

TYPE A CORONARY-PRONE BEHAVIOR CORRELATED
WITH CARDIAC RISK FACTORS

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CHAPTER 1

INTRODUCTION

In the United States, cardiovascular disease is the leading cause of death. Approximately 40,120,000 Americans have some form of heart and blood vessel disease. More than 4,190,000 persons are known to have coronary heart disease, meaning they have histories of heart attacks and/or angina pectoris. Of these Americans, 350,000 die each year of heart attacks before they ever reach a hospital. The American Heart Association estimated further that in 1979 a massive 52% of all deaths would be attributable to cardiovascular disease (Heart Facts Reference Sheet, 1979). These overwhelming statistics clearly indicate deficits of present efforts to prevent and ameliorate heart disease.

The most universally accepted ideology regarding the etiology of heart disease is that it is "pluricausal," involving the cardiac risk factors either in combination or in singular extremes. Epidemiological studies have isolated over the past several decades standard cardiac risk factors, as well as numerous possible contributing factors. Yet, mortality rates for heart attack victims have not

decreased (DiGirolamo & Schlant, 1974). "The best combinations of standard 'risk factors' fail to identify most new cases of this disease" (Jenkins, 1971a, p. 244). In recent years the role of behavior, or stress and the coronary-prone personality, has stimulated intense controversy. Many practitioners still fail to recognize stress as a significant or valid cardiac risk factor. The magnitude of heart disease as a national health problem, considered by many to be epidemic in proportion, is stimulating researchers to look for all contributing factors in order to understand and thus prevent the development of the disease. The mandate of nursing and the entire health care community is to determine the role of behavior in the etiology and progression of heart disease.

Statement of Problem

The problem of this study was: Is there an association between the Type A coronary-prone behavior pattern and selected standard cardiac risk factors in post-myocardial infarction patients? A subproblem was: Is there a selected standard cardiac risk factor or set of factors which predict Type A coronary-prone behavior in postmyocardial infarction patients?

Purposes

The purposes of this study were to determine:

1. The extent that selected standard cardiac risk factors were present in postmyocardial infarction patients.
2. The extent that Type A coronary-prone behavior was present in postmyocardial infarction patients.
3. The association between the selected standard cardiac risk factors and the Type A coronary-prone behavior pattern in postmyocardial infarction patients.
4. Which selected standard cardiac risk factor or set of factors best predicted the Type A coronary-prone behavior pattern.

Background and Significance

Geographically, the developed countries of the Western world have the highest incidence of arteriosclerotic heart disease, of which the United States is second only to Finland in mortality rates (DiGirolamo & Schlant, 1974). Researchers have postulated numerous theories as to causality including diet, race, economic status, geographical conditions, life styles, and environmental stressors. Important to note is a study of international populations with unusually low rates of heart disease which were compared by Bruhn and Wolf (1970). They found a high incidence of one or more risk factors in many of the

populations in which heart disease was rare. This finding of a lower incidence of heart disease among lesser developed nations despite a high incidence of one or more risk factors is quite significant because it indicates that the major cardiac risk factors are often inadequate in identifying coronary-prone persons. Determination of other risks is necessary to promote significant prevention of coronary disease. Such research is critical as evidenced by the World Health Organization's statement (cited by Jenkins, 1971a):

Ischaemic heart disease, or coronary heart disease, has reached enormous proportions, striking more and more at younger subjects. It will result in coming years in the greatest epidemic mankind has faced unless we are able to reverse the trend by concentrated research into its cause and prevention. (p. 244)

It has been reported that "angina pectoris was quite rare and myocardial infarction almost infrequent until the past four or five decades," during which an increase in incidence of myocardial infarction has been seen particularly in middle-aged males (Rosenman & Friedman, 1971, p. 77). To determine the factors or cardiac risks related to this increased incidence of heart disease, a multitude of studies have been conducted. The most significant study to date regarding heart disease is the Framingham study begun in 1949 (Kannel, Castelli, Verter, & McNamara, 1971). From the Framingham study several

major cardiac risk factors have been identified which include age, sex, cigarette smoking, elevated blood pressure, high levels of serum cholesterol, glucose intolerance, and electrocardiogram abnormalities (Gordon & Kannel, 1973). Other risk factors were identified, but the above factors are considered most predictive. DiGirolamo and Schlant (1974) described "specific" risk factors as age, sex, familial history of premature coronary heart disease, elevated serum lipids (cholesterol and triglycerides), diet, hypertension, cigarette smoking, and carbohydrate intolerance. DiGirolamo and Schlant (1974) also described the "minor" risk factors as obesity, sedentary living, personality type, psychosocial tensions, and 18 other possible contributing factors to coronary heart disease.

