the TABP was the single best predictor of CHD (Rosenman et al., 1970). At $8\frac{1}{2}$ year follow-up, the approximate relative risk was 1.9 for men aged 39 to 49 and 2.2 for men aged 50 to 59 (at entry) (Rosenman, Brand, Sholtz, & Friedman, 1976).

In the Framingham Heart Study similar results were obtained (Haynes, Feinleib, & Kannel, 1980). Over the course of an eight year period, men and women who had initially been free of CHD and classified as Type A were twice as likely to develop heart disease as men and women classified as Type B. The Framingham study involved 1674 coronary free individuals. Further, direct comparison and application of the Framingham predictive equation to WCGS subjects (Brand, Rosenman, Sholtz, & Friedman, 1976) results in a comparable number (to the observed) of CHD events. Thus, the Brand et al. (1976) results represent a cross validation of CHD predictive equations. Brand et al. state that the removal of the risk associated with TABP would correspond to a 31% reduction in CHD incidence in the WCGS.

The association between the TABP and CHD is not solely based upon initial clinical endpoints, e.g., diagnosable MI or death, but has also been examined with respect to arteriographic results and recurrent MI. Several studies have examined the severity of coronary artherosclerosis with arteriography (cardiac catheterization) and documented a positive relationship with the TABP (Blumenthal, Williams, King, Schanberg, & Thompson, 1978; Frank, Heller, Kornfeld, Sporn, & Weiss, 1978; Krantz, Sanmarco, Selvester, & Matthews, 1979; Williams et al., 1980; Zyzanski,
Jenkins, Ryan, Flessas, & Everist, 1976). However, Dimsdale and his colleagues (Dimsdale, Hackett, Catanzano, & White, 1979a; Dimsdale, Hackett, Hutter, Black, & Catanzano, 1978; Dimsdale et al., 1979b; Dimsdale, Hutter, Hackett, & Block, 1981) and Kornitzer et al., (1982) failed to document a positive relationship. The reason(s) for these apparently contradictory results is unclear (Williams et al., 1980), but may relate to population differences (Dimsdale et al., 1979) or to the methods of assessment (Kornitzer et al., 1982). In discussing their results, Kornitzer et al. suggest that their use of the Bortner Scale to assess TABP (see the later discussion of TABP assessment), may account for their negative results. Further, in assessing CAD severity, Dimsdale et al. (1978; 1979; 1981) used a weighting procedure which considers the amount of myocardium with impaired circulation. Studies obtaining a positive relationship be the TABP and disease severity have, most often, simply graded the four major coronary arteries on the degree of occlusion. In sum, five of the nine arteriographic reports support a positive relationship between the TABP and atherosclerosis and methodological differences may mitigate the conclusions of Dimsdale et al. (1978; 1979; 1981) and Kornitzer et al. (1982).

Three studies have examined the role of the TABP in recurrent myocardial infarction (Jenkins, Zyzanski, & Rosenman, 1976; Jenkins, Zyzanski, Rosenman, & Cleveland, 1971; Rosenman et al., 1967) and all reported a positive relationship. The Recurrent Coronary Prevention Project (Friedman et al., 1982; 1984; Powell, Friedman, Thoresen, Gill, & Ulmer, 1984; Thoresen, Friedman, Gill, & Ulmer, 1982) has also demonstrated that reductions in TABP are associated with reductions in
coronary events. In the most recent report from this project, the recurrence rate in patients exhibiting significant reductions in Type A behavior was one fourth that of patients not exhibiting significant reductions, i.e., 1.7% vs 8.6% (Friedman et al., 1984). In addition, Jenkins et al. (1971) reported on a small sample, 17 patients, who died of CHD. The TABP was not significantly related to death but the authors noted that the meaning of their observations, a trend toward increased speed and impatience among the fatal cases, awaits a larger sample of fatal CHD cases. Similarly, Rosenman et al. (1967) reported a non-significant trend of increased incidence of the TABP among MI fatalities compared to survivors.

Despite these rather consistent and impressive results, the TABP is not an area of investigation without controversy. For example, the preceding discussion details the statistical significance of the association between the TABP and CHD while the clinical significance remains to be fully documented. Newlin (1981) points out that the best predictor of recurrent CHD events is a previous history of heart attack and that by itself the TABP results in a population in which less than half eventually develop CHD. While postponing intervention until a heart attack occurs is less than desirable from a prevention standpoint, the wisdom of expending massive resources on non-at-risk populations in an age of public accountability can be questioned. The problem is the degree to which Type A behavior is truly coronary-prone behavior.

In discussing or considering TABP intervention, the reader should be aware that the utility of risk factor intervention of any magnitude or in any CHD risk factor has been questioned (e.g., Corday & Corday,
In addition, large scale risk factor interventions have resulted in either non-significant changes (Multiple Risk Factor Intervention Trial (MRFIT) Research Group, 1982), marginal changes (Stern, Farguhar, Maccoby, & Russell, 1976), or been open to question due to methodological inadequacies (Leventhal, Safer, Cleary, & Gutmann, 1980).

In an attempt to answer the preceding questions and resolve the controversy surrounding CHD and the TABP, the National Heart Lung and Blood Institute (NHLBI) recently sponsored the Review Panel on Coronary-prone Behavior and Coronary Heart Disease (1981). The Review Panel undertook "A comprehensive, impartial and objective review" (p 1199) of the data and concluded that Type A behavior was associated with increased risk of clinical CHD. The risk was considered to be greater than the risk due to age, elevated systolic blood pressure and cholesterol, and smoking. In addition, the relative risk of the TABP is considered to be of the same magnitude as elevated systolic blood pressure and cholesterol, and smoking. Additional portions of the report detail unresolved questions and provide suggestions for continuing research. Of particular relevance to the present proposal are the following Review Panel conclusions: (1) patients with known CHD should be given primary consideration for intervention trials, (2) the risks of such interventions are small and the benefits are potentially enormous, (3) the use of therapeutic interventions in Type A behavior remains a realm for investigation, (4) current information concerning short-term effectiveness is insufficient, (5) the goal of intervention should be a change in level (intensity) of the Type A pattern or a subset of its components, and (6) treatment groups need not be large (Review Panel, 1981).
Stress and TABP interaction

As has been mentioned, stress and the TABP seem to be a particularly potent combination which has received considerable attention in the literature (e.g., Friedman & Rosenman, 1959; Glass, 1978; Krantz et al., 1982). To summarize the experimental evidence, Type A's (a) show heightened achievement strivings, (b) suppress or ignore subjective states like fatigue which either threaten or interfere with their best performance, (c) rapidly pace themselves, (d) become impatient with delays, (e) express hostility and aggression when frustrated in task completion, (f) perform poorly when slow responding is required, and (g) focus on central tasks while ignoring peripheral cues which might deter performance (Glass, 1978; Krantz et al., 1982). These behaviors are thought to represent an attempt to gain environmental control and, thereby, avoid the anxiety which stems from the threat of losing control and not being able to cope. The Type A individual is, thus, engaged in a perpetual struggle for control even under minimally stressful conditions. In comparison, the Type B individual is relatively free from this control struggle and, thereby, free of the Type A characteristics (Glass, 1978). Unfortunately, Type A individuals find themselves in no-win situations for as they master or cope with successive stressors (or challenges), society reinforces their success (e.g., promotions, financial reward, recognition, etc.) and new challenges present themselves.

The Type A's pattern of responding has been elaborated upon by Glass (1978) and is based upon his work with Type A's and uncontrollable stress. Initially, when confronted by a stressor, the Type A shows hyperresponsiveness, presumably, reflecting effort to assert control and
master the situation. Even after experience with the stressor as uncontrollable, Type A's persevere and become even more motivated to assert control. However, after repeated failure, the uncontrollability is perceived and the Type A's become hyporesponsive. A similar pattern is shown by the Type B's but the Type B's are not as persistent in attempting to assert control and do not become as hyporesponsive when the stressor is perceived as uncontrollable. The Type A pattern of hyperresponsiveness followed by hyporesponsiveness is most pronounced under conditions of high stress rather than moderate stress. In other words, when the uncontrollability of stress is highly salient, e.g., loud noise, Type A's will initially respond rapidly, learn that the stress can not be controlled, and become hyporesponsive. Under moderate or low salience conditions, e.g., soft noise, Type A's exert less initial effort, less effectively learn the non-contingency, and fail to exhibit hyporesponsiveness. In addition, repeated exposure to salient, uncontrollable stress seems to result in enhanced susceptibility to hyporesponsiveness, or helplessness, and possible CHD (Glass, 1978).

Given these behavioral changes, the next logical step is the identification of the physiological mechanisms by which the TABP is translated into clinical CHD. While the definitive identification has yet to occur (Razin, 1982; Suinn, 1982), a reasonable hypothesis, which has considerable support, is that Type A behaviors are accompanied by physiological activity which facilitates the atherosclerotic process (Dembroski, MacDougall, Herd & Shields, 1979). While the bulk of the research has been conducted with healthy, young adults (Corse, Manuck, Cantwell, Giordani, & Matthews, 1982), there do appear to be some consistencies (Dembroski, 1981). Type A's, compared with Type B's,
respond significantly more to a wide variety of challenges, e.g., reaction time tasks, TV video games, and the cold pressor, with elevations in systolic blood pressure and heart rate. Studies which have examined CHD populations (e.g., Corse et al., 1982; Dembroski et al., 1979; Sime, Buell, & Eliot, 1980) suggest that CHD may compound the Type A physiological responsivity.

A methodological note of importance is that when the behavior pattern has been assessed with the Jenkins Activity Survey (see following discussion of assessment), physiological relationships have frequently been absent. In comparison, use of the Structured Interview (see following discussion of assessment) has resulted in significant associations between the TABP and physiological reactivity (Corse et al., 1982; Dembroski, 1981). In addition, the attributes of the TABP which are most closely associated with CHD, i.e., the overt, behavioral stylistics, provide the best prediction of physiological reactivity. As previously discussed in summarizing Glass' (1978) work with uncontrollable stress (the A/B distinctions were most prominent under conditions of high stress), the Type A elevations in blood pressure and heart rate were most pronounced under high challenge conditions (Dembroski et al., 1979). In conclusion, it appears that the TABP is frequently associated with heightened physiological responses to environmental challenges. That the association is not invariant is not surprising for the association between the TABP and CHD is far from a perfect correlation (Dembroski, 1981).

While a physiological explanation of the foregoing phenomena is only speculative at present, the evidence suggests that the chronic overactivity of neuroendocrine, lipid metabolism, and physiological
response systems may promote injury to the arterial endothelium (Ross & Glomset, 1976) which may be responsible for atherogenesis (Blumenthal & Williams, 1982). In particular, the sympathetic-adrenomedullary (SAM) system has been implicated as mediating stress, the TABP, and CHD (Krantz et al., 1982). The physiological consequences of increased SAM activity, i.e., increased blood pressure, heart rate, and myocardial oxygen consumption, elevated levels of epinephrine, norepinephrine, and free fatty acids, and increased plasma renin activity, all have the potential to predispose to cardiovascular diseases (Herd, 1978). Within this conceptualization, the catecholamines (epinephrine and norepinephrine), which are released during stress, are thought to play a crucial role via the induction of acute hemodynamic effects, e.g., increases in heart rate and blood pressure and the release of free fatty acids and other lipids (Krantz et al., 1982).

After endothelial injury has occurred (possibly due to hemodynamic or biochemical insult), a lesion forms. This lesion is the atheromatous plaque. Lipids and cholesterol and, possibly, platelets collect about the plaque which interferes with blood supply and leads to gradual enlargement. The end result is the clinical manifestations of CHD, i.e., angina, MI, and sudden death (Krantz et al., 1982). While the preceding discussion of the physiological process is tentative, it is not without support. Further, there is reason to believe that hemodynamic stresses are occurring more frequently and at a greater intensity in Type A individuals (Williams, 1978). The question now becomes one of how this process, i.e., stress, the TABP, and CHD, can be altered and the discussion turns to psychological intervention in the TABP.
Psychological intervention in the Type A behavior pattern

Considering the preceding discussion, intervention in the TABP seems clearly indicated. The following sections will review the assessment of Type A behavior and published investigations of out-patient interventions in the TABP, as well as, interventions with CHD populations. Particular attention will be paid to methodological issues in the past research.

Assessment. As already discussed, the TABP is considered an integrated or over-learned behavior pattern which is elicited by particular stimulus situations or conditions. As presently formulated, these conditions involve challenges or possess a potential for uncontrollability. Consequently, assessment procedures which invoke these conditions are most likely to precipitate the appearance of the TABP. Thus, the Structured Interview (SI) was developed (Rosenman et al., 1964). Since 1964, the SI has been revised and the most recent revision uses a videotaped procedure (Friedman et al., 1981). The questions in the Videotaped Structured Interview, VSI, are reproduced in Table 2 and the scoring procedures in Table 3 (Friedman et al., 1981). The SI results in either a four category classification system of Types A-1 (fully developed TABP), A-2 (incomplete and less extreme TABP), B (a relative absence of TABP), and X (equivalent Type A and B characteristics) or a five category system with category B yielding categories B-3 (a relative absence of TABP) and B-4 (fully developed Type B). However, most research has used a simple A-B dichotomy (Krantz et al., 1982). The scoring of the VSI, a new development designed to remove some of the SI's subjectivity, has not yet seen extensive use. As a reading of the interview and scoring protocol indicates, the VSI is
**Table 2**

Questions asked during videotaped interview

1. Mr. Jones, what is your occupation or profession?
2. Do you think that you are a hard-driving, no-nonsense sort of achiever, or do you believe that you tend to do things in a rather leisurely manner?
3. Has your wife ever told you to take things easier or slow down? How does she put it? Does she still have to tell you to slow down?
4. Would your 20-year-old self have been proud or disappointed in what you have accomplished and are at this time? (Expand if necessary.)
5. Do you feel that most of the important parts of your life are now behind you? (Expand if necessary.)
6. What do you think might have been the factors in or causes of your heart attack?
7. Some persons have claimed that they were glad they had suffered a heart attack. Were you? (Expand.)
8. Do you admire and have as much respect and faith in doctors as your father and mother probably had? (Why not?)
9. Do you think your behavior had anything to do with your heart attack? (Expand.)
10. Do you walk/eat fast? After you have finished eating, do you like to sit and dawdle at the table, or do you like to leave and do something else?
11. If there are 5 or 10 persons waiting to eat at a restaurant, would you wait? At a movie theater?
12. Most working people usually arise before 8:30 A.M. on weekdays although they may sleep longer on Saturdays and Sundays. Now in your case, during the weekdays, at what time do you, uh . . . .
13. When you are in the bathroom, do you sometimes do two things at once, such as reading trade or professional journals or washing your teeth as you shower?
14. (After asking about hobbies) When you have taken your black-and-white photographs (expand). . . . Well, after you did take the snapshots, the films, did you develop them? Uh . . . .
15. If you tell someone that you will be somewhere at 2:00 P.M., will you be there?

*Hesitation to determine whether patient interrupts and finishes sentence.

from Friedman et al. (1981)
Table 3: Diagnostic indicators of type A behavior

<table>
<thead>
<tr>
<th>Score</th>
<th>Scale T: time urgency</th>
<th>Scale H: hostility</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A</strong> Psychomotor manifestations</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M1</td>
<td>Characteristic facial tautness expressing tension</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>M2</td>
<td>Rapid horizontal eyeball movements during ordinary conversation</td>
</tr>
<tr>
<td>20</td>
<td>M3</td>
<td>Rapid eye blinking (over 40 blinks per minute)</td>
</tr>
<tr>
<td>20</td>
<td>M4</td>
<td>Knee jiggling or rapid vigorous tapping of fingers</td>
</tr>
<tr>
<td>20</td>
<td>M5</td>
<td>Rapid, frequently dysrhythmic speech involving elision of terminal words of sentences</td>
</tr>
<tr>
<td>15</td>
<td>M6</td>
<td>Lip clicking during ordinary speaking</td>
</tr>
<tr>
<td>15</td>
<td>M7</td>
<td>Rapid, tic-like eyebrow lifting</td>
</tr>
<tr>
<td>10</td>
<td>M8</td>
<td>Head nodding when speaking</td>
</tr>
<tr>
<td>20</td>
<td>M9</td>
<td>Sucking in of air during speech</td>
</tr>
<tr>
<td>20</td>
<td>M10</td>
<td>Humming (tuneless)</td>
</tr>
<tr>
<td>20</td>
<td>M11</td>
<td>Speech hurrying</td>
</tr>
<tr>
<td>15</td>
<td>M12</td>
<td>Tense posture</td>
</tr>
<tr>
<td>15</td>
<td>M13</td>
<td>Motorization accompanying responses</td>
</tr>
<tr>
<td>20</td>
<td>M14</td>
<td>Expiratory sighing</td>
</tr>
<tr>
<td>20</td>
<td>M15</td>
<td>Rapid body movements</td>
</tr>
</tbody>
</table>

**B** Direct behavioral tests (See questions 12, 14, and 19 in Table 1)

<table>
<thead>
<tr>
<th>Score</th>
<th>Scale T: time urgency</th>
<th>Scale H: hostility</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>B1</td>
<td>The interviewer, in posing a question whose answer is already clear from its content, hesitates, becomes laboriously tedious or repetitive, and then stammers. Does the subject interrupt the stammering with an answer?</td>
</tr>
<tr>
<td>10</td>
<td>B2</td>
<td>Same procedures but a second question is employed</td>
</tr>
<tr>
<td>10</td>
<td>B3</td>
<td>Same procedures but a third question is employed</td>
</tr>
</tbody>
</table>

**C** Physiological indicators

<table>
<thead>
<tr>
<th>Score</th>
<th>Scale T: time urgency</th>
<th>Scale H: hostility</th>
</tr>
</thead>
<tbody>
<tr>
<td>25</td>
<td>C1</td>
<td>Periorbital pigmentation</td>
</tr>
<tr>
<td>20</td>
<td>C2</td>
<td>Excessive forehead and upper lip perspiration</td>
</tr>
</tbody>
</table>

**D** Significant biographical content

<table>
<thead>
<tr>
<th>Score</th>
<th>Scale T: time urgency</th>
<th>Scale H: hostility</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>D1</td>
<td>Self-awareness of presence of Type A</td>
</tr>
<tr>
<td>20</td>
<td>D2</td>
<td>Polyphasic activities (e.g., reads while driving, reads while using electric shaver, thinks of other matters during conversation with others)</td>
</tr>
<tr>
<td>20</td>
<td>D3</td>
<td>Walks fast, eats fast, and does not dawdle at table (2 of 3)</td>
</tr>
<tr>
<td>20</td>
<td>D4</td>
<td>Makes fetish of always being on time under all circumstances</td>
</tr>
<tr>
<td>20</td>
<td>D5</td>
<td>Has been told to slow down in working and living habits by spouse</td>
</tr>
<tr>
<td>10</td>
<td>D6</td>
<td>Difficulty in sitting and doing nothing</td>
</tr>
<tr>
<td>10</td>
<td>D7</td>
<td>Subject habitually substitutes numerals for metaphors in speech</td>
</tr>
</tbody>
</table>

Score:

Scale T =

Scale H =

Total =

from Friedman et al. (1981)
designed to measure the stylistics of speech and behavior, in addition to the content of the responses. The stylistics have been shown to correlate more highly with the TABP (Krantz et al., 1982). The SI permits flexibility and is generally recognized as the most valid TABP measure (Blumenthal, 1982; Suinn, 1982). Although a global and somewhat subjective measure, interrater reliability has ranged from 74-90% (Blumenthal, 1982; Newlin, 1981) and test-retest reliability (one year) has been approximately 80% (Newlin 1981). While Rosenman (1977) states that the SI is the only valid technique for assessing the TABP, other measures have been developed and the Review Panel (1981) concluded that two of these, the Jenkins Activity Survey and the Framingham Type A Scale, have demonstrated validity.