Blakeslee and Stamper (1963) noted that for atherosclerosis "there is no single cause, but rather a constellation of causes" (p. 3). The factors they listed are:

high blood pressure, high levels of cholesterol, obesity, excessive eating, especially of certain types of fats and cholesterol, too little exercise and physical activity, diabetes, excessive cigarette smoking, tension and stresses and heredity. (p. 3)

Lough (1975), on the other hand, narrowed the list considerably in a brief article on smoking as a risk factor, by stating that the "principle risk factors are high blood pressure, high serum cholesterol concentration,

lack of physical activity, obesity and smoking" (p. 919). O'Rourke and Ross (1976) listed high blood pressure, elevated serum lipids (cholesterol and/or triglycerides), excessive cigarette smoking, diabetes, and age as "established" risk factors. They also stated that "physical inactivity, obesity and life stress also may increase the chances of having a heart attack, although these factors have not yet been proved to increase risk" (p. 91).

Despite enormous amounts of information obtained from both long term prospective and retrospective studies, no clear agreement among researchers exists as to the cardiac risk factors. In fact, sources are difficult to find that are in agreement regarding this topic. Although a review of the literature reveals the most generally accepted, or major risk factors, based on validated facts and general acceptance among the more notable researchers, controversy exists because of debate over the significance of the new or possible risk factors. Yet attention to the major risk factors has failed to reduce the national cardiovascular mortality rate. Werko (1976), in examining three major research studies including the Framingham study, the National Pooling Project, and the Stockholm Prospective Study, was critical of certain errors in both their design

and methodology. Werko also indicated that some of the recommendations made for the specific populations studied were then recommended for the total population. This is highly significant because thus far the major thrust of the national effort has been to reduce those major risk factors identified from the Framingham studies and others.

Yet, despite efforts to reduce the major risk factors which may be modified, such as hypertension, obesity, elevated cholesterol, and smoking, the mortality rates for coronary artery disease are increasing. In fact, from 1950 to 1967 the death rate from heart attacks among men between the ages of 25 to 64 years rose 5% (Heart Attack, 1970). Thus, many researchers are beginning to question the role of the major risk factors in cardiac disease and are searching for other contributing factors. Friedman and Rosenman (1974b) have speculated that the coronary-prone personality may be the central factor for which the other factors may be interrelated. The role of the coronary-prone personality, however, is highly controversial.

Of the multitude of risk factors discussed, the role of the Type A coronary-prone behavior pattern has received only minor recognition despite a wealth of information available on the subject. Many practitioners, both physicians and nurses, have failed to realize the importance of

this factor. Indeed, little or no preventive measures are discussed in the literature or in actual practice to alert patients to the effects of stress or "coronary-proneness."

The importance of the Type A coronary-prone personality should not be overlooked. Eliot (1974) indicated that "since both human stress and the incidence of cardiac death have increased in parallel during this century, interrelationships must be sought between the two" (p. x). Researchers have identified psychological, social, physiological, and strong biochemical interrelationships between the coronary-prone behavior pattern and stress. The broad implications of this concept will no doubt be far-reaching in the future.

The coronary-prone personality is not a new phenomenon. As far back as 1793, Hunter was aware of the effects of stress upon the heart as was Sir William Osler who in 1897 wrote:

I believe that the high pressure at which men live and the habit of working the machine to its maximum capacity are responsible for (arterial degeneration) rather than excesses in eating and drinking. (cited in Jenkins, 1975, p. 5)

Probably best known for their efforts to define and research the coronary-prone personality are Friedman and Rosenman (1974b) who have developed the concept of the Type A coronary-prone personality. They believed that the

Type A coronary-prone personality is the "major cause of coronary artery and heart disease" (p. 53).