The more well known of the two is the Jenkins Activity Survey, the JAS (Jenkins, Rosenman, & Friedman, 1967), which is a self-administered questionnaire originally developed for use in the WCGS. The JAS provides an overall Type A score and three subscales for speed and impatience, hard-driving, and job involvement. The subscales were derived by factor analysis and all four scales are continuous. Test-retest reliability coefficients over a one year period have ranged from .65 to .80 (Newlin, 1981). Because the JAS is a self-report measure, it relies on content and self-perceptions only and, thus, may be subject to distortion. In fact, Friedman (1978) states that Type A individuals often have little insight into their Type A behavior and are often totally inaccurate in their responses to questionnaires. More recently, however, Herman, Blumenthal, Black, and Chesney (1981) reported a significant relationship between self-ratings of Type A characteristics and SI classification. More importantly, the JAS Type A
scale is in agreement with the SI in about 65-70% of all cases (Krantz et al., 1982) and in 90% of cases when considering JAS scores ± one standard deviation from the mean (Newlin, 1981).

As Newlin (1981) notes, the ultimate validity of each measure must rely upon the prediction of CHD events and both measures, the SI and JAS, have been shown to predict CHD endpoints (atherosclerosis and recurrence both retrospectively and prospectively) (Jenkins, 1978). However, despite acceptable concordance rates, correlations between the JAS and the four point SI (A1, A2, B, and X) have been rather low, ranging between .25 and .40 (Krantz, et al., 1982). These results strongly suggest that the JAS and the SI are measuring different components of Type A behavior and that both should, consequently be used in research when feasible.

Among the additional measures of the TABP that have been developed, only one, the Framingham Type A Scale (Haynes, Feinleib, Levine, Scotch, & Kannel, 1978; Haynes et al., 1980) has been found to be a valid predictor of CHD risk (Review Panel, 1981). The Framingham Scale may also prove useful with subject populations dissimilar to the WCGS for the association of the TABP with angina and CHD was as high in women as in men (Haynes et al., 1980). Also noteworthy are the two measures developed by Bortner (1969) and Bortner and Rosenman (1967), one of which is a rating scale and the other a behavioral performance test. Both measures have demonstrated significant correlations with the SI and to distinguish between CHD cases and controls but have not been validated against CHD occurrence (Suinn, 1982). The final Type A measure is the VSI which has received extensive use but only in the Recurrent Coronary Prevention Project (RCPP). As the RCPP has
progressed, the scoring of the VSI has changed from the procedure illustrated in Table 3 to one in which the 38 indicators are weighted on a scale from one to three, reflecting their intensity (Powell et al., 1984). Using dichotomized A-B categories, the VSI classified 83.6% of subjects in agreement with the SI. Satisfactory reliability coefficients, .74-.79, and significant correlations with the Jenkins and Framingham Scales have been reported (Powell et al., 1984). As already discussed, significant reductions in VSI measured TABP have been associated with reductions in coronary events.

In summary, three assessment instruments, the Framingham scale, the JAS, and the SI, have been validated against CHD occurrence and of the three the JAS and the SI have been used most extensively. The VSI, a derivative of the SI, has been validated against the reoccurrence of CHD events. However, the Bortner rating scale (1969) has shown promise and been used in outcome evaluation research (Jenni & Wollersheim, 1978). Considering the ease of administration and scoring of both the Framingham and Bortner scales, there seems little reason to preclude their usage. In fact, a comparison and analysis of the relative contributions of the five measures may help elucidate the association between behavior and CHD. To the author's knowledge, this research has yet to be undertaken.

Non-CHD populations. While Friedman and his coworkers have stated that intervention in non-CHD populations is extremely difficult, if not fruitless (e.g., Friedman, 1979; Friedman et al., 1981), Thoresen, Telch, and Eagleston (1981) have correctly noted that reserving treatment for only post-MI patients is likely offering too little, too late, to too few. Consequently, researchers have attempted to alter the
TABP in clinically healthy populations. In Suinn's review of the literature (1982), he notes that seven studies have attempted to alter Type A behaviors in healthy persons. However, three of the seven are unpublished and, thus, are generally unavailable for critical review. Of the four published studies (Jenni & Wollersheim, 1978; Levenkron, Cohen, Mueller, & Fisher, 1983; Roskies, Spevack, Surkis, Cohen, & Gilman, 1978; Suinn & Bloom, 1978), the report by Jenni and Wollersheim also included several subjects with CHD and is, therefore, discussed in a later section.

All of the treatment programs involved some degree of anxiety management or relaxation training. In particular, Suinn and Bloom (1978) focused on anxiety management with an emphasis on muscle relaxation and covert rehearsal of successful coping while Roskies et al. (1978) focused on relaxation with record keeping. Finally, Levenkron et al. (1983) utilized a more comprehensive approach integrating several self-control procedures, including relaxation. An additional study by Blumenthal, Williams, Williams, and Wallace (1980) evaluated the effects of exercise on the TABP.

As the reader may conclude, summary statements based upon four studies must be tentative. In addition, all four studies suffer methodological shortcomings. The first problem is the limited use of comparison or control groups. Blumenthal et al. (1980) used only a treatment condition, i.e., no controls, and Suinn and Bloom's (1978) report lacks a comparison treatment condition. In contrast, Roskies et al. (1978) used a psychotherapy condition which attempted to provide a corrective emotional experience and Levenkron et al. (1983) compared their comprehensive behavior therapy condition to group support and
brief information conditions. The second shortcoming is the use of the JAS for TABP assessment (Blumenthal et al., 1980; Levenkron et al., 1983; Suinn & Bloom, 1978). The reader will recall that the JAS is, perhaps, not the preferred method of assessment. Conversely, Roskies et al. (1978) used the SI for subject selection but did not evaluate the TABP as an outcome measure. While granting that research into the alteration of the TABP is in its infancy and that researchers are seeking to demonstrate any impact, methodological inadequacy merely prolongs this infancy.

Despite the afore-mentioned problems, the results are hopeful and suggest that the TABP can be altered. Blumenthal et al. (1980) reported significant overall TABP changes for their Type A subjects but not for their Type B subjects. The three JAS factor scales did not demonstrate change. Suinn and Bloom's results also indicated significant change for treated subjects compared to waiting list controls but only on two of the JAS factors, speed and impatience and hard-driving. Roskies et al. (1978) did not report treatment differences but did report changes in both treatment conditions (behavior therapy and psychotherapy) on a measure of time pressure. Similarly, in the Levenkron et al. (1983) report both groups receiving treatment demonstrated similar significant change in the TABP, i.e., overall JAS, JAS hard-driving, JAS job involvement, and Framingham Type A scores. However, only the comprehensive behavior therapy group improved on JAS speed and impatience scores.

Physiological changes which were also noted included significant beneficial changes in cholesterol (Roskies et al.), high-density-lipoprotein (Blumenthal et al.), systolic blood pressure (Blumenthal et
al.; Roskies et al.), diastolic blood pressure (Blumenthal et al.),
triglycerides (Levenkron et al.), and weight (Blumenthal et al.; Roskies
et al.). Levenkron et al. also noted improved heart rate and blood
pressure responses to a stressful subtraction task but improvement was
not related to treatment condition. Unexpected physiological results
included an increase in triglycerides in Suinn and Bloom's treatment
group and an increase in low-density-lipoprotein in Levenkron et al.'s
behavior therapy condition.

A conclusion based upon these reports must, of course, be
conservative but the preceding evidence does suggest that the TABP and
physiological concomitants can be altered in healthy subjects.
However, all interventions, exercise, psychotherapy, and relaxation/
behavior therapy, seem to be equally effective. Because all four
reports have shortcomings (Razin, 1982; Suinn, 1982), further
investigation and replication is clearly indicated.

CHD populations. As already mentioned, Friedman (1979) does not
believe that intervention can achieve any significant success unless
patients have already suffered an infarction. He lists five reasons why
this is so: (a) the TABP is generally considered a prideful possession
by those with the TABP, (b) Type A's have the "other fellow" syndrome,
i.e., others will suffer from CHD but not them, (c) because Type A's are
pragmatic they do not understand how an emotional complex can result in
an actual lesion, (d) almost all cardiologists are Type A's and do not
have the patience to try and eliminate the more observable risk factors,
e.g., diet, smoking, and exercise, and (e) it is not easy to initiate
and maintain an indefinitely prolonged prophylactic regimen that never
demonstrates unequivocally its effectiveness (Friedman, 1979; Friedman
et al., 1981). In the same vein, post-MI patients can have their TABP altered because (a) they can no longer fool themselves that the TABP may lead to CHD in others but not themselves, (b) during the prolonged recovery from the MI, they find time to review their past habits and way of living, and (c) they frequently experience frightening symptoms (e.g., angina, palpitations, dyspnea, and easily induced fatigue) when they yield to Type A behavior (Friedman, 1979; Friedman et al., 1981). While the above statements have a certain face validity, they are based upon observation rather than empirical investigation.

Assuming that the above statements are, in fact, true and that the TABP can be altered in patients who have experienced a cardiac event, what does the experimental evidence indicate? Unfortunately, it is premature for a firm conclusion (Suinn, 1982) and findings should be considered no more than tentative (Razin, 1982). In fact, many of the recent statements concerning the TABP have been prescriptive rather than evaluative (e.g., Friedman, 1979; Friedman et al., 1981; Gentry, 1978; Suinn, 1978) or have been review articles lamenting the paucity of adequate research (e.g., Doehrman, 1977; Frank, Heller, & Kornfeld, 1979; Razin, 1982; Suinn, 1982; Thoresen et al., 1981; Williams, 1981).

The available published research of psychological interventions in the TABP is summarized from a methodological standpoint in Table 4. Nondata based publications have also appeared in the literature (e.g., Bloom, 1979; Hoebel, 1976; Mone, 1970) but will not be discussed due to their lack of data and, therefore, the difficulty in drawing conclusions. Four methodological problems are immediately apparent from Table 4: (a) in no case was intervention compared with a credible attention/placebo control condition and a no treatment condition, (b) in
no case were patients controlled for the severity of CHD, (c) in only one report (Ibrahim et al., 1974) was patient assignment entirely random, and (d) in only two studies (Jenni & Wollersheim, 1978; RCPP [e.g., Friedman et al., 1984; Powell et al., 1984]) were subjects evaluated for changes in the TABP. However, Jenni and Wollersheim elected to use the Bortner scale as an outcome measure, although patients were selected with the SI. The reader will recall that the Bortner scale is not a preferred method for TABP assessment (Review Panel, 1981). In the RCPP, the VSI was used at entry and the three year follow-up. Thus, the RCPP stands alone in assessing the TABP as an outcome measure though the demonstrated validity of the VSI is limited. An additional problem with CHD interventions is attrition which is most often discussed indirectly, if at all. For example, Suinn (1974; 1975) and Suinn, Brock, and Edie (1975) make no mention of attrition. Attrition to the final data collection point seems to be between 20% and 25%, but may be as high as 50% as occurred in Bilodeau and Hackett's (1971) study. Additional attrition rates include the following: 10 of 42 (Jenni & Wollersheim, 1978), 8 of 34 (Roskies et al., 1979), 7 (4 drop-outs and 3 deaths) of 61 (Rahe, Ward, & Haynes, 1979), 4 of 16 (Adsett & Bruhn, 1968), 19 of 118 (Ibrahim et al., 1974), and 325 of 1035 (Friedman et al., 1984). To Friedman et al.'s credit only 77 of their patients were unavailable for the three year follow-up. As Table 4 illustrates, virtually the only common theme among the 11 studies is that at least some of the participants had some CHD symptomatology which was predominantly an MI. As already stated concerning the non-CHD studies, inadequate methodology seems to reflect the state of the field,
Table 4

Patient Characteristics of Interventions with CHD Samples

<table>
<thead>
<tr>
<th>Study</th>
<th>Controlled</th>
<th>CHD Variables or Type A Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adsett &amp; Bruhn (1968; also Bruhn et al., 1971)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>post-MI</td>
<td>Sex (male), married, &lt;55 yo, ≥1 yr post-MI, problem adapting to cardiac status, high drive &amp; frustration. Controls: age, sex, &amp; race.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Bilodeau &amp; Hackett (1971)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>post-MI</td>
<td>Sex (male), &lt;55 yo</td>
<td></td>
</tr>
<tr>
<td>(first)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Thockloth et al. (1973)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>post-MI</td>
<td>&lt;60 yo, sex (male), ≥2 wks as in-patient, work status</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Ibrahim et al. (1974)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>post-MI</td>
<td>History or ECG of MI, hospitalized in ICCU, 35-65 yo</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>
Table 4 (cont.)

<table>
<thead>
<tr>
<th>Study</th>
<th>Population</th>
<th>Pretreatment Analyses</th>
<th>Type A Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suinn (1974)</td>
<td>post- card</td>
<td>?*</td>
<td>Yes</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>No</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(Method?)</td>
</tr>
<tr>
<td>Suinn (1975)</td>
<td>35 post- MI 2 CAD</td>
<td>?</td>
<td>Yes</td>
</tr>
<tr>
<td>Suinn et al. (1975)</td>
<td>Same as Suinn (1974)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rosenman &amp; Friedman (1977)</td>
<td>? . Sex (male)</td>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>Jenni &amp; Wollersheim (1979)</td>
<td>7 Ss with Sex (male), high history of Type A heart attack</td>
<td></td>
<td>SI</td>
</tr>
<tr>
<td>Naismith et al. (1979)</td>
<td>post-MI &lt;60 yo, sex (male), diagnosis</td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>Rahe et al. (1979; also 1973, 1975)</td>
<td>post-MI (first) Age, smoking, height, weight, Norris index, location of infarct</td>
<td></td>
<td>No</td>
</tr>
</tbody>
</table>
Table 4 (cont.)

<table>
<thead>
<tr>
<th>Study</th>
<th>Population Description</th>
<th>Pretreatment Analyses</th>
<th>Entry</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roskies et al. (1979)</td>
<td>7 Ss with CHD symptoms on stress test</td>
<td>Type A, 39-59 yo, full time job, &gt;$25,000 income, no medication affecting outcome medications, sex (male)</td>
<td>SI</td>
<td>No</td>
</tr>
<tr>
<td>Friedman et al. (1982; 1984; Powell et al., 1984; Thoresen et al., 1982)</td>
<td>post-MI &lt;65 yo, 26 mos. post-MI, 26 mos. no smoking, non-diabetic</td>
<td>No</td>
<td>VSI</td>
<td>VSI</td>
</tr>
<tr>
<td>Langosch et al. (1982)</td>
<td>post-MI (?) Sex (male), in-patient</td>
<td>medically cleared</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

* can not be determined from publication
Table 5
Treatment Characteristics of Interventions with CHD Samples

<table>
<thead>
<tr>
<th>Study</th>
<th>Conditions</th>
<th>Initial N</th>
<th>Age</th>
<th>Random Assignment</th>
<th>Duration</th>
<th>Treatment Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adsett &amp; Bruhn (1968; also Bruhn et al., 1971)</td>
<td>Group therapy</td>
<td>10</td>
<td>47.5</td>
<td>?*</td>
<td>10</td>
<td>9 yrs.</td>
</tr>
<tr>
<td></td>
<td>No treatment</td>
<td>6</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dropouts</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bilodeau &amp; Hackett (1971)</td>
<td>Group therapy</td>
<td>10</td>
<td>?</td>
<td>Random Selection</td>
<td>12</td>
<td>None</td>
</tr>
<tr>
<td>Thockloth et al. (1973)</td>
<td>Planned Rehab.</td>
<td>50</td>
<td>?</td>
<td>Yes</td>
<td>?</td>
<td>3-18 mos.</td>
</tr>
<tr>
<td></td>
<td>No Rehab.</td>
<td>50</td>
<td>?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>No treatment</td>
<td>60</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suinn (1974)</td>
<td>Cardiac stress</td>
<td>10</td>
<td>?</td>
<td></td>
<td>5</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>mgmt training</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(CMST)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>No treatment</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 5 (cont.)

<table>
<thead>
<tr>
<th>Study</th>
<th>Conditions</th>
<th>N</th>
<th>Age</th>
<th>Assignment</th>
<th>Sessions</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suinn (1975)</td>
<td>CMST</td>
<td>10</td>
<td>52.1</td>
<td>?</td>
<td>5</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>CSMT (replication)</td>
<td>17</td>
<td>52.6</td>
<td></td>
<td>5</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>No treatment</td>
<td>10</td>
<td>53.7</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suinn et al. (1975)</td>
<td>Same as Suinn (1974)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>group therapy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(18 mos.)</td>
</tr>
<tr>
<td>Jenni &amp; Wollersheim (1979)</td>
<td>CSMT</td>
<td>14</td>
<td>42.1</td>
<td>Yes</td>
<td>6</td>
<td>6 wk.</td>
</tr>
<tr>
<td></td>
<td>Cognitive-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Behavioral</td>
<td>14</td>
<td>42.6</td>
<td>(CHD:No)</td>
<td>6</td>
<td>6 wk.</td>
</tr>
<tr>
<td></td>
<td>No treatment</td>
<td>14</td>
<td>42.8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Naismith et al. (1979)</td>
<td>Intensive Rehab.</td>
<td>76</td>
<td>50.5</td>
<td>Yes</td>
<td>?</td>
<td>6 mos.</td>
</tr>
<tr>
<td></td>
<td>Standard Rehab.</td>
<td>77</td>
<td></td>
<td></td>
<td>?</td>
<td>6 mos.</td>
</tr>
<tr>
<td>Rahe et al. (1979; also 1973; 1975)</td>
<td>Group therapy</td>
<td>22</td>
<td>46.9</td>
<td>Yes</td>
<td>6</td>
<td>4 yr.</td>
</tr>
<tr>
<td></td>
<td>No treatment</td>
<td>22</td>
<td>51.2</td>
<td>Yes</td>
<td>6</td>
<td>4 yr.</td>
</tr>
<tr>
<td></td>
<td>Group therapy</td>
<td>17</td>
<td>48.5</td>
<td>No</td>
<td>6</td>
<td>3 yr.</td>
</tr>
<tr>
<td></td>
<td>(replication)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Conditions</td>
<td>Initial</td>
<td>Random</td>
<td>Treatment Follow-up</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------------------</td>
<td>-----------------------------</td>
<td>---------</td>
<td>--------</td>
<td>---------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Roskies et al. (1979)</td>
<td>Behavioral (CHD Ss)</td>
<td>7</td>
<td>49.5</td>
<td>No</td>
<td>14</td>
<td>6 mos.</td>
</tr>
<tr>
<td></td>
<td>Behavioral (no CHD)</td>
<td>14</td>
<td>48.3</td>
<td>Yes</td>
<td>14</td>
<td>6 mos.</td>
</tr>
<tr>
<td></td>
<td>Psychotherapy (no CHD)</td>
<td>13</td>
<td>46.9</td>
<td>Yes</td>
<td>14</td>
<td>6 mos.</td>
</tr>
<tr>
<td>Friedman et al. (1982; 1984; Powell et al., 1984; Thoresen et al., 1982)</td>
<td>Cardiologic counseling</td>
<td>270</td>
<td>53.4</td>
<td>Yes</td>
<td>24 in 3 yrs.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Type A Behavioral counseling</td>
<td>614</td>
<td>53.1</td>
<td>Yes</td>
<td>44 in 3 yrs.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No treatment</td>
<td>151</td>
<td>54.2</td>
<td>No</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Langosch et al. (1982)</td>
<td>Stress Mngmt.</td>
<td>46</td>
<td>48.1</td>
<td>No</td>
<td>8</td>
<td>6 mos.</td>
</tr>
<tr>
<td></td>
<td>Relaxation</td>
<td>43</td>
<td>50.4</td>
<td>No</td>
<td>8</td>
<td>6 mos.</td>
</tr>
<tr>
<td></td>
<td>No Treatment</td>
<td>30</td>
<td>49.1</td>
<td></td>
<td></td>
<td>-</td>
</tr>
</tbody>
</table>

* can not be determined from publication
i.e., investigators have sought to demonstrate any impact upon CHD patients. However, the time seems ripe for a methodologically sound investigation before intervention strategies become more and more complex.

Despite the preceding shortcomings, research with CHD patients does suggest that intervention can be successful but the most effective type of intervention has yet to be identified. However, a major problem in comparing outcomes across studies is that the majority of studies have developed their own self-report measures and it is not clear how these measures correlate with standardized Type A assessment and CHD (Suinn, 1982). Obviating this problem is the use of clinical CHD endpoints but only four studies were of sufficient duration to report recurrent CHD events (Adsett & Bruhn, 1968; Friedman et al., 1984; Ibrahim et al., 1974; Rahe et al., 1979). All but Adsett and Bruhn reported that treatment reduced the occurrence of CHD events, i.e., reinfarction. However, comparisons of treatment conditions can only be made in Friedman et al. who reported that the behavioral counseling was more effective than cardiologic counseling with a three year cumulative cardiac recurrence rate of 7.2% vs 13.2%, p<.005. The behavioral counseling group was also more effective than the no treatment comparison condition, a recurrence rate of 14%.

The evidence concerning other than CHD endpoints is less consistent. As Table 4 indicates, only two studies assessed the TABP as an outcome measure. Jenni and Wollersheim (1978) reported significant changes on the Bortner scale but only among high Type A's (presumably, Type Al on the SI but not defined) and only in the cognitive behavioral treatment condition. In the most recent RCPP report (Friedman et al.,
significant reductions in the VSI measured behavior pattern had occurred among active participants in both treatment conditions with the behavioral condition evidencing significantly greater reductions than the cardiologic counseling condition. These results were corroborated by questionnaires completed by participants, spouses, and work colleagues and were generally robust even when considering all initially enrolled participants. Thus the RCPP offers the strongest evidence, at present, that the behavior pattern can be successfully ameliorated.

Physiological changes have also been reported, but problems exist with the interpretation of the results. For example, Suinn (1975) reported "substantial" changes in lipid (cholesterol and triglycerides) levels in the cardiac stress management training (CSMT) conditions but did not report significance levels. In addition, weight loss in the CSMT conditions, versus weight gain in the no treatment condition, may have accounted for these differences. Additional studies have reported a decrease in cholesterol (Adsett & Bruhn, 1964; Roskies et al., 1979; Friedman et al., 1984), no change in cholesterol (Ibrahim et al., 1974; Rahe et al., 1979), and an increase in cholesterol (Jenni & Wollersheim, 1978). Similar contradictory results have been reported for blood pressure with Roskies et al. reporting beneficial change and Adsett and Bruhn, Ibrahim et al., and Jenni and Wollersheim reporting no changes. Friedman et al. (1984) reported reductions in hypertension and congestive heart failure but attributed these changes to a different classification format at follow-up. Friedman et al. also reported a significantly greater incidence of angina in the cardiologic condition compared to the behavioral condition. This difference was not present at entry. No differences in drug ingestion, arrhythmias, or bypass
surgeries were noted between the two conditions. Interestingly, none of the preceding investigations challenged subjects with a stressor and evaluated subject response. The reader will recall that responding to stressful situations with Type A behavior is hypothesized to be critical to the evolution of CHD. The fact that subjects have not been challenged is even more curious considering the intervention emphasis upon relaxation and stress management.

In summary, a review of the published literature to date does not allow a conclusion to be made concerning the efficacy of psychological interventions in CHD populations. Possibly, intervention does have an impact upon future CHD events but the short-term results of short-term treatment programs are open to speculation. While intervention may have an impact upon the TABP (e.g., Jenni & Wollersheim, 1978; Friedman et al., 1984), methodological inadequacies and a multiplicity of individually developed outcome measures preclude any firm statements.

**Problem**

As the preceding literature review indicates, the Type A individual responds to stressful or challenging situations in a manner which places him or her at risk for the development of coronary heart disease. Or if CHD is already present, the likelihood of a recurrent CHD event is increased in the individual who manifests the Type A behavior pattern. The present author believes that the research evidence supports this conceptualization and, also, believes that Friedman's (1979) statements that the TABP is more easily modified in CHD patients (than asymptomatic non-patients) are accurate. Therefore, this study was designed to determine if a methodologically sound behavioral intervention can affect the TABP and its cognitive, behavioral, and physiological concomitants.
As already mentioned, various authors (e.g., Friedman, 1979; Friedman et al., 1981; Gentry, 1978; Thoresen et al., 1981; Suinn, 1982) have hypothesized upon an effective treatment intervention. To briefly summarize, a multi-modal rather than single method approach was utilized in order to maximize the likelihood of effecting a positive change (Suinn, 1982). As outlined by Gentry (1978), the treatment program focused upon (a) changing the antecedents of Type A behavior, e.g., altering schedules to avoid situations that elicit Type A behaviors, (b) changing personal responses to stressful situations, e.g., replacing Type A behavior with relaxation and altering cognitive behavior, and (c) changing the consequences of behavior, e.g., overt and covert reinforcement for non-Type A behavior and overt and covert response cost for Type A behavior. There was, also, an emphasis upon homework and self-monitoring of individual patient progress in order to insure both skills acquisition and generalization (Friedman, 1979; Friedman et al., 1981).

The behavioral treatment program for stress management was compared with a credible attention/placebo condition and a waiting list control condition. The attention/placebo condition was equated with the stress management condition for number of sessions and duration of sessions. The format of the attention/placebo condition was non-directive group therapy with an emphasis on discussion and catharsis rather than skills acquisition. The group leaders were non-directive and acted as group facilitators.

The present study, also, represented a major improvement over past studies of CHD patients (see Table 4) through (a) the use of complete random assignment to all experimental conditions, (b) the use of the Structured Interview for assessment (with patient assignment to
conditions from stratified blocks) and outcome, and (c) secondarily controlling for the severity of CHD through the use of an index of coronary artery disease and the systolic ejection fraction, a measure of left ventricular heart function (the reader is referred to the method section for a further discussion of these measures). Hypothesis 1 predicted that participation in the behavior stress management program would effectively alter the TABP as assessed by the SI. Hypothesis 1 was formally stated as:

Hypothesis 1: Among the three experimental conditions, alteration of the TABP, as assessed by the SI, will be superior among patients receiving the behavior stress management program.

While additional measures of the TABP (the Bortner and Framingham scales and the JAS) were utilized no specific hypotheses were offered as these measures have either not been demonstrated to be as predictive of initial and recurrent CHD events, i.e., the JAS, or have had limited use, i.e., the Bortner and Framingham scales. In addition, no hypotheses were offered with respect to the therapeutic outcome of either the group therapy condition or the no treatment condition. Although the group therapy condition was not considered to be an inert treatment condition, previous studies have reported similar conditions to either resemble the active treatment condition or the no treatment condition. Thus, no hypotheses specific to the group therapy and no treatment conditions were offered.

As the preceding discussion of stress and the TABP indicated, the Type A individual becomes particularly aroused in stressful, challenging situations. However, previous interventions have not examined the possibility that intervention had/did not have an effect upon patients' response to stress. Therefore, this investigation utilized two
stressors to examine the actual efficacy of the stress management program in handling stress. The first stressor was the Quiz Electrocardiogram (QE) developed by Schiffer, Hartley, Schulman, and Abelmann (1976). The QE, delivered in a tape recorded format, consists of 35 questions resembling intelligence quotient test items. Heart rate and blood pressure values during the quiz have shown significantly greater elevations in business executives with angina compared with executives without angina (Schiffer et al., 1976) and in post-MI patients compared with non-CHD controls (Sime, Buell, & Eliot, 1980). The second stressor was a cold pressor task involving immersion of the patient's hand into an insulated bucket of ice water, 1°-3°C. As with the stress quiz, the cold pressor task has been shown to reliably produce heart rate and blood pressure elevations (e.g., Dembroski et al., 1979). During both stressors, heart rate and blood pressure were monitored.

Hypothesis 2 predicted that patients who participated in the behavioral stress management program would demonstrate improved cardiovascular performance in response to both stressors while subjects in both control conditions would fail to demonstrate improved performance. Hypothesis 2 was formally stated as follows:

Hypothesis 2: Patients who participated in the behavioral stress management program will demonstrate improved cardiovascular performance, i.e., lower heart rate and blood pressure elevations, on both the stress quiz and cold pressor task compared to the two control conditions.

While additional dependent measures were examined, prior investigations have been so contradictory that hypothesized outcomes were precluded. Thus, this study was designed to determine if a behavioral stress management program would have a demonstrable effect upon the Type
A behavior pattern in diagnosed CHD patients and if the stress management program could demonstrably alter CHD patients' response to stressful events.
Method

Subjects

Subjects, henceforth identified as patients, were patients selected from the active participants in the Veterans Administration Cardiac Rehabilitation Program in Augusta, Georgia. Recruitment was done on-site via posted notices and brief discussions of the proposed program before and after regularly scheduled exercise activities. Inclusion criteria were: (a) current participation in the Cardiac Rehabilitation Program, (b) permission of the attending cardiologist, and (c) signing of informed consent (see Appendices 1, 2, and 3). Interested patients were urged to speak with the investigator at greater length and if they continued to be interested in participation, they were asked to schedule a pretreatment interview with the investigator. During the interview, the program was thoroughly discussed, informed consent was obtained, and pretreatment assessment completed.

A total of 52 patients were assessed for the TABP. This patient sample represented 65% of the patients on the cardiac Rehabilitation rolls who had attended at least one exercise session during the two week period.

Some elaboration of criterion "a" is pertinent. While all volunteer Rehabilitation patients were assessed, those patients exhibiting the greatest degree of the TABP were given priority for participation, and only those patients exhibiting the TABP were considered in data analyses. Friedman et al., (1981) reported that among 50 randomly selected post-MI patients the mean VSI score was 287 (s.d.=90 and range=135-575). A score greater than 88 indicated some degree of the TABP. In a corresponding manner, and SI rating of A1, A2, or X indicates a preponderance (A1 and A2) or even mix (X) of Type A characteristics. Therefore, patients classified as B3 or B4 were eliminated from data analysis.
period during which the interviews were conducted. These patients also constituted 83% of the rehabilitation programs' regular attendees, i.e., attended at least four of six exercise sessions during this same two week period. Thus, the patient sample was representative of those patients who were sufficiently motivated to maintain a physical activity level that might potentiate the progression of their CAD. Among the 52 patients, 47 (90% of the sample) were rated as Type A1, A2, or X (see Figure 1; Murphy, 1984) and 5 patients (10% of the sample) were rated as Type B3 or B4. These five patients were eliminated from the data analysis. In addition, one pilot patient received the treatment program individually and one patient withdrew from participation prior to completion of the pretreatment assessment. These two patients were also eliminated from data analyses. Thus, the final sample consisted of 45 patients.

Additional patient characteristics are shown in Table 6. As can be seen in the Table, the average patient was a married older gentleman who had a high school education. He had been retired for 6-7 years and had spent approximately half of this time in the rehabilitation program. With respect to CAD, patients had significant disease but relatively normal ejection fractions. Almost all patients had sustained an MI and a majority had received bypass surgery. Despite multiple medications, most patients continued to be symptomatic, e.g., angina, shortness of breath. With respect to traditional risk factors, most patients had stopped smoking, were moderately overweight, had a positive family history of cardiovascular disease, and had been diagnosed as hypertensive. In sum, this patient sample was not healthy and had experienced the consequences of heart disease for many years.
Figure 1. Patient distribution by the Structured Interview.

Percent Patients

STRUCTURED INTERVIEW

A1 A2 X B3 B4
Table 6

Values for Patient Variables at Pretreatment

<table>
<thead>
<tr>
<th>Variable</th>
<th>Behavioral</th>
<th>Supportive</th>
<th>Waiting List</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>14</td>
<td>16</td>
<td>15</td>
</tr>
<tr>
<td>Age (mean)</td>
<td>58.7 (7.5)</td>
<td>61.5 (4.9)</td>
<td>60.2 (5.9)</td>
</tr>
<tr>
<td>Married (n)</td>
<td>12</td>
<td>15</td>
<td>13</td>
</tr>
<tr>
<td>Education^a (mean)</td>
<td>4.1 (1.7)</td>
<td>4.2 (1.6)</td>
<td>4.0 (2.0)</td>
</tr>
<tr>
<td>Retired (n)</td>
<td>12</td>
<td>14</td>
<td>13</td>
</tr>
<tr>
<td>Years Retired (mean)</td>
<td>6.1 (3.3)</td>
<td>6.8 (3.9)</td>
<td>7.0 (4.2)</td>
</tr>
<tr>
<td>Smoking^b (mean)</td>
<td>3.5 (2.5)</td>
<td>2.0 (1.9)</td>
<td>2.3 (1.5)</td>
</tr>
<tr>
<td>Weight (mean)</td>
<td>191.2 (25.2)</td>
<td>181.3 (23.8)</td>
<td>184.4 (29.9)</td>
</tr>
<tr>
<td>% Overweight (mean)</td>
<td>28.3 (15.0)</td>
<td>22.7 (12.7)</td>
<td>24.4 (16.6)</td>
</tr>
<tr>
<td>Years in Rehabilitation (mean)</td>
<td>3.6 (2.0)</td>
<td>3.5 (2.1)</td>
<td>4.6 (2.5)</td>
</tr>
<tr>
<td>Cardiac Catheterization (n)</td>
<td>13</td>
<td>13</td>
<td>15</td>
</tr>
<tr>
<td>CAD Score (mean)</td>
<td>22.2 (8.6)</td>
<td>20.7 (9.1)</td>
<td>22.4 (8.7)</td>
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</table>

(table continues)
<table>
<thead>
<tr>
<th>Variable</th>
<th>Behavioral</th>
<th>Supportive</th>
<th>Waiting List</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ejection Fraction (mean)</td>
<td>63.5 (8.0)</td>
<td>67.3 (13.5)</td>
<td>59.9 (16.2)</td>
</tr>
<tr>
<td>Myocardial Infarctions (n)</td>
<td>12</td>
<td>15</td>
<td>14</td>
</tr>
<tr>
<td>Artery Bypasses (n)</td>
<td>8</td>
<td>7</td>
<td>10</td>
</tr>
<tr>
<td>Medication: Beta Blockers (n)</td>
<td>13</td>
<td>15</td>
<td>13</td>
</tr>
<tr>
<td>Calcium Channel (n)</td>
<td>6</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Diuretics (n)</td>
<td>8</td>
<td>11</td>
<td>6</td>
</tr>
<tr>
<td>Diagnosed Hypertension (n)</td>
<td>10</td>
<td>11</td>
<td>6</td>
</tr>
<tr>
<td>Positive Family History (n)</td>
<td>6</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Presently Symptomatic (n)</td>
<td>10</td>
<td>13</td>
<td>10</td>
</tr>
</tbody>
</table>

Note. Standard deviations are enclosed in parenthesis.