The Type A coronary-prone behavior pattern may be defined as a pattern of existence characterized by extremes of response that results in a continual self-imposed state of stress. Friedman and Rosenman (1974b) defined Type A behavior as follows:

Type A Behavior Pattern is an action-complex that can be observed in any person who is aggressively involved in a chronic, incessant struggle to achieve more and more in less and less time, and if required to do so, against the opposing efforts of other things or other persons. It is not a psychosis or a complex of worries or fears or phobias or obsessions, but a socially acceptable--indeed often praised--form of conflict. Persons possessing this pattern also are quite prone to exhibit a free-floating but extraordinarily well-rationalized hostility. As might be expected, there are degrees in the intensity of this behavior pattern. Moreover, because the pattern represents the reaction that takes place when particular personality traits of an afflicted individual are challenged or aroused by a specific environmental agent, the results of this reaction (that is, the behavior pattern itself) may not be felt or exhibited by him if he happens to be in or confronted by an environment that presents no challenge. For example, a usually hard-driving, competitive, aggressive editor of an urban newspaper, if hospitalized with a trivial illness, may not exhibit a single sign of Type A Behavior Pattern. In short, for Type A Behavior to explode into being, the "environmental challenge must always serve as the fuse for this explosion." (pp. 67-68)

Pattern A behavior has been minutely defined by Friedman and Rosenman (1974b) to consist of many overt behaviors. Such behaviors, for example, include specific speech patterns, time urgency, "polyphasic thought or

performance," undue impatience, aggressiveness, and many other similar actions (p. 84). Physical characteristics have also been postulated. Pattern A individuals are:

Characterized by being male, short, stocky and muscular, and excessively aggressive, smoking large numbers of cigarettes, being obese, taking little exercise, being under "emotional pressure," and having elevated cholesterol and triglyceride levels, and often, glucose intolerance. (Netter & Yonkman, 1971, p. 86)

Pattern B behavior, on the other hand, is just the opposite of Pattern A. Type B persons may best be characterized by easy-going qualities as they generally take life in stride and avoid stressful stimuli. Type B persons are not enslaved by their environments and have the ability to relax and enjoy life. Friedman and Rosenman (1974b) have compared the Type A individual to the "rabbit" and the Type B person to the "tortoise" as in the classic tale.

Despite the immense amount of data available regarding Type A behavior, stress, and personality, much controversy remains. Some researchers speculate that Pattern A behavior is a primary cause of cardiovascular disease with the other identified risks being merely effects. For example, the real problem behind the risks of obesity and smoking could be the behavior that initiated their existence. Others say no relationship can be drawn between Pattern A behavior and the other biochemical factors, while

still others say that Pattern A behavior is indeed valid-- but only one of a plurality of factors which may contribute to cardiovascular disease.

There are two major reasons why Pattern A behavior is so controversial. First, many leaders in the cardiovascular field feel that the primary national efforts should continue to be directed toward further investigation of and validation of the major biochemical cardiac risk factors. In fact, almost all of the national effort has been directed toward preventing and/or ameliorating these factors by means of massive public education programs and research. Yet, the major cardiac risk factors have been repeatedly documented as unreliable. The national mortality statistics clearly support this fact (Heart Facts Reference Sheet, 1979). Keys, Aravanis, Blackburn, van Buchem, Buzina, Djordjevic, Fidanza, Kavonen, Menotti, Pudov, and Taylor (1974) reported that the classic risk factors account for only about half of the cases of coronary heart disease.

Secondly, Kannel et al. (1971) have pointed out that acceptance of relationships between "emotional stress" and coronary heart disease have been most severely hampered by problems with methodology.