\(^a\)Code: 1 (0-4 years); 2 (5-8 years); 3 (some high school); 4 (high school graduate); 5 (trade or business school); 6 (some college); 7 (college graduate); 8 (post-graduate).

\(^b\)Code: 0 (never); 1 (stopped (\(\geq\)10 years); 2 (stopped 1-9 years); 3 (stopped <1 year); 4 (<5/day); 5 (5-9/day); 6 (10-19/day); 7 (20-29/day); 8 (\(\geq\)30/day).
Therapists

The experimenter served as the primary therapist for both treatment conditions and was assisted (in both conditions) by a clinical nurse specialist with extensive experience with cardiac rehabilitation patients. In order to control for unintentional bias, i.e., the experimenter leading both groups, a procedural check (see Appendix 4) was used to assess treatment credibility (Kazdin, 1979). In addition, videotapes of the second and last sessions were made for both groups. Following the conclusion of treatment, the videotapes were played for the staff of the Rehabilitation program. The staff then rated each treatment program using the credibility questionnaire.

Experimental Design

Patients were randomly assigned from stratified blocks according to scores on the VSI to three experimental conditions: (a) behavioral stress management, (b) group psychotherapy which was also intended to control for non-specific treatment variables, and (c) no treatment control condition (see Table 7). All qualified patients were assigned. As previously mentioned, an attempt was made to evaluate the severity of CHD. Severity was examined by the use of the ejection fraction and an index of the severity of CAD. The ejection fraction is a method for evaluating myocardial performance which is determined by coronary arteriography (Dodge & Sandler, 1974). The formula for the ejection fraction is \((\text{EDV}-\text{ESV})/\text{EDV}\) where \(\text{EDV}\) is the end-diastolic volume (i.e., the left ventricular volume at the end of expansion) and \(\text{ESV}\) is the end-systolic volume (i.e., the left ventricular volume at the end of contraction). Ejection fractions for normal adults show a narrow range of variation about a mean of .67, while an ejection fraction below .50
Table 7

Study Protocol

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Week</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
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<tr>
<td>Recruitment</td>
<td>X</td>
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<tr>
<td>Review Reports of</td>
<td></td>
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<td>Catheterization</td>
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<td>Structured Interview</td>
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<tr>
<td>Paper and Pencil</td>
<td></td>
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<tr>
<td>Questionnaires</td>
<td>X</td>
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<tr>
<td>Blood Work</td>
<td>X</td>
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<tr>
<td>Stress Quiz</td>
<td>X</td>
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<tr>
<td>Cold Pressor</td>
<td>X</td>
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<tr>
<td>Randomization</td>
<td>X</td>
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<tr>
<td>Group Meetings&lt;sup&gt;a&lt;/sup&gt;</td>
<td></td>
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</tbody>
</table>

<sup>a</sup>: 10 meetings over 11 weeks due to holidays.
is usually considered abnormal and indicative of myocardial disease (Dodge & Sandler, 1974). Patient arteriograms were also be examined to determine the degree of occlusion in the four coronary arteries (see Figure 2 which is a schematic of the four coronary arteries) which encircle the heart and provide the heart with oxygen and nutrients. The arteriographic report (see Figure 3), which is computerized, was scored using a scoring system which parallels the report (Conti, 1977), i.e., normal = 0, less than 25% = 1, 25 - 49% = 2, etc. The overall degree of CAD for the four arteries was represented by a cumulative score for the 15 arterial branches. The use of the arteriographic report in this manner, i.e., ejection fraction and CAD severity, would, hopefully, help clarify the discrepant results correlating the TABP with severity of coronary occlusion.

**Treatment Procedure**

The proposed intervention program for both treatment conditions consisted of 10 weekly treatment sessions of approximately 1½ hours duration over a ten week period. Assessment was performed prior to the first session and following the last session. This protocol is shown in Table 7.

The behavioral stress management program (SMP) consisted of structured teaching, demonstrations, in-session practice, and handouts. Patients also received homework (inter-session practice) and self-monitored their adherence to program procedures. As outlined in Table 8, the SMP began with an overview of Type A behavior, stress, the interaction of the TABP and stress, and how Type A behavior and stress might be altered/managed. The overview proceeded to a discussion of individual patient problems which were targeted for self-monitoring.
### Figure 2

**CORONARY ARTERIOGRAM**

![Diagram of coronary arteries]

1. Right coronary artery
2. Left coronary artery
3. Left anterior descending artery
4. Circumflex artery

### Figure 3

**Arteriographic Report of Arterial Occlusion**

<table>
<thead>
<tr>
<th>Branch</th>
<th>Normal</th>
<th>Gives Collaterals</th>
<th>Small</th>
<th>25%</th>
<th>50%</th>
<th>75%</th>
<th>90%</th>
<th>99%</th>
<th>100%</th>
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<td>CIRC</td>
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Table 8
Behavioral Stress Management Program

Session:

Pre-treatment Interview: Brief Description and Informed Consent
  Attendance Contracts
  Structured Interview
  Stress Tests
  Paper and Pencil Tests
  Blood Work

1: Introduction: Type A Behavior, Stress and Interaction
  Alteration/Management of Type A Behavior/Stress
  Problem Identification
  Self-monitoring (throughout treatment)

2: Review of Self-monitoring
  Introduction to Problem Solving

3: Further Problem Solving
  a) Altering stimulus conditions

4: Introduction to S-O-R Model and Relaxation

5: Further Relaxation
  a) Condensation of relaxation procedures

6: Further Relaxation
  a) Imaginal stressful events

7: Introduction to Cognitive Processes
  a) Self-instruction
  b) Irrational thoughts

8: Further Cognitive Processes
  a) Thought stopping

9: Introduction to Reinforcement and Punishment
  a) Overt and covert procedures

10: Further Reinforcement and Punishment
  a) Contingency contracting
  Wrap-up and Congratulations
during the week. At session two, the self-monitoring records were thoroughly reviewed to stress their importance, insure accuracy, and promote continued adherence. This review led into a discussion of how to solve problems using examples provided by the group. The emphasis was upon stimulus conditions which elicit Type A behaviors/stress. The focus upon problem solving continued to session four. At session four, the S-O-R model of behavior was formally introduced (patients had been monitoring S-O-R variables) and progressive relaxation training (Bernstein & Borkovec, 1973) began. Relaxation was demonstrated and practiced by the patients. Homework consisted of twice a day practice of at least 15 minutes duration. Sessions five and six consisted of further relaxation training with condensation of the procedures (seven muscle groups, four muscle groups, and recall) and imaginal presentation of stressful events. Beginning with session seven and continuing through session eight, cognitive skills were emphasized. In particular, emphasis was placed upon self-instruction, alteration of irrational beliefs, and thought stopping. Weekly treatment sessions concluded with sessions nine and ten and discussion of overt and covert contracting.

The group therapy condition was designed primarily as an alternative treatment condition and secondarily as an attention/placebo control condition to control for non-specific factors such as therapist attention, group support, and participation in a psychological treatment program. The focus was upon the discussion of emotional issues which patients had experienced in the past or were currently experiencing. The format was non-directive and particular recommendations for dealing with stress were not made (nor were homework assignments utilized).
Therapists did not initiate discussions of methods of behavior change and when patients initiated such discussions, the therapists did not become involved. Rather, the therapists reflected the discussion back to group members.

The no treatment condition patients underwent the pretreatment and posttreatment assessments concurrently with the treated patients. Following the end of the weekly treatment sessions, the control patients were offered a relaxation treatment program.

Measures

All dependent measures were obtained at pretreatment and posttreatment (see Table 7). Primary dependent measures were: (a) the SI, (Rosenman, 1978) the VSI (Friedman et al., 1981) for the assessment of the TABP 3, as well as, the JAS (Jenkins et al., 1967), the Framingham scale (Haynes et al., 1978), and the Bortner scale (Bortner, 1969), and (b) physiological responses, i.e., heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP), to the stressors. Instructions for the Quiz Electrocardiogram (QE) and the cold pressor emphasized the challenging nature of the stressors, task difficulty, and the need for concentration/will power. Heart rate and blood pressure were monitored with an automatic oscillometric device (Model ASD400, Automated Screening Devices, Inc., Costa Mesa, CA). During both stressors, two measurements of heart rate and blood pressure were made

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3 The experimenter was assisted by Edward McCranie, Ph.D., in the administration and scoring of the SI. Both Dr. McCranie and the experimenter have received training from Drs. Margaret Chesney and Ray Rosenman in the SI. In addition, Dr. McCranie will assist in determining inter-rater reliability of the Type A assessments. The VSI was scored according to VSI criteria (Friedman et al., 1981) and SI criteria (Rosenman, 1978).
during baseline and return to baseline (cessation of the stressor). During the stress quiz, blood pressure and HR recordings were made at approximately four minute intervals resulting in two recordings during the quiz. During the cold pressor, the first recording was made 30 seconds after hand immersion and proceeded at one minute intervals as long as the patient kept his hand immersed. The first return to baseline recording was made one minute after completion of the stressor and the last recording was made three minutes after completion of the stressor.

Instructions for the QE and the cold pressor were presented in a standardized fashion. The actual QE was presented on a tape recorder. For the cold pressor task, the patient was instructed to insert his right hand to a point two inches above the wrist into an insulated cooler of ice water. A plastic mesh screen prevented contact between the ice and the patient's hand. The water was circulated prior to each test. Patients were seated for both procedures.

Secondary dependent measures included: (a) lipoprotein fractions, i.e., very-low-density lipoprotein (VLDL), low-density lipoprotein (LDL), and high-density lipoprotein (HDL), total cholesterol, and triglycerides (Blood analyses were performed by the V.A. with lipid fractions being determined electrophoretically and cholesterol and triglycerides enzymatically.), (b) the state and trait scales of the State-Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970), and (c) the Locke-Wallace Marital Adjustment Scale (Locke & Wallace, 1959). Ancillary measures included: (a) a weekly checklist of stressful events and subjective responses to the events (Brantley, 1980, see Appendix 5), (b) assessments of program utility, and (c) monitoring
of weight for any concomitant change which might affect the posttreatment lipid results.

**Data Analyses**

The first analyses to be performed were condition comparisons of patients' pretreatment characteristics, i.e., age, education, marital status, employment status, time since unemployment, additional risk factor status (percent overweight, hypertension, smoking, familial history of CHD, and blood variables), ejection fraction, severity of CAD, beta-blockade medication (cf., Krantz et al., 1982), and TABP score on the SI. One way analyses of variance (ANOVA) were used for the comparisons of continuous variables and $X^2$ for comparisons of discrete variables. Clearly, non-significant differences were desired. Had significant differences occurred, analysis of covariance would have been used in subsequent comparisons with the significantly different measure used as the covariate.

The second analysis involved a t-test of the overall score on the treatment credibility questionnaire which was completed by participants and the rehabilitation staff. Again, a non-significant difference was desired.

A final analysis prior to the examination of treatment effects was a correlational (Pearson product moment) determination of the inter-relationship among the various Type A measurements and CHD, i.e., the ejection fraction and index of CAD severity. The relationships between the five measures of the TABP and CHD were of particular interest because previous evidence is quite contradictory. Consequently, no hypothesis was posited for this analysis.
**Treatment effects.** The efficacy of treatment and the evaluation of Hypothesis 1 was examined by a multivariate analysis of variance (MANOVA) of the pretreatment to post-treatment change scores on the Type A measures by the treatment condition. When a significant effect was obtained, as hypothesized, (the time [pretreatment and posttreatment] by group [behavioral, supportive, and waiting list] indicated the hypothesized effect), ANOVA's and Duncan's multiple range tests were performed to assess the magnitude and direction of changes. In these analyses, and all other analyses, the alpha level for acceptable statistical significance was selected to be $p<.05$. The reader will recall that Hypothesis 1 predicted a significant decrease in SI score for the behavioral stress management condition but not for the other two conditions.

The analysis of Hypothesis 2, which involved physiological responses (SBP, DBP, and HR) to a psychological and a physical stressor, used a MANOVA of Treatment Conditions by Time by Periods (baseline, stressor, and return to baseline). Baseline and return to baseline values were the two values obtained by the BP monitor from each period. Similarly, stressor values represented the values obtained during the application of the stressors. Univariate analyses were performed if significant results were obtained. Hypothesis 2 predicted that the behavioral stress management patients would evidence diminished physiological reactivity upon post-treatment testing. The group therapy and no treatment conditions would not evidence diminished reactivity.

Further analyses examined changes in the other dependent variables over the course of treatment, i.e., the additional Type A assessments, lipoprotein fractions, cholesterol, triglycerides, the State-Trait
Anxiety Inventory, the Locke-Wallace Marital Inventory, and the weekly stressful events questionnaire. As with the procedure outlined for the patient characteristics, one way ANOVAs of change scores were used to determine if the treatment condition affected outcome. Due to the inter-relationships among the lipid variables a MANOVA was used for the lipid analysis. Significant differences were followed by ANOVA's and Duncan's multiple range tests, as appropriate.
Results

Reliability of patient assessment

A total of 52 patients were assessed for the Type A behavior pattern using the VSI. A random sample of 21 of the 52 interviews was rated for reliability by another trained interviewer using the five categories of SI classification, i.e., Al, A2, X, B3, and B4. The reader will recall that a classification as Al represents rather pure demonstration of Type A behaviors and B4 represents the absence of Type A behaviors. Categories A2 and B3 represent a predominance of Type A or Type B characteristics and Type X individuals demonstrate a relatively even mix of Type A and Type B characteristics. With 15 interviews, there was no discrepancy in classification and with four interviews, there was a discrepancy of one category. Thus, the overall reliability of interview ratings was 91%. At posttreatment, another random sample of interviews, 19, was selected for independent rating. This time, there was no discrepancy with 10 interviews and a discrepancy of one category with eight interviews yielding an overall reliability of 95%. A sample of the paper and pencil Type A questionnaires was also independently scored at pre- and posttreatment with 100% reliability.

Behavior pattern assessment

The distributions of patients' scores on the Type A measures, i.e., SI, VSI, JAS, Framingham, and Bortner Scales, were highly dependent upon the particular Type A measure (Murphy, 1980; Murphy, Norman, Lee, Boineau, & Somes, 1985) as shown in Figures 1, 4, 5, and 6 and Table 9.

4 In training interviewers, Chesney and Rosenman consider a rating within one classification category as accurate, e.g.; an Al individual rated as Al or A2 is accurately rated.
In Figures 4-6 (Figure 1 is discussed in the preceding Method section), the indicated means and standard deviations were obtained in this sample of 52 patients (see Table 9). The seven patients excluded from data analysis are included in the Figures and the Table to enhance the range of scores and comparability with Figure 3. Each bar in the Figures constitutes the percentage of patients whose score was within \( \frac{1}{2} \) standard deviation of scores for the means and respective standard deviations, e.g., in Figure 4, 30% of the patients had a score between 163 and 122 (142.3 ± 20.4) on the Time urgency scale of the VSI. As seen in the Figures, scores on all of the Type A measures except the SI showed relatively normal distributions. With the SI the patient distribution was skewed toward a Type A classification. Scores on the VSI, though normally distributed, also indicated a preponderance of Type A behavior, as did the Framingham Scale. In contrast, mean scores on the JAS and Bortner Scale showed shifts toward a Type B classification, i.e., JAS (Type A and factor scales) scores of 0 indicate an even mix of Type A and Type B characteristics and mean Bortner scores of 178 were obtained from interview defined Type B's (Bortner, 1969). Table 9 also suggests that work status had little effect upon Type A scores, i.e., JAS scores of employed patients (JAS-C) were comparable to the scores of unemployed patients (JAS-N).

To further examine the relationships between the measures of Type A behavior, test scores were intercorrelated (see Table 10). In general, the interview procedures (SI and VSI) were significantly intercorrelated and the paper and pencil measures were significantly intercorrelated. The Speed and Impatience Scale of the JAS correlated significantly with both the interview and paper and pencil measures and the JAS Job
Figure 4. Patient distribution by the Videotaped Structured Interview.
Figure 5. Patient Distribution by the Jenkins Activity Survey

- dark bars: TYPE A
- white bars: SPEED & IMPATIENCE
- gray bars: INVOLVEMENT
- hashed bars: HARD-DRIVING & COMPETITIVE
Figure 6. Patient distribution by the Framingham Scale and the Bortner Scale.
Table 9

Mean Patient Values on Type A Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>n</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
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</thead>
<tbody>
<tr>
<td>VS1: Scale T (Time Urgency)</td>
<td>52</td>
<td>142.31</td>
<td>40.83</td>
<td>70-240</td>
</tr>
<tr>
<td>Scale H (Excess Competitiveness and Hostility)</td>
<td>52</td>
<td>53.17</td>
<td>39.01</td>
<td>0-710</td>
</tr>
<tr>
<td>Total</td>
<td>52</td>
<td>195.48</td>
<td>66.58</td>
<td>95-385</td>
</tr>
<tr>
<td>JAS-N(^{a}): Type A</td>
<td>45</td>
<td>2.53</td>
<td>10.15</td>
<td>19.2-21.5</td>
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<tr>
<td>Factor S (Speed and Impatience)</td>
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<td>1.14</td>
<td>10.22</td>
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</tr>
<tr>
<td>Factor J (Involvement)</td>
<td>45</td>
<td>-13.04</td>
<td>7.55</td>
<td>26.6-10.9</td>
</tr>
<tr>
<td>Factor H (Hard-Driving and Competitive)</td>
<td>45</td>
<td>-0.68</td>
<td>9.97</td>
<td>17.2-27.0</td>
</tr>
<tr>
<td>JAS-C(^{a}): Type A</td>
<td>5</td>
<td>3.06</td>
<td>13.51</td>
<td>13.3-28.6</td>
</tr>
<tr>
<td>Factor S (Speed and Impatience)</td>
<td>5</td>
<td>1.22</td>
<td>7.14</td>
<td>6.6-7.6</td>
</tr>
<tr>
<td>Factor J (Job Involvement)</td>
<td>5</td>
<td>-8.88</td>
<td>11.59</td>
<td>18.4-10.9</td>
</tr>
<tr>
<td>Factor H (Hard-Driving and Competitive)</td>
<td>5</td>
<td>-1.24</td>
<td>12.46</td>
<td>15.4-12.8</td>
</tr>
<tr>
<td>Framingham</td>
<td>50</td>
<td>.50</td>
<td>.23</td>
<td>.1-.97</td>
</tr>
<tr>
<td>Bortner</td>
<td>50</td>
<td>180.70</td>
<td>41.59</td>
<td>105-334</td>
</tr>
<tr>
<td>SI: (Speech Stylistics: A1=1 to B4=5)</td>
<td>52</td>
<td>2.08</td>
<td>1.10</td>
<td>1-5</td>
</tr>
</tbody>
</table>

\(^{a}\)Negative JAS scores indicate Type B behavior.
Table 10

**Intercorrelations among Type A Measures**

<table>
<thead>
<tr>
<th></th>
<th>VSI-T</th>
<th>VSI-H</th>
<th>JAS-A</th>
<th>JAS-S</th>
<th>JAS-J</th>
<th>JAS-H</th>
<th>Framingham</th>
<th>Bortner</th>
</tr>
</thead>
<tbody>
<tr>
<td>SI</td>
<td>.79**</td>
<td>.62**</td>
<td>.26</td>
<td>.32*</td>
<td>.23</td>
<td>.20</td>
<td>.23</td>
<td>.26</td>
</tr>
<tr>
<td>VSI-T</td>
<td>.39**</td>
<td>.37**</td>
<td>.42**</td>
<td>.15</td>
<td>.22</td>
<td>.25</td>
<td>.30*</td>
<td></td>
</tr>
<tr>
<td>VSI-H</td>
<td>.26</td>
<td>.37**</td>
<td>.12</td>
<td>.12</td>
<td>.09</td>
<td>.18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>JAS-A</td>
<td></td>
<td></td>
<td>.73**</td>
<td>.05</td>
<td>.64**</td>
<td>.39**</td>
<td>.63**</td>
<td></td>
</tr>
<tr>
<td>JAS-S</td>
<td></td>
<td></td>
<td></td>
<td>.08</td>
<td>.49**</td>
<td>.48**</td>
<td>.51**</td>
<td></td>
</tr>
<tr>
<td>JAS-J</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.06</td>
<td>.10</td>
<td>.12</td>
<td></td>
</tr>
<tr>
<td>JAS-H</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.49**</td>
<td>.58**</td>
<td></td>
</tr>
<tr>
<td>Framingham</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.41**</td>
</tr>
</tbody>
</table>

* p < .05; ** p < .01.
Involvement scale failed to demonstrate a significant association with any of the other measures. Thus, the two types of assessment procedures, interview or paper and pencil, do not seem to demonstrate strong agreement based upon correlational analyses. To further examine this possibility, the agreement between measures in classifying patients as Type A or not Type A was evaluated. Using the SI as the criterion, patients were considered Type A if they received a classification of A1 or A2. Classifications of X, B3, or B4 were considered to be not Type A. The remaining Type A measures provide continuous numerical scores and patients were dichotomized at the mean score of each measure. For purposes of these analyses, patients scoring above the mean were considered Type A and patients scoring below the mean were considered not to be Type A. Results (see Table 11) indicated that classification agreement ranged from a low of 54% on JAS Type A to a high of 84% on VSI Time Urgency. The proportion of agreements was tested as being significantly different from chance (.5) using a z-test (normal approximation of binomial distribution). Agreement with the SI was significantly better than chance for the VSI, the Framingham Scale, and the Bortner Scale.

Type A and CHD

A correlational analysis of scores of the various Type A measures and the two measures of heart disease (see Table 12) indicated that the behavior pattern was not significantly related to either the severity of artery disease or the ejection fraction (Murphy et al., 1985). In fact, correlations were sometimes negative and, thereby, suggesting that Type A behavior in this patient sample was associated with reduced arterial occlusion and enhanced left ventricular functioning. However,
Table 11
Agreement in Behavior Pattern Classification

<table>
<thead>
<tr>
<th>Measure</th>
<th>A (n=32)(^b)</th>
<th>Not A (n=20)(^c)</th>
<th>% Agreement</th>
</tr>
</thead>
<tbody>
<tr>
<td>VSI-T</td>
<td>23</td>
<td>20</td>
<td>83%**</td>
</tr>
<tr>
<td>VSI-H</td>
<td>27</td>
<td>27</td>
<td>65%*</td>
</tr>
<tr>
<td>JAS-A(^d)</td>
<td>16</td>
<td>11</td>
<td>54%</td>
</tr>
<tr>
<td>JAS-S(^d)</td>
<td>19</td>
<td>11</td>
<td>60%</td>
</tr>
<tr>
<td>JAS-J(^d)</td>
<td>19</td>
<td>10</td>
<td>58%</td>
</tr>
<tr>
<td>JAS-H(^d)</td>
<td>17</td>
<td>14</td>
<td>62%</td>
</tr>
<tr>
<td>Framingham(^d)</td>
<td>21</td>
<td>12</td>
<td>66%*</td>
</tr>
<tr>
<td>Bortner(^d)</td>
<td>19</td>
<td>14</td>
<td>66%*</td>
</tr>
</tbody>
</table>

\(^a\)Sample divided at mean patient score. \(^b\)Types A1 and A2 and above mean score of second measure. \(^c\)Types X, B3 and B4 and below mean score of second measure. \(^d\)Completed by only 50 patients due to one death (Type X) and one dropout (Type B3).

*p<.05; **p<.01.
Table 12

Correlation Coefficients of Type A Measures with CAD Severity

<table>
<thead>
<tr>
<th>TYPE A MEASURE</th>
<th>ARTERY DISEASE</th>
<th>EJECTION FRACTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>SI</td>
<td>.11</td>
<td>-.04</td>
</tr>
<tr>
<td>VSI: SCALE T</td>
<td>-.07</td>
<td>.01</td>
</tr>
<tr>
<td>SCALE H</td>
<td>-.06</td>
<td>-.08</td>
</tr>
<tr>
<td>JAS: TYPE A</td>
<td>.18</td>
<td>-.08</td>
</tr>
<tr>
<td>FACTOR S</td>
<td>.19</td>
<td>.02</td>
</tr>
<tr>
<td>FACTOR J</td>
<td>-.15</td>
<td>.04</td>
</tr>
<tr>
<td>FACTOR H</td>
<td>.08</td>
<td>.09</td>
</tr>
<tr>
<td>FRAMINGHAM</td>
<td>-.01</td>
<td>-.13</td>
</tr>
<tr>
<td>BORTNER</td>
<td>.03</td>
<td>-.09</td>
</tr>
</tbody>
</table>

ALL VALUES NONSIGNIFICANT.
irrespective of the Type A measure, the behavior pattern bore little relationship to CHD and could, at best, explain only 3.6% of the variance in CAD (JAS Factor S) and 1.7% of the variance in ejection fractions (Framingham Scale).

**Pretreatment patient characteristics**

Values for patient characteristics by treatment condition at pretreatment are shown in Table 6 (see Method). Analyses of variance for continuous variables and \( \chi^2 \) for discrete variables indicated that the groups were comparable on all variables. Consequently, covariates were not introduced in the statistical analyses.

**Experimental group comparisons**

Prior to analyzing changes during treatment, groups were compared for the comparability of treatment credibility (see Appendix 4). First, the comparison of the two active treatment conditions was nonsignificant, \( F(1,28) = 1.41; p=.24 \). Next, spouse perceptions of treatment efficacy, including spouses of patients in the no treatment condition, were compared (see Appendix ) and again a nonsignificant effect was obtained, \( F(2,37) = 2.20; p=.124 \). Finally, the perceptions of the professional members of the rehabilitation team, physician, physicians assistant, psychologist, social worker, exercise physiologist, and dietitian, were assessed through video taped segments of the second and final sessions (see Appendix 4). The team members saw both groups as equally credible, \( F(1,10)= 2.24; p=.166 \).

**Attendance.** Attendance at the sessions was variable among the patients. During the treatment period, there were two deaths; one each from the behavioral and waiting list conditions. One death was due to cerebral hemorrhage, secondary to atherosclerosis, and the other was due
to cancer. Patients in the supportive condition attended approximately one more session than those patients in the behavioral condition but this difference was not significant, $p > .05$. The mean number of sessions attended by the supportive and behavioral conditions were 8.2 and 6.9, respectively.

**Type A Assessments.** A multivariate analysis of variance, MANOVA, using the Hotelling-Lawley trace (used with all MANOVA's unless otherwise stated) indicated that the time (pretest to posttest) by group (behavioral, supportive, or waiting list) interaction was not statistically significant, $F(18, 60) = 1.02$, $p = .51$. Thus, the effect of group assignment and treatment did not demonstrably alter Type A behavior. Similarly, the overall group effect was not statistically significant, $F(18, 66) = .72$, $p = .77$. However, the effect of time (pretest to posttest) did show a significant effect, $F(9, 31) = 3.05$, $p = .01$. Subsequent analyses of variance, ANOVA, indicated significant change over time as assessed by the Framingham Scale, $F(1, 39) = 7.65$, $p = .009$, and the Bortner Scale, $F(1, 39) = 4.37$, $p = .043$, as well as the Structured Interview, $F(1, 39) = 6.71$, $p = .013$. As seen in Table 13, the changes in Type A scores were modest and, at times, showed increases rather than decreases in Type A score. For example, with the VSI scores on the Time Urgency Scale increased in both of the active treatment conditions. In contrast, the VSI-T scores of the Waiting List condition decreased from pre- to posttreatment. However, Type A scores generally decreased with the exception of JAS scores, but the decreases of the Waiting List conditions approximated the decreases of the treatment conditions.
Table 13

Mean Scores on Type A Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>Behavioral Pre</th>
<th>Behavioral Post</th>
<th>Supportive Pre</th>
<th>Supportive Post</th>
<th>Waiting List Pre</th>
<th>Waiting List Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>JAS-A&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-2.05</td>
<td>-.63</td>
<td>-5.49</td>
<td>-3.85</td>
<td>-.42</td>
<td>-.42</td>
</tr>
<tr>
<td>JAS-SI</td>
<td>-1.29</td>
<td>-.58</td>
<td>-4.11</td>
<td>-3.45</td>
<td>1.87</td>
<td>3.12</td>
</tr>
<tr>
<td>JAS-I</td>
<td>-11.79</td>
<td>-10.04</td>
<td>-10.85</td>
<td>-12.91</td>
<td>-14.96</td>
<td>-12.05</td>
</tr>
<tr>
<td>JAS-HD</td>
<td>-4.10</td>
<td>-.35</td>
<td>-.21</td>
<td>-.17</td>
<td>1.20</td>
<td>-1.32</td>
</tr>
<tr>
<td>Framingham&lt;sup&gt;b&lt;/sup&gt;</td>
<td>.44</td>
<td>.39</td>
<td>.47</td>
<td>.37</td>
<td>.56</td>
<td>.45</td>
</tr>
<tr>
<td>Bortner&lt;sup&gt;b&lt;/sup&gt;</td>
<td>186.86</td>
<td>180.00</td>
<td>173.88</td>
<td>154.00</td>
<td>184.80</td>
<td>178.64</td>
</tr>
<tr>
<td>VSI-T</td>
<td>142.67</td>
<td>147.50</td>
<td>139.24</td>
<td>142.33</td>
<td>151.67</td>
<td>144.64</td>
</tr>
<tr>
<td>VSI-H</td>
<td>61.67</td>
<td>59.29</td>
<td>52.95</td>
<td>49.00</td>
<td>50.00</td>
<td>59.29</td>
</tr>
<tr>
<td>SI&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.07</td>
<td>3.86</td>
<td>4.06</td>
<td>3.74</td>
<td>4.14</td>
<td>3.72</td>
</tr>
</tbody>
</table>

Note. Higher scores indicate a higher Type A score.

<sup>a</sup>Negative JAS scores indicate Type B behavior.  <sup>b</sup>Significant reductions for all patients which were not affected by treatment condition.
Quiz Electrocardiogram. MANOVA's were again used to analyze the QE, see Figures 7, 8, and 9, and again results showed nonsignificant differences among conditions over the course of treatment, $F(36,40) = .89$, $p = .63$, and for the overall group effect, $F(36,53) = .89$, $p = .64$. Unlike the Type A assessment, however, change from pretreatment to posttreatment was also nonsignificant, $F(18,21) = 1.64$, $p = .14$. Therefore, treatment had little effect on the cardiovascular response to the QE. This result is generally apparent from the corresponding figures which show that all three variables, SBP, DBP, and HR, demonstrated modest decrements from pre- to posttreatment which were quite similar among the three conditions.

Considering SBP (Figure 7), the Behavioral condition demonstrated the greatest decrements during the quiz, 13 mmHg and 18 mmHg during the two quiz measurements, but these changes were not statistically different from the changes in the other two conditions. As shown in Figure 8, DBP decrements were quite similar across the three conditions, e.g., mean decreases of 7.3 mmHg, 8.1 mmHg, and 6.4 mmHg in the Behavioral, Supportive, and Waiting List conditions, respectively, during the first QE measurement. In contrast with the BP changes, HR (Figure 9) showed very little reactivity to either the QE or posttreatment assessment, i.e., HR generally varied from 1-3 bpm.

Cold pressor. The results of the cold pressor assessment are illustrated in Figures 10, 11, and 12. As can be seen in the Figures, the cold pressor was effective in provoking a hemodynamic response. Across all patients, the mean pretreatment response from the end of baseline (second measurement) to the first task response (third measurement) was 20.3 mmHg for SBP, 14.6 mmHg for DBP, and 3.7 bpm for
Figure 7. Systolic blood pressure responses to the QE.

- **BASELINE**
- **QUIZ**
- **RECOVERY**

**BEHAVIORAL**

**SUPPORTIVE**

**WAITING LIST**

- • Pre-treatment
- ○ Post-treatment
Figure 8. Diastolic blood pressure responses to the QE.

- **Behavioral**
  - Baseline: 70 mmHg
  - Quiz: 75 mmHg
  - Recovery: 65 mmHg

- **Supportive**
  - Baseline: 60 mmHg
  - Quiz: 65 mmHg
  - Recovery: 55 mmHg

- **Waiting List**
  - Baseline: 75 mmHg
  - Quiz: 80 mmHg
  - Recovery: 70 mmHg

**Legend:**
- ●●●●●● Pre-treatment
- ○○○○○○ Post-treatment
Figure 9. Heart rate responses to the QE.

HEART RATE (BPM)

BASELINE QUIZ BEHAVIORAL RECOVERY

BASELINE QUIZ RECOVERY SUPPORTIVE

BASELINE QUIZ RECOVERY WAITING LIST

- Pre-treatment
- Post-treatment
HR. However, posttreatment values during this same time period from the end of baseline to the first cold pressor measurement, i.e., SBP and DBP increases of 21.2 mmHg and 13.0 mmHg and a HR increase of 3.2 bpm, were very similar to the pretreatment responses. A MANOVA could not be performed upon the cold pressor results due to missing values, i.e., the final data set included only 17 observations. The reduced subject number is due to subjects removing their hands from the water prior to automatic termination at five minutes. Therefore, ANOVA's were utilized to assess the time by group interaction at each measurement point. None of these analyses attained statistical significance though the third HR during hand immersion approached significance, \( p = .056 \). However, considering the number of analyses performed little, if any, importance should be attached to this result. The effect of time was also consistently nonsignificant, all \( p ' s > .05 \). As with the stress quiz, the figures of SBP, DBP, and HR show that little change occurred in the response to the cold pressor from pre- to posttreatment assessment and that not participating in treatment was as effective as participating in treatment in producing the response changes which did occur.

**Lipids.** Lipid changes over the course of the treatment period are shown in Table 14. As discussed with previous outcome variables, the MANOVA of the time x group interaction failed to show that lipid changes were differentially affected by the treatment conditions, \( F(10,48) = .15, p = .99 \). Again, the MANOVA for time of assessment was significant, \( F(5,25) = 6.56, p < .0005 \). The subsequent ANOVA's for the particular lipids indicated that the LDL change and VLDL change were both significant at posttreatment, \( F(1,29) = 8.49, p = .007 \) and \( F(1,29) = 11.02, p = .002 \), respectively. However, as seen in Table 14, the VLDL change was a
Figure 10. Systolic blood pressure responses to the cold pressor.

NOTE: Hand immersion occurs from the third to seventh measurements
Figure 11. Diastolic blood pressure responses to the cold pressor.

NOTE: Hand immersion occurs from the third to seventh measurements.
Figure 12. Heart rate responses to the cold pressor.

NOTE. Hand immersion occurs during the third to seventh measurements.
Table 14

Mean Lipid Levels by Treatment Condition

<table>
<thead>
<tr>
<th>Condition</th>
<th>Behavioral Pre</th>
<th>Behavioral Post</th>
<th>Supportive Pre</th>
<th>Supportive Post</th>
<th>No Treatment Pre</th>
<th>No Treatment Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol</td>
<td>197.9</td>
<td>204.4</td>
<td>227.4</td>
<td>236.0</td>
<td>195.5</td>
<td>204.8</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>335.9</td>
<td>324.3</td>
<td>227.8</td>
<td>347.9</td>
<td>233.2</td>
<td>231.2</td>
</tr>
<tr>
<td>HDL</td>
<td>16.9</td>
<td>17.4</td>
<td>16.7</td>
<td>17.4</td>
<td>18.6</td>
<td>19.5</td>
</tr>
<tr>
<td>LDL</td>
<td>61.7</td>
<td>65.0</td>
<td>65.1</td>
<td>67.1</td>
<td>64.4</td>
<td>69.7</td>
</tr>
<tr>
<td>VLDL</td>
<td>21.3</td>
<td>19.3</td>
<td>18.2</td>
<td>17.1</td>
<td>17.7</td>
<td>12.4</td>
</tr>
</tbody>
</table>

\(^a\text{mg/dl};\) \(^b\text{% cholesterol (to convert percentages to mg/dl, multiply the cholesterol value by the value of the lipoprotein fraction).}\)
decrease, whereas the LDL change was an increase in value. Thus the treatment program had no demonstrable effect upon the patients' lipid profiles.