The principal handicaps have been the lack of agreement on a uniform acceptable definition of the

phenomenon and a failure to develop reliable and valid methods for measuring the intensity of stress. (p. 102)

Type A behavior as it relates to cardiovascular disease is particularly important to consider as it has been described as belonging to civilized nations. Toffler (1970) dramatically pointed out the effects of the present civilization and stressed the importance of considering what effects future change may have upon man's "psychobiological condition" (p. 2). Consider, for example, communications as a stressor of the Western world. The mass media provides us with a constant bombardment of news which stimulates a variety of reactions and emotions. The public is informed of much information for which it has absolutely no control.

Friedman and Rosenman (1974b) and many others are convinced that Type A behavior is the "major cause of premature coronary heart disease" (p. ix). The literature abounds with almost every conceivable variation or method for study of Type A behavior. Jenkins (1971b), in an article on the psychologic and social precursors of coronary disease, reviewed over 160 papers of which "a clear majority reported positive findings between one or more of the behavioral variables and coronary heart disease" (p. 314).

In a follow-up study of the Western Collaborative Group study (cited by Eliot, 1974), it was found that:

Initially well subjects assessed at intake as possessing Type A Behavior Pattern were more than twice as prone to the onset of clinical CHD as the group of subjects originally assessed as Type B, whether the disease emerged clinically as a myocardial infarct or as angina pectoris. (p. 130)

Moreover, the initially Type A subjects were five times more prone to a second myocardial infarct than Type B subjects during this 8.5 year interval. Fatal heart attacks also occurred twice as frequently in Type A subjects as in Type B subjects (Eliot, 1974).

The data supporting Type A behavior as a valid risk is staggering. The key concept to the vast mystery of cardiovascular disease may well be stress and the resulting coronary-prone behavior personality. The relationship between Type A coronary-prone behavior and the classic cardiac risks must be determined. Jenkins in 1975 had noted that no correlation was found between the Jenkins Activity Survey to measure Type A behavior and the standard risk factors. However, he also noted that the Jenkins Activity Survey (JAS) "still misclassifies too many individuals to allow it to be used in the usual clinical setting" and that the tool is still in the developmental stage (Appendix A, Jenkins correspondence, October 30, 1975). Jenkins has recommended the Bortner scale for

purposes of this research (Appendix A, Jenkins correspondence, October 30, 1975).

The JAS is considered by many to be the most promising means to determine Type A behavior. The previously used interview-observation technique was quite reliable but also impractical. This again reinforces the need for more research. Critics of Type A behavior are quick to point out that methodological problems weaken the strength of the theory of Type A behavior. In order to validate Type A behavior, a written instrument that is consistently reliable is critically needed. Since behavioral research is relatively new and one of the most difficult areas to study, it should be expected that many changes will occur in the future of personality research.

However, despite the known methodological problems in studying behavior, advances have been made in the study of Type A behavior. Gilmore (1974), in his discussion of the physiology of stress, concluded the following:

Apparent innocuous stress to which man is exposed on a day-to-day basis can produce significant cardiovascular changes. It is probable that when the stresses are applied periodically but chronically, the ability of the heart to compensate is exceeded and irreversible pathologic changes may ensue. Although the defense reaction has provided an important survival mechanism in man's evolution, it may also be a primary cause of cardiovascular pathology. (p. 88)

Shapiro (1974) similarly, in discussing the psychophysiology of stress, concluded that:

Behavioral factors may play a critical role in determining normal and abnormal physiological functioning. (p. 91)

He further hypothesized that "the afflictions of living in highly developed countries are peculiarly 'psycho-physiological' in nature" (p. 92). The literature amply abounds with documentation of Type A behavior as it is related to social, psychological, and physiological risk factors.

The primary impetus for doing this study was based on the following points. First, Jenkins (1975) reported that the coronary-prone behavior pattern is an initial forerunner of a myocardial infarction as well as its recurrence. He also stated that there is "evidence to suggest that Type A Behavior is an even stronger risk factor for recurrent myocardial infarction than it is for the initial episode, a characteristic not shared by many standard coronary risk factors" (p. 17).

Secondly, the research allowed for equal assessment of the standard cardiac risk factors as well as Type A behavior in postmyocardial infarction patients. Their existence as well as the degree to which they were associated was determined. More information is vital to determine the significance of all cardiac risk factors.