Additional outcome variables. ANOVA's of change scores (posttest minus pretest) were used to determine if the three conditions showed differences on these measures. First, changes in marital satisfaction, as measured by the Locke-Wallace Inventory, were not significantly different, $F(2,36) = .22, p = .80$, despite changes which were two to three times greater in the two treatment conditions. The mean respective changes for the behavioral, supportive, and waiting list conditions were 6.1, 7.5, and 2.6. Similarly, changes on the Spielberger State-Trait Anxiety Inventory failed to demonstrate significant differences. Mean group changes on State anxiety were $-2.9, 2.0$ (an increase), and $.5$ (an increase) for the behavioral, supportive, and waiting list, respectively, $F(2,40) = .57, p = .57$. The corresponding Trait changes were $-3.2, .1$ (an increase), and $-2.1, F(2,40) = .54, p = .59$. The Holmes Rahe Inventory of Life Events also showed nonsignificant changes, $F(2,40) = .80, p = .45$. All groups reported increases in stressful life events with means of 46.4, 265.3, and 376.9 for the behavioral, supportive, and waiting list conditions, respectively. Finally, weight changes did not differ reliably among conditions, $F(2,41) = .58, p = .57$. The mean weight changes (in pounds) for the behavioral, supportive, and waiting list conditions were $.7, 2.4,$ and $1.5$ (all increases).

The mean weekly stress ratings which had a range from 0 (no stress) to 10 (stress equal to most stressful week ever had) are shown in Table 15. As seen in the table, stress ratings were within low and tolerable limits and group differences were nonsignificant through the sixth week.
In 10 of the 11 assessment periods, the behavioral condition reported less stress than the supportive condition. This difference was significant during the seventh and eighth weeks. At the final session, both treatment groups reported significantly less stress than the no treatment waiting list condition.
Table 15

Mean Weekly Stress Rating by Treatment Condition

<table>
<thead>
<tr>
<th>Week</th>
<th>Behavioral</th>
<th>Supportive</th>
<th>No treatment</th>
<th>F(df),p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pretreatment</td>
<td>2.85</td>
<td>3.86</td>
<td>4.40</td>
<td>NS</td>
</tr>
<tr>
<td>1</td>
<td>2.33</td>
<td>3.08</td>
<td>N/A</td>
<td>NS</td>
</tr>
<tr>
<td>2</td>
<td>3.44</td>
<td>3.00</td>
<td>N/A</td>
<td>NS</td>
</tr>
<tr>
<td>3</td>
<td>2.33</td>
<td>3.23</td>
<td>N/A</td>
<td>NS</td>
</tr>
<tr>
<td>4</td>
<td>2.67</td>
<td>2.86</td>
<td>N/A</td>
<td>NS</td>
</tr>
<tr>
<td>5</td>
<td>2.80</td>
<td>3.54</td>
<td>N/A</td>
<td>NS</td>
</tr>
<tr>
<td>6</td>
<td>3.00</td>
<td>3.17</td>
<td>N/A</td>
<td>NS</td>
</tr>
<tr>
<td>7</td>
<td>2.11\textsuperscript{a}</td>
<td>3.17\textsuperscript{b}</td>
<td>N/A</td>
<td>5.30(1,19),.03</td>
</tr>
<tr>
<td>8</td>
<td>2.00\textsuperscript{a}</td>
<td>3.38\textsuperscript{b}</td>
<td>N/A</td>
<td>5.61(1,12),.04</td>
</tr>
<tr>
<td>9</td>
<td>1.83</td>
<td>3.00</td>
<td>N/A</td>
<td>NS</td>
</tr>
<tr>
<td>10</td>
<td>2.50\textsuperscript{a}</td>
<td>2.88\textsuperscript{a}</td>
<td>3.93\textsuperscript{b}</td>
<td>4.24(2,39),.02</td>
</tr>
</tbody>
</table>

\textsuperscript{a, b} Different superscripts denote significant, p<.05, group differences (Duncan's Multiple Range Test).
Discussion

Overall, the results of this investigation did not support the hypotheses that a behavioral stress management program would ameliorate Type A behavior and cardiovascular reactivity in this sample of cardiac rehabilitation patients. In fact, the magnitude of the changes which did occur during the treatment were generally similar in the no treatment control group and in the two active treatment conditions. However, results may not have been due to limitations of the behavioral program. Rather, patients in the supportive and waiting list conditions may have benefited from their participation in this investigation and, more importantly, previous and concurrent participation in the rehabilitation program. These ideas are developed in the following discussion.

Change in the Behavior Pattern

Previous investigations (see Introduction) have generally reported successful Type A interventions. In the Recurrent Coronary Prevention Project's (RCPP) latest report (Friedman et al., 1984), 44% of patients in the Type A behavioral counseling condition evidenced a reduction in Type A behavior at the end of three years compared to 25% of the patients only receiving cardiologic counseling. More importantly, the Type A counseling condition experienced a three year cumulative cardiac recurrence rate of 7.2%. The recurrence rate was 13% in the cardiologic counseling condition and the difference between the two conditions was statistically significant, p<.005.

Treatment Considerations. The RCPP results may point up a limitation in the present study, i.e., a treatment program without
follow-up. A lengthy follow-up might have demonstrated treatment effects. While the hypothesis that treatment efficacy could be demonstrated over a prolonged period is tantalizing, this hypothesis does not seem tenable. First, RCPP reports indicated that changes in the TABP were noted prior to changes in morbidity, i.e., significant differences in Type A change were apparent before recurrence rates were significantly different (Powell et al., 1984; Thoresen, Friedman, Powell, Gill, & Ulmer, 1985). Patients showing high TABP change (a standard deviation or more on the VSI score) during the first year had one fourth the cardiac recurrence rate during the second and third years of patients not showing this degree of Type A change (1.7% vs 8.6%, p<.001). Of these high change patients, 91% were in the behavioral condition (Thoresen et al., 1985). Thus, the differences between the RCPP results and the present results may be explicable in terms of differences in the treatment programs. The primary difference seems to involve the duration of treatment and the timing of data analysis. The RCPP involved a one year treatment program after which participants were enrolled in an on-going follow-up program. In addition, treatment results were not analyzed prior to the one year conclusion of treatment. Therefore, comparisons with the RCPP are somewhat inappropriate as changes which occurred during the initial three to four month (comparable to the present study) period are unknown.

However, short-term studies, though often not in patient samples, (e.g., Blumenthal et al., 1980; Levenkron et al., 1983; Lobitz, Brammell, Stoll, & Niccoli, 1983; Suinn & Bloom, 1978) have demonstrated pre- to posttreatment changes in the TABP. Two recent reports of patient interventions (Baer et al., 1985; Razin, 1984) have also
reported TABP changes during a brief treatment period. Unfortunately, these studies are marred by the methodological problems of the studies in Table 4, i.e., different pre to posttreatment assessment of the TABP (Baer et al., 1985) and use of a single group pre-post design (Razin, 1984). None-the-less, the pattern of beneficial TABP change is fairly consistent. These studies used treatment protocols quite similar to the present study and why the discrepancy between the present results and previous investigations occurred does not seem to be explicable in terms of the treatment protocol.

Another possibility may be that the group leaders were ineffective. However, the program was rated as logical and useful by both the program participants and the professional staff of the rehabilitation program. While the efficacy of the group leaders was not directly rated, ineffective or incapable leadership would, presumably, have been reflected in the credibility questionnaire and this was not the case. Therefore differences in treatment procedures do not seem to be an adequate explanation of why significant changes in the TABP were not observed.

Assessment considerations. Another possible explanation for the lack of beneficial TABP change concerns the assessment of the behavior pattern, i.e., perhaps Type A was assessed inadequately or inaccurately. This explanation, also, appears to be inadequate for several reasons. First, the interviews were conducted by an interviewer trained by Rosenman and associates and the behavioral ratings of the interviewer were reliable with the ratings of a second interviewer who had also been trained by Rosenman. Second, the behavior pattern was assessed using several measures of the behavior pattern which should have provided a
rather sensitive test for Type A change. While others (e.g., Byrne, Rosenman, Schiller, & Chesney, 1985; Chesney, Black, Chadwick, & Rosenman, 1981) have demonstrated consistent significant intercorrelations among Type A measures, this study did not (see Table 10). However, these studies employed substantially larger samples, N=468 (Byrne et al.) and N=384 (Chesney et al.), than the present study. More importantly, the general pattern, i.e., interview procedures significantly intercorrelated and self-report questionnaires significantly intercorrelated, and magnitude, i.e., .3 to .8, of the significant correlations in this study are similar to those obtained in previous studies (Byrne et al. 1985; Chesney et al., 1981; Powell et al., 1984). Thus inadequate assessment of the behavior pattern does not seem to be a plausible explanation for the lack of positive results in this study.

Assessment procedures may, however, offer a partial explanation. The reader will recall that the VSI was used as the basis for random assignment of patients. Since Friedman's original exposition of the VSI (Friedman et al., 1981), scoring procedures for the VSI have changed though not drastically (cf Powell et al., 1984). In retrospect, assignment of patients should have been based upon the SI. However, the SI and VSI were significantly correlated and the absence of a perfect correlation can not fully explain the present results.

Type A - CAD considerations. A third explanation for the lack of positive treatment outcome involves the subjects and the relationship between their Type A behavior and their heart disease. The Type A - CAD relationship had shown a nonsignificant negative association (see Table 12) while the majority of previous studies had shown significant
positive associations. Therefore, previous investigations of the Type A - CAD relationship were reexamined and the methods of determining CAD severity were determined (see Table 16; Murphy, Norman, & Somes, 1985). These methods illustrate a wide diversity of CAD assessment to which must be added the method used in this study. Subsequently, the severity of CAD of each patient was recalculated with each scoring system and was correlated with the Type A scores which had been obtained at the pretreatment assessment. These results (see Table 17) demonstrated that Type A behavior was not associated with more severe CAD. In fact, when the behavior pattern correlated in a significant or marginally significant manner, the relationship was an inverse relationship, i.e., high TABP and low CAD or low TABP, i.e., Type B behavior, and high CAD.

This evidence, suggesting that the TABP may not be pathogenetic in this patient sample, may be explicable due to the retrospective nature of the investigation, medication usage, or the age of the subjects. As indicated, patients had been catheterized prior to the Type A assessment, a mean of 3.2 years prior to Type A assessment. However, CAD is most often a progressive disease even in patent bypass grafts (Campeau et al., 1984), CAD regression in humans has been documented infrequently (Hammond, 1983), and the contribution of the behavior pattern to progression or nonprogression has not been reliably documented (Krantz et al., 1979). Thus, there is little reason to believe that sufficient regression had occurred in patients to produce the reported negative correlations. Medication usage, also seems implausible despite reports that beta blockade ameliorates the exhibition of Type A behavior (Schmieder, Friedrich, Neus, Rudel, & Von
### Scoring Systems Used in Assessing the Type A/CAD Relationship

<table>
<thead>
<tr>
<th>Studies</th>
<th>Number of Segments&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Disease Definition&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(occlusion:score)</td>
<td></td>
</tr>
<tr>
<td>1. Friedman et al. (1968)</td>
<td>2(RCA,LCA)</td>
<td>0:0; &lt;25%:1; 25%-&lt;50%:2; 50%-&lt;75%:3; 75%-99%: 100%:5</td>
</tr>
<tr>
<td>2. Frank et al. (1978)</td>
<td>4(RCA,LAC,LAD,CFX)</td>
<td>0-50%:0; &gt;50%:1</td>
</tr>
<tr>
<td>Dimsdale et al. (1979a,b)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Williams et al. (1980)</td>
<td>4(RCA,LCA,LAD,CFX)</td>
<td>0-74%:0; &gt;75%:1</td>
</tr>
<tr>
<td>Zyzanski et al. (1976)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Dembroski et al. (1985)</td>
<td>4(RCA,LCA,LAD,CFX)</td>
<td>0-75%:0; &gt;75%:1</td>
</tr>
<tr>
<td>5. Blumenthal et al. (1978)</td>
<td>4(RCA,LCA,LAD,CFX)</td>
<td>0:0; &lt;75%:1; 75%-99%:2; 100%:3</td>
</tr>
<tr>
<td>Dembroski et al. (1985)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Krantz et al. (1981)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Zyzanski et al. (1976)</td>
<td>4(RCA,LCA,LAD,CFX)</td>
<td>0-&lt;50%:0; &gt;50%:1</td>
</tr>
<tr>
<td>Dimsdale et al (1978)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scherwitz et al. (1983)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Krantz et al. (1979)</td>
<td>4(RCA,LCA,LAD,CFX)</td>
<td>0-69%:0; &gt;70%:1</td>
</tr>
<tr>
<td>9. Kornitzer et al. (1982)</td>
<td>4(RCA,LCA,LAD,CFX)</td>
<td>0:0; &lt;50%:1; 50%-74%:2; several of 50% or one 50%-&lt;90%:3; 90%-&lt;100%:4; 100%:5</td>
</tr>
<tr>
<td>10. Dimsdale et al. (1981)</td>
<td>6(LAD:3;CFX:2;RCA:1)</td>
<td>0-70%:0; &gt;70%:2</td>
</tr>
<tr>
<td>11. Scherwitz et al. (1983)</td>
<td>9 (?)</td>
<td>0:0; &lt;50%:1; 50%-74%:2; 75%-100%:3</td>
</tr>
</tbody>
</table>

<sup>a</sup>RCA: right coronary artery, LCA: left coronary artery, LAD: left anterior descending artery, CFX: circumflex artery.

<sup>b</sup>Disease Definition: 0:0; <25%:1; 25%-<50%:2; 50%-<75%:3; 75%-99%: 100%:5
Table 17

Correlations among Type A measures and CAD scoring systems.

<table>
<thead>
<tr>
<th>Scoring System&lt;sup&gt;a&lt;/sup&gt;</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>SI</td>
<td>-.19</td>
<td>-.22</td>
<td>-.23*</td>
<td>-.26*</td>
<td>-.26*</td>
<td>-.25*</td>
<td>-.21</td>
<td>-.12</td>
<td>-.26*</td>
<td>-.28**</td>
</tr>
<tr>
<td>VSI: Scale T</td>
<td>-.06</td>
<td>-.18</td>
<td>-.17</td>
<td>-.21</td>
<td>-.20</td>
<td>-.18</td>
<td>-.17</td>
<td>-.02</td>
<td>-.22</td>
<td>-.21</td>
</tr>
<tr>
<td>VSI: Scale H</td>
<td>-.19</td>
<td>-.17</td>
<td>-.16</td>
<td>-.09</td>
<td>-.24*</td>
<td>-.29**</td>
<td>-.14</td>
<td>-.12</td>
<td>-.21</td>
<td>-.17</td>
</tr>
<tr>
<td>JAS: Type A</td>
<td>.05</td>
<td>.04</td>
<td>.02</td>
<td>-.04</td>
<td>.09</td>
<td>.07</td>
<td>.02</td>
<td>.17</td>
<td>.06</td>
<td>-.17</td>
</tr>
<tr>
<td>Factor S</td>
<td>.14</td>
<td>.11</td>
<td>.13</td>
<td>.05</td>
<td>.09</td>
<td>.06</td>
<td>.07</td>
<td>.09</td>
<td>.06</td>
<td>-.07</td>
</tr>
<tr>
<td>Factor J</td>
<td>-.01&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-.09</td>
<td>.07</td>
<td>.03</td>
<td>-.06</td>
<td>-.11</td>
<td>-.07</td>
<td>-.07</td>
<td>-.04</td>
<td>-.12</td>
</tr>
<tr>
<td>Factor H</td>
<td>.06</td>
<td>-.01</td>
<td>-.02</td>
<td>-.3</td>
<td>-.10</td>
<td>.12</td>
<td>-.03</td>
<td>.16</td>
<td>-.04</td>
<td>-.28&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Framingham</td>
<td>.08</td>
<td>.08</td>
<td>.06</td>
<td>.16</td>
<td>-.02</td>
<td>.05</td>
<td>.04</td>
<td>.08</td>
<td>.05</td>
<td>-.15</td>
</tr>
<tr>
<td>Bortner</td>
<td>.05</td>
<td>.03</td>
<td>.03</td>
<td>.04</td>
<td>.01</td>
<td>-.01</td>
<td>.01</td>
<td>.08</td>
<td>-.01&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-.17</td>
</tr>
</tbody>
</table>

<sup>a</sup>Scoring systems correspond to the systems identified in Table 1, e.g., scoring system 1 is the system used by Friedman et al. [5] (method used by Scherwitz et al. [17] to divide the coronary arteries could not be determined). <sup>b</sup>Value actually < .01. <sup>c</sup>Identical correlations at different p values are indicative of different degrees of freedom.

* p < .10; ** p < .05.
Eiff, 1983). In the present sample, 92% of the patients were receiving beta blockers. Nonetheless, the SI showed a preponderance of Type A behavior with proportions of patients in each category similar to other prospective reports (e.g., Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985; MacDougall, Dembroski, Dimsdale, & Hackett, 1985) in which medication usage is not likely to have been as widespread. Correspondingly, VSI scores were similar to those reported by Friedman et al. (1981). Therefore, medication usage seems to be an inadequate explanation.

Finally, the subjects' ages may provide the best explanation for the negative correlations. While age failed to significantly correlate with any of the CAD severity scores, a significant correlation with the JAS Type A score was obtained, $r = -0.32$, $p = 0.023$. Our subjects were older, mean age of 60.1, than subjects in previous studies, e.g., mean ages of 47 years (Blumenthal et al., 1978) and 50 (Dimsdale et al., 1981) and reports from the WCGS (Rosenman et al., 1967; 1976) and the Framingham Heart Study (Haynes et al., 1980) have indicated that the significance of the Type A behavior pattern is reduced with advancing age both in terms of initial and recurrent coronary events. Based upon a review of over 2100 angiography patients, Williams (1985) reported that among patients younger than 45, Type A's had more severe CAD than Type B's. Among patients 55 and older, the Type B's, rather than the Type A's, had more severe CAD. Corroborative findings have been reported by Blumenthal and Herman (1985). In particular, subjects younger than 40 years of age perceived themselves as becoming more Type A. In contrast, 40 to 50 year-olds viewed their Type A behavior as unchanging and subjects older than 50 saw themselves as experiencing
significant reductions in their Type A behavior. Similarly, the subjects in the present study may have been particularly hardy Type A's or have passed a "critical" age at which the behavior pattern had a decreasing pathogenetic effect. The hypothesis that older Type A's are hardier than younger Type A's has some support in the research of Kobasa (Kobasa, Maddi, & Zola, 1983). This research has demonstrated that hardiness is inversely related to illness and that Type A's high in hardiness are less susceptible to illness than Type A's who are low in hardiness. Friedman, Hall, and Harris (1985) have also suggested that there may be two categories of Type A individuals which were differentiated on the basis of nonverbal expressiveness. Using subjects from the MRFIT study, one Type A group was found to be healthy, talkative, and charismatic while the second Type A group was found to be repressed, tense, and illness-prone.

Another subject variable that may be important is the period of time that had passed since the patient's cardiovascular events. As shown in Table 9, the mean number of years that the patients had been enrolled in the rehab program ranged from 3.5 to 4.6 years across the three conditions. This prolonged period of participation far exceeds that of other studies and may suggest that this particular patient sample was not an ideal sample. Friedman (1979) suggested that post-MI patients were good candidates for TABP interventions because they find time to review their lives during recovery and experience frightening symptoms. The patients in this sample had probably had more than adequate time to review their lives and become comfortable with their medical status. Studies investigating psychological recovery from an MI (Doehrman, 1977 for a review) have generally shown that the extent
of distress declines as time since the incident increases. Subjects had also become comfortable with their lifestyle, e.g., 40 of the 45 patients were retired and had been so for, on average, over six years and had no intention of returning to work. Furthermore, 33 of the patients regularly experienced coronary symptoms, e.g., angina, shortness of breath, tachycardia, and were rarely fearful of these symptoms. Therefore, the motivation to change may not have been as great as with the patients in other studies.

A perusal of Table 4 indicates that investigators often required patients to be less than a specified age and to have experienced their MI (generally the first MI) within a certain time frame before commencing treatment. Patients in this study almost always exceeded both of these parameters. The potential role of age in affecting treatment outcome can also be seen in the mean ages of subjects listed in Table 5. Ages have generally been between early 40's and early 50's which makes the subjects in previous investigations 10 to 20 years younger than the subjects in the present investigation.

Concurrent therapy (exercise) considerations. A final consideration in explaining the lack of demonstrable changes in the TABP concerns the background upon which the treatment program had been superimposed. All of the patients were enrolled in an on-going exercise rehabilitation program designed for the recovery of cardiovascular functioning. The evidence supporting the benefits of exercise in the prevention of cardiovascular disease (e.g., Haskell, 1984; Paffenberger, Hyde, Wing, & Steinmetz, 1984) and the rehabilitation of cardiovascular functioning (e.g., Fletcher, 1984; Shepherd, 1983) is extensive and beyond the scope of this paper. A
Recent review (Roman, 1985) concluded that the short-term results of exercise programs were conclusive and excellent and that the long-term results were suggestive but not conclusive. Reviews of the psychologic effects of exercise (e.g., Folkins & Sime, 1981; Goff & Dimsdale, 1985; Taylor, Sallis, & Needle, 1985) have concluded that while methodologic problems exist and mechanisms of action have yet to be clearly elucidated, psychologic benefits accrue to exercisers. Thus there seems to be good reasons for over 700 cardiac rehabilitation programs which are primarily exercise oriented across the United States (Stoner, 1983). The beneficial effects of exercise upon cardiovascular risk factors, e.g., blood pressure, cholesterol, HDL, triglycerides, and weight, have been repeatedly demonstrated.

More germane to the present discussion is the Blumenthal et al. (1980) report showing beneficial changes in Type A behavior during an aerobic conditioning program. Recently, Lobitz et al. (1983) also demonstrated TABP decreases with aerobic exercise. In both studies, subjects were not patients and TABP change was defined by the JAS. Lobitz et al. also reported that the exercise program was superior to an anxiety management condition based upon Suinn's (1975) work. Additional exercise studies which have not directly assessed the behavior pattern have reported positive changes in components of the behavior pattern, e.g., belligerence, negativism, and suspiciousness (Stern & Cleary, 1981) and anger (Berger & Owen, 1983). While not all studies have reported improvement in Type A behavior (e.g., Jasnoski, Holmes, Solomon, & Aguiar, 1982; Rejeski, Morley, & Miller, 1984), the overall evidence suggests that aerobic exercise has beneficial effects on the behavior pattern. While this possibility can not be directly
determined, the reader will recall that despite their notable artery disease, the mean ejection fraction of 63.6%, was within normal limits of 67% ± 8% (Dodge & Sandler, 1974). As the outcome of an infarction is independently determined by ischemic damage, as well as left ventricular dysfunction (Bigger, Coromilas, Weld, Reiffel, & Rolnitzky, 1984), the normal ventricular functioning may have allowed patients to more fully participate in the exercise portion of the rehabilitation program, as well as exercising at home. Further, patients were compliant with the exercise program and given their cardiovascular condition were, perhaps, doing as well as they could. Thus, exercise may have had a palliative effect upon CAD progression and a remediative effect upon Type A reduction.

In summary, the lack of demonstrable changes in Type A behavior during the course of the present study may be explicable. The foremost reason would seem to be the patients' ages, i.e., these patients were hardy or sociable Type A's who may have passed a critical age at which the behavior pattern began to lose its pathophysiological significance. Another significant consideration is the patients' prolonged participation in an exercise oriented cardiac rehabilitation program which may have moderated their Type A behavior. For these reasons, any type of TABP intervention might not have been able to demonstrate significant efficacy.

Change in Cardiovascular Reactivity

The second shortcoming of the present study is the failure to reduce cardiovascular (CV) reactivity. Comparisons with investigations discussed in the introduction are difficult for only one study utilized a stressor as part of the treatment evaluation. In that study
(Levenkron et al., 1983) of nonpatient Type A's, the mean pre- to posttreatment reductions in SBP (5.6 mmHg) and DBP (4.4 mmHg) were significant but were not related to the treatment conditions. Reductions in HR were neither significant nor reported. Therefore, to compare the CV responses of this study's patients to the two stressors, previous investigations using these stressors were reviewed to determine the magnitude of CV responses.

Unfortunately, the QE has not received much usage as a stressor. In the original study by Schiffer et al. (1976), HR changes were in the order of 10 BPM and BP changes were in the order of 7 mmHg and 22 mmHg for DBP and SBP, respectively. More recently, Sime et al. (1980) investigated CV responses in post-MI patients and reported changes of a similar magnitude. Using an abbreviated history quiz, Dembroski, MacDougall, and Lushene (1979) reported SBP response of approximately 24 mmHg and DBP responses of 11 mmHg in another post-MI sample. Thus, while little research with the QE has been undertaken in CAD patients, the magnitude of the response is fairly consistent. With the cardiac rehabilitation patients, the mean CV reactivity was substantially less than in these studies. Mean changes (mean of two baseline values minus mean of two quiz values) across all subjects at pretreatment in HR, SBP, and DBP were .9 BPM, 14.7 mmHg, and 7.7 mmHg, respectively. Given this level of CV change, especially for HR, the likelihood of substantial CV reduction as a result of treatment seems remote, at best. In fact the posttreatment assessment did demonstrate modest mean CV reductions of 5.7 mmHg for SBP, 3.3 mmHg for DBP, and .5 BPM for HR for all participants. As changes were nonsignificantly different across the three conditions, the reductions may simply represent
habituation to the stressor. However, another possibility is that the reductions were merely random error due to the use of an unreliable stressor. A recent study by Williamson, Waters, Bernard, Faulstich, and Blouin (1985) with college students indicated that the quiz was unreliable across a two week period. While a comparable investigation with CAD patients has not been conducted, the correlations between pre- and posttreatment assessments in the waiting list condition (n=14) were consistently significant and are shown in Table 18. In fact, CV responses during the quiz were more reliable than during baseline or recovery and the magnitude of the correlations is quite satisfactory (cf. Krantz & Manuck, 1984). Thus, in the present study, the reliability of the stress quiz is not a satisfactory explanation for the nonsignificant changes in CV reactivity during treatment. However, a more likely explanation lies in the patients' medication regimens. The role of medication in attenuating CV reactivity is discussed following the discussion of cold pressor reactivity.

The second stressor, the cold pressor (Hines & Brown, 1936), has received more usage than the QE and for comprehensive reviews the reader is referred to Lovallo (1975) and Ruddel, McKinney, Buell, Blumsohn, and Eliot (1984). In their discussion of reliability, Ruddel et al. concluded that the CV pattern to the cold pressor remains stable if the repeat tests are performed in the same way on each occasion and that an untreated control group should also undergo the repeated testing. As this study adhered to these recommendations, the results may be viewed with some confidence. However, as with the QE, the cold pressor demonstrated nonsignificant changes due to treatment.
Table 18
Mean Correlations between Pre- and Posttreatment QE for the Waiting List Control Group

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Quiz</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP</td>
<td>.73**</td>
<td>.91**</td>
<td>.66*</td>
</tr>
<tr>
<td>DBP</td>
<td>.67**</td>
<td>.73**</td>
<td>.59*</td>
</tr>
<tr>
<td>HR</td>
<td>.78**</td>
<td>.89**</td>
<td>.79**</td>
</tr>
</tbody>
</table>

*p<.05; **p<.01.
As with the QE, the magnitude of CV reactivity to the cold pressor was determined in previous studies using CAD patients as subjects. In general, the CV response to the cold pressor has been in the range of 25-50 mmHg for SBP and approximately 50% of the SBP response for the DBP response, i.e., 15-35 mmHg which is accompanied by a HR change of 10 BPM (Palmer, Ziegler, & Lake, 1978; Voudoukis, 1971; Wasserman et al., 1983; Waters et al., 1983). This HR response is equivalent to that of healthy subjects while the BP response is greater than the BP response of healthy subjects (Ruddel et al., 1984). The Voudoukis study is also pertinent for the comparison of patients with CAD and hypertension to patients with only CAD. In this study, the BP response of patients with hypertension superimposed upon heart disease was reported to be 45% greater than in those patients with only heart disease. Correspondingly, the CV response of older subjects (40+ years) has been reported to be significantly greater than that of subjects younger than 40 years of age (Palmer et al., 1978). Thus, subjects in the present study might have been expected to demonstrate dramatic increases in HR and BP due to their age and BP status (29 of 45 patients with hypertension).

However, the patients' CV responses to the cold pressor were substantially muted. Specifically, the average HR response across all patients was only .7 BPM. Blood pressure responses were greater, mean SBP response of 20.8 mmHg and mean DBP response of 10.2 mmHg, but less than the BP responses reported in previous investigations. As might be expected, the difference (reduction) at posttreatment was also muted. The mean changes were decreases of .1 BPM, 3.4 mmHg for SBP, and an increase of .6 mmHg for DBP. Again the reduced magnitude of CV
response likely reflects the effects of medication which may have precluded the demonstration of treatment efficacy.

**Concurrent therapy (medication) considerations.** As mentioned in the discussion of CV reactivity to the stress quiz and the cold pressor, patients demonstrated blunted HR and BP responses. The patients' medications, especially beta blockers, were probably responsible for these effects. Beta blockers were introduced approximately two decades ago and now are widely used in the treatment of cardiovascular disorders. Beta blockers primarily compete with epinephrine and norepinephrine at the adrenergic receptors. As a consequence, their effects are greatest during the activation of the sympathetic nervous system, i.e., during times of either physical or psychological stress. For further discussion of the mechanisms of beta blockade, the reader is referred to a cogent review by Durel, Krantz, Eisold, and Lazar (1985) or more medically oriented reviews by Nies and Shand (1975) and Frolich, Dunn, and Messerli (1983).

Several studies have examined the effects of acute and chronic administration of beta blockers upon CV reactivity in both healthy and diseased subjects (e.g., Bonelli, 1982; Erdmann, & Lindern, 1980; Krantz et al., 1982; Langer et al., 1985; Schmeider et al., 1983; Smyth, Hughson, Walters, & Ranney, 1984; Weiss, Del Bo, Reichek, & Engelman, 1980). Consistently, HR decreases which were generally statistically and clinically, e.g., 25 BPM to the stress of mental arithmetic (Bonelli, 1982), significant have been demonstrated. Blood pressure reductions have also been noted with SBP showing more pronounced physiologic, as well as statistically demonstrable, effects than DBP. The BP effects, however, may in part be artifact for BP
effects are associated with long-term treatment rather than short-term or acute administration (Frolich et al., 1983). Additional considerations in evaluating the effects of beta blockade are the individual's response to beta blockade, i.e., all subjects/patients do not show the desired physiologic response, and the potency of the particular beta blocker, e.g., timolol has eight times the beta blocking potency of propanolol.

Another class of drugs which many of the patients (13 of 45 patients) were receiving was calcium channel blockers. The main effect of calcium channel blockers is a decrease in arterial smooth muscle tone which has the cardiovascular effects of decreasing blood pressure and raising the threshold of anginal pain (Luft, Aronoff, Sloan, Fineberg, & Weinberger, 1985). The HR effect is variable and dependent upon the particular pharmacologic agent (Mitchell, Schroeder, & Mason, 1982). For a more detailed explanation of the mechanisms of these agents, the reader is referred to the two previously cited papers or Flaim and Zelis (1981). The HR changes are generally modest and as likely to be increases as decreases. Calcium channel agents have not been in use for as long a period as beta blockers (10 vs 20 years) and their effects have not been fully evaluated. However, investigations of the effects of exercise and other primarily physical stressors have noted reduced BP reactions with medication (Flaim & Zelis, 1981).

In sum, the pharmacological regimes which patients in the present study were receiving likely had a significant impact upon the evaluation of Hypothesis 2, i.e., treatment would have a beneficial effect upon CV reactivity. Beta blockers were being administered to 91% of the patients and calcium channel blockers to 29% of the
patients. Ideally, medication would have been stopped prior to the pre- and posttreatment evaluations. However, medication could not be stopped due to the patients' cardiac status and even if medication had been discontinued another potentially confounding variable might have affected the reactivity results.

Concurrent therapy (exercise) considerations. In addition to possibly altering the patients Type A behavior (see previous discussion), aerobic exercise may have altered their CV reactivity. The hypothesis that exercise training (or fitness level if already trained) can alter reactivity has recently been examined in several studies but the evidence is not conclusive. In two recent studies with young adults (Lake, Suarez, Schneiderman, & Tocci, 1985; Sinyor, Schwartz, Peronnet, Brisson, & Seraganian, 1983), the CV responses of trained and untrained subjects were compared. Sinyor et al. reported that the trained subjects had a significantly lower HR at baseline and throughout recovery but that differences were not significant during the application of the stressors. Lake et al. also reported baseline differences between their fit and unfit subjects but did demonstrate significantly greater SBP and DBP reactivity in the unfit subjects. The differences in BP reactivity were apparent only during the SI. Lake et al. speculated that the interpersonal interview may be more typical of daily stresses than their other stressors, e.g., a card game, cold pressor. Therefore, the greater CV responses of the sedentary subjects during the SI may be more pathognomonic than the CV responses during the unusual stressors.

Using older adults as subjects, Hull, Young and Ziegler (1984) reported reduced HR and DBP responses but similar SBP responses in fit
and unfit subjects. Finally, a training study by Keller and Seraganian (1984) indicated that the responses of untrained (unfit) subjects became similar to the responses of trained (fit) subjects over the course of a nine week fitness program. Our group at the University of Tennessee (Moes, Alpert, & Murphy, 1985) has also been investigating the relationship between fitness and reactivity in adolescents. We have found that maximal oxygen consumption (a measure of fitness) as determined during a maximal exercise test on a cycle ergometer was significantly and inversely related to the maximal HR obtained during the stress of a video game. A recent review of exercise as a modulator of CV reactivity (Dimsdale, Alpert, & Schneiderman, in press) indicated that the evidence is suggestive but that further research is needed to more clearly define the relationship between fitness and reactivity.

To summarize the CV reactivity results, participation in the stress management program did not ameliorate patients' reactivity to either the QE or the cold pressor. The primary hypotheses advanced to account for this lack of change are that the patients' medication regimens precluded the demonstration of substantial reactivity (and, therefore, reactivity reduction) and that their long-standing exercise regimen may have induced reactivity reductions prior to the initiation of the stress management program. These factors may have exerted their effects independently or in concert.