This research also provided for assessment of basic demographic data in postmyocardial infarction patients.

Cardiac risk factor data, demographic data, and Type A behavior data have potential for use in the hospital setting to assess cardiac risk factors as a basis for planning preventive and rehabilitative measures for cardiac patients. Further, the research was worthwhile in that data were collected regarding Type A behavior by means of the Bortner Scale which is noted to correlate highly with the Jenkins Activity Survey (JAS). The JAS is internationally recognized as the most reliable means for determining Type A behavior. The Bortner Scale is particularly significant as a pencil-paper technique for determining Type A behavior, and it has been shown to be highly correlated with the JAS (Bortner, 1969).

In summary, numerous research studies have shown that the standard cardiac risks do not adequately identify persons at risk for myocardial infarction with accuracy as previously noted. National health projects as well as public teaching aimed at reducing the standard cardiac risks especially of weight, blood pressure, dietary cholesterol, and physical fitness have failed to affect the incidence of heart disease in the United States. The fact that the major factors have been repeatedly validated individually, yet have offered no significant national results, poses many questions. New answers must be sought.

Type A coronary behavior as a cardiac risk factor warrants recognition and further research. National mortality rates from cardiovascular disease are unsurpassed. More frightening is the observation that younger persons are being killed or disabled by this unharnessed devastator yearly. Prevention, amelioration, and rehabilitation from cardiovascular disease is truly the mandate of industrialized nations.

Null Hypotheses

This study tested the following nine null hypotheses:

There is no association between the Type A coronary-prone behavior pattern and the following selected major cardiac risk factors:

1. Age
2. Sex
3. Familial history of premature coronary heart disease
4. Hypertension
5. Elevated serum cholesterol and/or triglycerides
6. Cigarette smoking
7. Glucose intolerance (Diabetes Mellitus)
8. Obesity
9. Lack of exercise/sedentary living

Definition of Terms

For the purposes of this study, the following working definitions were used:

1. The Type A, coronary-prone behavior pattern is a pattern of existence characterized by extremes of response that result in a continual self-imposed state of stress. The Bortner self-rating tool was used in this study to measure Type A behavior.
2. Cardiac risk factors are those risks validated through research as contributory to heart disease. Cardiac risk factors were measured in this study by use of the Cardiac Risk Factor Assessment Tool (CRAT) (Appendix B).
3. The selected standard or major cardiac risk factors included:
 - a. Age--Persons over 18 years of age were included in this study. Age is a non-modifiable cardiac risk factor.
 - b. Sex--Both genders were included in the population for study. Sex is a non-modifiable cardiac risk factor.
 - c. Family history of premature coronary heart disease--This was defined as the presence of any form of heart disease prior to age 50 years (DiGirolamo & Schlant, 1974). The CRAT was used to determine this item.

- d. Hypertension--This was defined as the known history of blood pressure above 150/90 mm Hg as determined by the participating cardiologist or, when applicable, the primary internists of the subject.
- e. Elevated serum cholesterol and/or triglyceride levels--Subjects' levels were determined by the participating cardiologists or, when applicable, the primary internists and were based on the laboratory methods and values particular to the hospitals as well as historical data utilized by the cardiologists. Enzymatic assessment is the most common and specific method utilized by most hospitals today to determine cholesterol and triglyceride levels; values for cholesterol and triglyceride levels using the enzymatic assessment method have been found to be relatively standard among most hospitals and are reflected in chart form (Appendix C).
- f. Cigarette smoking--This was considered a risk factor if present in any quantity on a routine basis over any period of time. For the purpose of this paper cigar and pipe smoking were not considered a risk factor (DiGirolamo & Schlant, 1974; Kannel et al., 1971; Surgeon General's Report,

- 1973). Determination of this factor was obtained by use of the CRAT.
- g. Glucose intolerance (Diabetes Mellitus)--This is defined as a known history of glucose intolerance or diabetes as determined by the participating cardiologists or, when applicable, the primary internists of the subjects.
 - h. Obesity--The Metropolitan Life Insurance Company's Table of Desired Weight, commonly used to determine obesity (Metropolitan Life Insurance Co., 1969, p. 12), was used to compare the participants' weights/heights at the time of the study to the table. Those participants with weights in excess of the table's parameters were considered obese. The participants provided their height and weight on the CRAT. The investigator estimated the body frame as small, medium, or large in order to make appropriate comparisons to the Table of Desired Weights.
 - i. Lack of exercise/sedentary living--This was defined as a lack of routine exercise for relaxation or fitness. A patient's normal work regimen was not considered as exercise. Lack of exercise was self-rated by the participants on the CRAT.