Change in Secondary Variables

Now, turning to the secondary outcome variables utilized in this investigation, results of treatment paralleled results for the behavior pattern and reactivity. These particular measures, i.e., lipids, State-Trait Anxiety Inventory (Spielberger et al., 1970), Locke-Wallace
Marital Adjustment Scale (Locke & Wallace, 1959), and Holmes-Rahe Inventory of Life Events (Holmes & Rahe, 1967) were selected due to their usage in previous investigations (see Introduction) or due to the interest of the investigator, i.e., marital satisfaction (cf. Murphy et al., 1982). First, lipids, particularly cholesterol, have been examined in several intervention studies showing beneficial change (Adsett & Bruhn, 1964; Friedman et al., 1984; Roskies et al., 1979), detrimental change (Jenni & Wollersheim, 1978), and no change (Ibrahim et al., 1974; Rahe et al., 1979). However, because initial cholesterol levels (see Table 14) were not elevated (generally less than the 50th percentile), beneficial cholesterol changes may have occurred prior to this investigation, as with other outcome variables in this study. Thus, cholesterol change may have been affected by a relative "floor" effect in these patients. The LDL decrease which occurred across all patients was beneficial as LDL levels are directly related to CHD risk. The trade-off was an increase in VLDL concentration. However, the VLDL-CHD association is not as strong as the LDL-CHD association. Changes in the two lipoprotein fractions were not related to treatment. As with changes in the behavior pattern and reactivity, evidence suggests that lipid levels may have been affected by the patients' exercise training (e.g., Seals, Hagberg, Hurley, Ehsani, & Holloszy, 1984) and their medication (e.g., Weinberger, 1985).

As with the lipid changes, reductions in anxiety have not been shown to be consistently associated with Type A interventions. While Suinn (1982) suggests that highly anxious subjects benefit the most from treatment, this suggestion may also be confounded by regression to the mean among the highly anxious subjects. A more recent study (Byrne
et al., 1985) concluded that anxiety, as a trait or state, formed no real part of the TABP. The present study suggests that the Byrne et al. conclusion may be accurate. Score changes on the STAI were very modest, mean group changes ranged from a decrease of 3.2 points to an increase of 2 points, and changes were not affected by patients' treatment condition. However, because behavior pattern changes were also modest and unaffected by treatment condition, the efficacy of a Type A intervention upon anxiety can not be assessed adequately in the present study.

In contrast with the small changes exhibited on the STAI, changes on the Inventory of Life Events were sometimes very substantial and indicated increases in stressful life events. Again, changes from pre-to posttreatment were not significantly affected by treatment condition. The Holmes-Rahe Inventory has not been used in previous studies for these studies, like the present study, were short-term studies and there is little reason to believe that many significant events would occur over a three to four month period. However, the present study was originally conceived to include a follow-up period during which important life events could occur. None-the-less, the increase in life event scores suggest that the treatment program (and assessment procedures in the waiting list conditions) may have had a sensitizing effect upon patients perceptions or recollections of life events. If patients, in fact, perceived their lives as more stressful at the conclusion of the treatment, then the awareness of these stressors may have been accompanied by increased coping with these life events for, as already noted, the life event changes were not accompanied by negative changes in the TABP or CV reactivity. This
hypothesis is tenuous and would, clearly, need corroboration from future research. Another measure which has not been used in previous Type A investigations is the Locke-Wallace Scale. This scale was selected as an outcome evaluation measure because changes in the TABP could hypothetically improve marital adjustment. However, the married subjects in the present study were already well-adjusted in their marriages as attested to by the group pretreatment means of 111.4, 109.9, and 112.8 for the behavioral, supportive, and waiting list groups, respectively. Locke and Wallace (1959) considered a score of 100 as the criterion for well-adjusted marriages. Thus, patients may have been handicapped by a relative ceiling effect in this study though small improvements in the adjustment scores were noted in all three groups at posttreatment.

Finally, the weekly stress ratings provide another suggestion, albeit a slight suggestion in terms of the overall pattern of results, that treatment was beneficial. Differences between the two treatment conditions were significant during the seventh and eighth weeks. Also, the comparison of the three experimental conditions, which was nonsignificant at pretreatment, was significant at posttreatment when the two treatment conditions reported significantly less stressful weeks than the no treatment condition. However, the clinical significance of a one point change (posttreatment mean minus pretreatment mean from Table 15) on an 11 point rating scale would seem to be modest at best. Therefore, the changes in weekly stress ratings, in combination of the Holmes-Rahe Life Events changes, may be considered as tentative evidence of a modest degree of therapeutic efficacy. This conclusion is quite tentative for considering the
number of analyses performed, these findings may be merely artifact, i.e., some significant results might be expected on the basis of chance alone.

In conclusion, the present study failed to support the hypothesis that a stress management program would be effective in altering the Type A behavior pattern and cardiovascular reactivity. While these results may be due to the characteristics of the stress management program, a more cogent explanation involves this particular patient sample. The patients who participated in the stress management program were older than the patients in previous investigations and were receiving concurrent therapy. This concurrent therapy (exercise and medication) had been received for a prolonged period of time and previous work suggests alterations of the Type A behavior pattern, reactivity, psychological well-being, and blood chemistry. An equal, if not more important, consideration is that in this patient sample the behavior pattern was not related to the severity of CAD. Modest evidence suggested that the patients' abilities to cope with stress was slightly improved with participation in treatment. Further, the demonstration that patients' values on the outcome measures generally decreased in all conditions during the treatment period suggests that assessment may have had a sensitizing effect upon the patients, i.e., through assessment patients may have become more aware of their behavior and taken appropriate corrective action. While the cited studies support these conclusions and explanations, the explanation of why this treatment program was ineffective with this patient sample is very speculative and based upon subjective comparisons with previous studies. The hypotheses advanced in this discussion can only be
confirmed by future investigations which prospectively examine these potentially mitigating variables. Future research should also seek to determine what treatment can help which patients ameliorate the TABP and CV reactivity and when the TABP and CV reactivity are pathognomonic for recurrent coronary events.
Summary

This investigation sought to determine the efficacy of a stress management program in the amelioration of the Type A behavior pattern. The subjects were 45 patients enrolled in the Cardiac Rehabilitation Program of the Veterans Administration Medical Center in Augusta, Georgia. Patients were randomly assigned to the experimental conditions, i.e., behavioral, supportive, and waiting list, based upon exhibition of the Type A behavior pattern. The treatment program consisted of 10 sessions of 1-1½ hours duration. The outcome of the treatment program was based upon an evaluation of changes in a variety of behavioral, physiologic, and self-report indices.

The results did not support the two major hypotheses: (a) patients in the behavioral stress management program would evidence superior change in Type A behavior as assessed by the Structured Interview, and (b) patients in the behavior stress management program would evidence superior change in cardiovascular reactivity to the stress of the cold pressor and the Quiz Electrocardiogram. Though changes occurred from pre- to posttreatment, these changes were not significantly affected by the treatment condition. Secondary dependent measures of anxiety, life events, marital satisfaction, and lipids also failed to demonstrate changes which were affected by treatment. Finally, results provide a suggestion that treatment was effective in ameliorating every day stress. Treatment was perceived as beneficial by the participants and their spouses and as potentially beneficial by members of the cardiac rehabilitation staff.
The failure of the treatment program to demonstrate beneficial change was discussed in terms of four interrelated factors: (a) the negative association between the behavior pattern and the severity of coronary artery disease in this patient sample, (b) the patients' ages, (c) the patients' medication regimens, and (d) the patients' prolonged participation in an exercise oriented rehabilitation program. In sum, patient characteristics and previously established treatment regimens may have precluded an adequate examination of the efficacy of a short-term intervention program in the amelioration of the Type A behavior pattern. Future research should seek to define those parameters of subjects and treatment which may independently or synergistically affect Type A interventions.


Murphy, J., Norman, C., & Somes, G. (1985). The Type A - CAD relationship: Is the difficulty the assessment of the behavior pattern or the severity of CAD? Manuscript submitted for publication.


Appendices
I have been invited to participate in a research study on stress management. This study is designed to study the usefulness of stress management in cardiac rehabilitation. I understand that I have been asked to participate because I am participating in the Cardiac Rehabilitation Program.

I understand that past research suggests that stress management programs are helpful for cardiac rehabilitation. However, the research does not show what type of program help patients the most. Therefore, this research study is going to compare being in two different kinds of stress management programs with not being in any stress management program. That is, there will be three conditions of participation: (1) a group focusing on behavior, (2) a group focusing upon emotions, and (3) a group that does not receive treatment. I understand that all patients will be assigned to the groups at random and I may not be in the group that I had initially wanted to be in. I understand that the stress management program does not replace the Cardiac Rehabilitation Program and that I will continue to receive treatment and participate in the Cardiac Rehabilitation Program and that I will continue to receive treatment and participate in the Cardiac Rehabilitation Program even if I am in the no treatment stress management program.

All Participants

I am being asked to complete several questionnaires concerning information about how I handle stress and how it affects my life. I will experience no substantial physical discomfort from this procedure. I am also being asked to donate three (3) ten (10) milliliter blood samples. One sample will be collected before the groups begin, one after the weekly group meetings end, and the third sample will be collected six months after the end of the weekly meetings. Each blood sample represents about one (1) tablespoon of blood. This is a part of the Cardiac Rehabilitation Program and their program evaluation. That is, the same samples will be used by Cardiac Rehabilitation and the stress management study. Blood will be drawn in a manner consistent with established Cardiac Rehabilitation Procedures. I will also be asked to place my hand in a container of ice water to measure my response to stress. I understand that I may remove my hand when I want to and will be asked to remove it after five (5) minutes. This procedure will be performed in a manner consistent with already established cardiology procedures. The blood work and ice water stress test will possibly involve mild to moderate physical discomfort. A final procedure, will be a videotaped structured interview. This
interview has been used extensively with cardiac patients and will be
used in the present study in such a manner. While there may be some
psychological discomfort due to being videotaped, there will be no
physical discomfort. I understand that I am one of 45-60 patients to
participate in this study and that my completion of the assessment
instruments will help determine how useful the stress management program
is. My total participation in the study will take approximately 30
hours.

Participants receiving group treatment

I understand that in order to participate in the Stress Management
Program I must attend at least eight (8) of the ten (10) stress
management treatment meetings and complete the post-treatment
assessments. During follow-up, I must attend four (4) of the six
follow-up meetings and complete the follow-up assessments.

Participants not receiving group treatment

I understand that assignment to conditions will be random and that I
may not be able to participate in one of the two group treatments.
However, I will still complete the measurements at pre-treatment, post-
treatment, and follow-up. My completion of the assessment measurements
will help determine if stress management is helpful for cardiac
rehabilitation patients and if so, which type of program is more
helpful. After the six month follow-up I will be offered the treatment
program shown to be most effective.

I understand that the possible benefits of this study are that data
may be obtained which will give psychologists, physicians and others
interested in cardiac rehabilitation a better understanding of possible
psychological factors that are involved and possible information which
might aid in treatment. Perhaps more importantly, I may also learn to
better control my responses to stress.

I understand that this evaluation of my ability to handle stress does
involve direct psychological treatment procedures. The assessment
procedures (tests) supply data which is frequently used in the
psychological assessment of stress as well s in cardiac rehabilitation
programs.

I understand that my records will become part of the hospital chart
and that I will not be personally identified in any publication of the
results of this study.

In case of any adverse effect or physical injury resulting from this
study, eligible veterans are entitled to medical treatment.
Compensation may be payable under Title 38 U.S.C. 351 or in some
circumstances under the Federal Tort Claims Act. Non-eligible veterans
or non-veterans are entitled to medical care and treatment on
humanitarian emergency basis. Compensation would be limited to
situations involving negligence and would be controlled by the provisions of the Federal Tort Claims Act.

I understand that Joseph K. Murphy, M.A., who can be reached at extension 2869, will answer any and all questions that I may have at any time concerning the study, the procedures, and any injuries that may appear to be related to the research. If I have any questions or concerns about the rights of research subjects, I may contact Dr. Thomas A. Huff at 828-2131.

My participation in this study is voluntary. I understand, however, that I may revoke my consent and withdraw from the study now or at any time in the future, without penalty or loss of care or other benefits to which I am otherwise entitled.

I voluntarily consent to participate in this study.

SUBJECT'S SIGNATURE ____________________________ DATE
(* or legal guardian or patient's representative)

INVESTIGATOR'S SIGNATURE _________________________ WITNESS' SIGNATURE _________________________

* The undersigned hereby verified that he/she is the Legal Guardian or Parent's Representative of ___________________________ and as such as legal authority to consent to the study outlined above.
February 22, 1983

Joseph K. Murphy, M.A.
Department of Psychology
116-B
VA Hospital
Augusta, Georgia 30910

RE: Project Title - "Stress Management In Cardiac Rehabilitation"
Approval Date - February 22, 1983

Dear Dr. Murphy:

The Human Assurance Committee has reviewed and approved the addendum submitted January 12, 1983 to the above referenced project "Stress Management In Cardiac Rehabilitation" in accordance with the DHHS policy and the institutional assurance on file with the DHHS.

Sincerely,

Thomas A. Huff, M.D.
Chairman
HUMAN ASSURANCE COMMITTEE

TAH/jb
Appendix 3

Veterans Administration

Date: March 8, 1983

To: Joseph K. Murphy, M.A.

Subj: "Stress Management in Cardiac Rehabilitation"

Memorandum

From: Chairman, R&D Committee

The Research and Development Committee concurred with the recommendation of the Human Assurance Committee and approved the above referenced protocol for use at the Veterans Administration Medical Center in their meeting held on February 22, 1983.

JOHN P. BOINEAU, M.D.
Appendix 4

Treatment Credibility Questionnaire

<table>
<thead>
<tr>
<th>Strongly Yes</th>
<th>Moderately Yes</th>
<th>Unsure</th>
<th>Moderately No</th>
<th>Strongly No</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Do you consider this Type A/stress management program to be a logical and reasonable approach to altering Type A behavior and/or stress management?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Do you feel that the program that you received was successful for you?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Do you feel that the Type A/stress management procedures used in your group would work for most other people with problems similar to yours?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Are you confident that you will be able to at least maintain the improvements made in treatment?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Would you recommend this program for your friends who would like to alter their Type A behavior or their response to stress?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Questions were scored on a scale from 1 (strongly no) to 5 (strongly yes) and summed to obtain a total score. For the Rehabilitation Program staff, questions 2 - 4 were reworded to refer to cardiac patients in general.
Appendix 5

<table>
<thead>
<tr>
<th>NAME _____________________________</th>
<th>DATE __________________________</th>
</tr>
</thead>
</table>

Below are listed a variety of events that may be viewed as stressful or unpleasant. In the space next to each item indicate if the event occurred this week and, if so, the degree of subjective stress that you experienced. Also, in the spaces provided at the end, list any additional unpleasant events that you experienced and the degree of subjective stress associated with each event. Please answer the following items as honestly as you can so that we can obtain accurate data.

X = did not occur  2 = a little stress  5 = very much stress  
0 = not stressful  3 = some stress  6 = panic  
1 = very little stress  4 = much stress

1. Late for work/appointment  
2. Poor performance at task  
3. Poor performance due to others  
4. Thinking about unfinished work  
5. Hurrying to meet deadline  
6. Interrupted during task/activity  
7. Someone spoils your completed task  
8. Doing something you are unskilled at  
9. Someone you supervise performs poorly  
10. Unable to complete a task  
11. Being unorganized  
12. Criticized or verbally attacked  
13. Ignored by others  
14. Speaking or performing in public  
15. Dealing with rude waiter/salesperson  
16. Interrupted while talking  
17. Meeting a new person  
18. Unexpected visitors  
19. Being forced to socialize  
20. Someone breaks a promise/appointment  
21. Unsolicited salesperson/charity  
22. Competing with someone  
23. Being stared at  
24. Not hearing from someone you expected to hear from  
25. Experiencing unwanted physical contact  
26. Being misunderstood  
27. Being embarrassed  
28. Malfunction of machinery or appliance  
29. Running out of food/personal articles  
30. Problems with utilities/postal service  
31. Arguing with spouse/boyfriend/girlfriend  
32. Arguing with another person  
33. Problems with kid(s)  
34. Dealing with parents  
35. Waiting longer than you wanted  
36. Interrupted while thinking/relaxing  
37. Someone cuts ahead of you in a line  
38. Poor performance at sports/games  
39. Doing something you don't want to do  
40. Inability to complete all plans for today  
41. Car trouble  
42. Difficulty parking car  
43. Difficulty in traffic  
44. Auto accident  
45. Money problems  
46. Being overcharged for a purchase
141

X = did not occur  2 = a little stress  5 = very much stress
0 = not stressful  3 = some stress  6 = panic
1 = very little stress  4 = much stress

47. Store lacks a desired item
48. Problem with vending machine
49. Misplacing something
50. Worrying about other people's problems
51. Bad weather
52. Unexpected expenses (i.e., fines, traffic tickets)
53. Having your sleep disturbed
54. Forgetting something
55. Fear of illness/pregnancy
56. Experiencing illness/physical discomfort
57. Someone borrows something without permission
58. Your property is damaged/destroyed
59. Minor accidents (break something, tear clothing)
60. Thinking of the future
61. Hearing bad news
62. Concern over personal appearance
63. Confrontation with authority figure
64. Visiting a doctor/dentist
65. Dieting
66. Exposure to fear situations or objects
67. Exposure to upsetting TV show, movie, book
68. "Pet Peesves" violated (i.e., someone fails to knock)
69. Failing to understand something
70. Experiencing a narrow escape from danger

OVERALL WEEKLY STRESS RATING

Please circle the number that corresponds to your overall level of stress this week. Ratings should reflect your own subjective feelings of discomfort (i.e., the degree to which you feel pressured, worried, hassled).

0 1 2 3 4 5 6 7 8 9 10

No Comfortable Uncomfortable Very Stress Uncomfortable Equal to the most stressful week I have had
Joseph K. Murphy was born in Augusta, Maine on February 2, 1951. He received the Bachelor of Arts degree from McGill University in November of 1976 and the Master of Arts degree from Louisiana State University in December of 1980. A predoctoral internship in clinical psychology was completed at the Veterans Administration Medical Center and Medical College of Georgia Consortium. He is currently a doctoral candidate in clinical psychology at Louisiana State University and is employed as a research associate in the Department of Pediatrics at the University of Tennessee Center for the Health Sciences in Memphis, Tennessee.
Candidate: Joseph Kemp Murphy

Major Field: Clinical Psychology

Title of Dissertation: Stress Management and the Amelioration of Type A Behavior In Cardiac Rehabilitation

Approved:

[Signatures]

Dean of the Graduate School

EXAMINING COMMITTEE:

[Signatures]

Date of Examination:

November 6, 1985