Limitations

Because of the investigator's inability to control all aspects of this study, the following limitations were noted:

1. Physiological factors may have influenced the participants' responses to the questionnaires.
2. The effects of medications may have altered participants' responses to the questionnaires.
3. The participants may not have been fully aware of their health histories or problems.
4. Some participants may not have been completely truthful, or may have been incapable of accurate self-rating.
5. The possible effects of illness or hospitalization may have affected participants' responses to the questionnaires.
6. Stressors and reactions to stressors vary among individuals and may have affected responses of the participants.

Delimitations

The delimitations of this study included the following:

1. The sample for study consisted of patients diagnosed by a physician as having had a myocardial infarction.
2. Hospitalized patients of both sexes were studied no sooner than their eighth day postmyocardial infarction.

3. The study sample was limited to only those persons above 18 years of age.
4. Persons with known psychological disturbances diagnosed by a physician were eliminated from the study. Psychological disturbances were considered as behaviors ranging from acute anxiety to true mental pathology.
5. Patients were not studied if they were:
 - a. not fully alert and cooperative,
 - b. experiencing any pain or distress,
 - c. being monitored,
 - d. requiring oxygen, an IV or any other prophylactic or supportive device, and/or
 - e. not ambulatory.

Assumptions

For the purposes of this study, the following assumptions were made:

1. Personality components are factors that influence how one reacts or adapts to a given situation.
2. Inhabitants of the Western countries and other more "developed" areas of the world are exposed to more potential stressors related specifically to heart disease than inhabitants of lesser developed nations.
3. Change is anxiety producing.
4. Stressors are always present.

5. The effects of stress depend on the reactions and adaptability of the individual.
6. Social, psychological, and physiological factors influence how one reacts to stress.

Summary

National mortality from cardiovascular disease is epidemic in proportion. The standard cardiac risk factors have proven inadequate as predictors of the disease. Despite massive programs to educate the public regarding the classic risks, no significant reductions in mortality can be noted. Furthermore, younger persons are being affected by this killer.

Societal pressures of rapidly changing civilization offer all the necessary ingredients for this pandemic. Research in the past has been poorly conducted in terms of all the risk factors and especially in terms of behavior. Much effort should be directed in this area.

Chapter 1 has provided a synopsis of significant literature related to Type A behavior and the standard cardiac risk factors. The problem statement, purposes, hypotheses, definitions, limitations, delimitations, and assumptions have also been presented.

CHAPTER 2

REVIEW OF LITERATURE

Chapter 2 provides an in-depth review of the literature regarding Type A coronary-prone behavior. The selected standard or major cardiac risk factors which include age, sex, family history of premature heart disease, hypertension, elevated serum cholesterol and/or triglyceride levels, cigarette smoking, glucose intolerance, obesity, and lack of exercise/sedentary living are discussed in terms of the major criteria regarding their significance.

Latest Statistical Facts Related to Decreased Incidence of Coronary Heart Disease

An important development related to this study must first be noted. The April-June 1979 Statistical Bulletin published by the Metropolitan Life Insurance Company (1979) shows that there has been a "pronounced" decline in cardiovascular deaths in the 1970s (p. 3). During the 1969 to 1977 period, cardiovascular death rates decreased for all age groups among men "with the smallest reduction (10%) at ages 35-39" (p. 3). Women, similarly during this period, showed a 16% decrease at ages 75 and over and largest decreases of 36% at ages 20-24 (p. 3). The Statistical

Bulletin stated specifically in regard to ischemic and related heart disease the following:

Although the death rates were at a somewhat lower level, the mortality experience from ischemic and related heart disease paralleled the downward trend noted for all cardiovascular disease. During the 1969-1977 period, age adjusted death rates increased from 329 to 264 per 100,000, or by 20 percent, among white men in the general population, and from 154 to 117 per 100,000, or by 24 percent, among the women. (Metropolitan Life Insurance Co., 1979, p. 3)

The Statistical Bulletin identified several factors that are generally agreed to have contributed to this decline which include public education campaigns, particularly in regard to smoking, hypertension and cholesterol, better diagnostic techniques, and specialized coronary care units. The Statistical Bulletin noted that

Increased detection and treatment of hypertension is considered a major factor in declining mortality, while the reduction in smoking and the decrease in the use of high cholesterol foods have also been important. (p. 3)

Similarly, Heart Facts 1980, published in 1979 by the American Heart Association, stated that "1968-1977 age-adjusted death rates for coronary heart disease, stroke, hypertension, and rheumatic heart disease are all on a significant decline" (p. 14). Improved surgical techniques are discussed in Heart Facts 1980 as significant in the management of heart disease.

Despite this decline, heart disease remains the major cause of death in the United States. The fact still

remains that more than 1,500,000 Americans will suffer a heart attack this year and, of these, approximately 650,000 will die (Heart Facts, 1979). Further, the incidence of premature cardiovascular disease is significant. At least one fourth of all persons killed by cardiovascular disease are under age 65 (Heart Facts, 1979).

The following literature review will examine Type A coronary-prone behavior in depth and present the selected major cardiac risk factors only in terms of their significance as risks. Major studies pertinent to Type A behavior will be emphasized.

Historical Review

Jenkins (1975) noted that angina pectoris was described at least 200 years ago, but it has only been in the 20th century that myocardial infarction has been recognized. Jenkins stated that coronary heart disease is historically a "recent phenomenon" and that "the modern epidemic of myocardial infarction is one of our civilization's newer products" (p. 5). Indeed, the epidemic of coronary heart disease has been primarily recognized within the last 50 years, particularly in the countries of the Western world, considered "affluent" nations (DiGirolamo & Schlant, 1974, p. 988).

In the early 1900s Osler described behavioral characteristics of his patients with angina pectoris (Jenkins, 1975, p. 5). Later as the incidence of coronary heart disease emerged others also began to recognize common psychological characteristics of the coronary patient. Menninger and Menninger (1936) described aggressive traits of coronary patients that were often repressed. In the 1940s Dunbar (cited in Verghese, 1971) recognized that postmyocardial infarction patients displayed specific traits she described as a coronary personality, which often included traits such as "asceticism and hard work," "calm surface with little apparent strain," "air of self-sufficiency," "domination in social occasions," "quick decisiveness," and "threat to authoritative role" (p. 9). As these characteristics emerged and were published, more specific details of the coronary patients' personality profile emerged. Arlow (cited in Verghese, 1971) in 1945 noted that frequently a patient with coronary heart disease would display a faulty identification with his father. Kemple (cited by Jenkins, 1975) described coronary patients as having "limited introversive experiences, limited creative thought, a constriction of imagination and a lack of sensitivity to subtle nuances within their environment" (p. 5). Gildea (cited in Verghese, 1971)

noted that coronary patients showed a "great need for respect and authority" and tended to have a "planned career" (p. 10).

Romo, Siltanen, Theorell, and Rahe (1974) summarized their historical review of coronary behavior noting that from the time of Dunbar forward over the next 30 years numerous psychological studies were carried out which frequently used small numbers of subjects and often were "inconsistent" and reported "negative results" (p. 2). They reported "generally consistent and positive" results as clinical interviews emerged for study of coronary patients (p. 2). These authors noted the

Most consistently reported behavior dimension of CHD subjects was their dedicated approach to their work--aggressive intense, dependent upon objective signs of achievement, long hours of overwork, with a tendency of subjects to work even during their vacation times. (p. 2)

In the late 1950s Friedman and Rosenman first began their study of the role of behavior in the etiology of coronary heart disease. These physicians coined the term Type A and B coronary prone personality. In their book, Type A Behavior and Your Heart, Friedman and Rosenman (1974b) recalled that initially they began to ponder whether they were truly helping coronary patients with their "therapeutic regimen" which in the mid and late 1950s rarely included assessment or intervention in regard to

behavior by themselves or others in their field (Friedman & Rosenman, 1974b, pp. 53-66). While writing an article on the role of dietary cholesterol in the development of coronary heart disease, Friedman and Rosenman (1974b) recognized that cholesterol frequently failed to explain why persons who consumed diets high in fat and cholesterol failed to get coronary heart disease (p. 55). In fact, while studying the dietary habits of a group of male and female married subjects who both consumed the same diets, they began to document the inconsistencies related to cholesterol theory. The lower incidence of coronary heart disease in white females was and is generally attributed to females being protected by hormones until menopause. Friedman and Rosenman (1974b) reject this theory stating that "white women of various countries other than in the United States are as prone to coronary heart disease as their husbands" (p. 56). They further noted that several studies done in the different areas of the United States have shown the "black woman to be slightly 'more' susceptible than the black husband to coronary heart disease" (p. 56).

From this point in the late 1950s forward, Friedman and Rosenman (1974b) have worked to define the role of behavior, particularly Type A behavior, in relation to coronary heart

disease. Their early efforts and those of others were greatly hampered by the lack of a suitable tool to measure personality, particularly Pattern A, until very recently. Other difficulties involved in behavioral research along with the large number of retrospective studies that were inherent in early attempts to study behavior also contributed to the lack of acceptance of Type A behavior.

In the 1960s Friedman and Rosenman (1974b, pp. 80-81) developed the standard interview technique to determine Type A behavior. This provided the first really valid means to isolate the behavior and it still remains the single best methodology.

In the late 1960s, two major paper-pencil techniques were introduced to determine Pattern A behavior. These tools and the standard interview technique, discussed later in the chapter, greatly contributed to reliable means of documenting Pattern A behavior. Many studies during this period reflected efforts to validate these tools after some revisions and in different populations. Major prospective studies related to Type A behavior were launched in the late 1960s. The most significant prospective study to date is the Western Collaborative Group Study which was begun in 1960 (Friedman & Rosenman, 1974b). Also, during the decade of the 60s, numerous studies were produced that

reflected efforts to define Pattern A as well as the role of stress and life dissatisfaction.

Jenkins in 1971(b) reviewed over 160 papers related to psychologic and social factors of coronary heart disease. He summarized his review of the extensive data by stating that "the time has come to shift the emphasis from descriptive data gathering to tightly designed inferential studies" (p. 316).

Theorell and Rahe in 1972 summarized the major psychological studies of coronary heart disease patients into two major groupings. First are the studies which have focused on stressors occurring to coronary heart disease subjects shortly preceding their development of angina or myocardial infarction. "The second group of psychological studies of coronary heart disease subjects has attempted to delineate more or less specific and often long-standing behavioral and life satisfactions characteristic of these individuals" (Theorell & Rahe, 1972, p. 140).

The literature encompasses a massive amount of research related to behavior and heart disease. Data related to stress, life dissatisfactions, social and psychologic precursors of heart disease is overwhelming. Therefore, discussion of Pattern A behavior will be limited to the typology as an entity only.

Definition: Type A and B Coronary Prone
Behavior Pattern

In addition to the definition presented in Chapter 1, Pattern A behavior is described by Jenkins (1971b) as:

The overt behavioral syndrome or style of living characterized by extremes of competitiveness, striving for achievement, aggressiveness (sometimes stringently repressed), haste, impatience, restlessness, hyper-alertness, explosiveness of speech, tenseness of facial musculature and feelings of being under the pressure of time and under the challenge of responsibility. Persons having this pattern are often so deeply committed to their vocation or profession that other aspects of their lives are relatively neglected. Not all aspects of this syndrome or pattern need be present for a person to be classified as possessing it. The pattern is neither a personality trait nor a standard reaction to a challenging situation, but rather the effect of a challenging situation on a characterologically predisposed person. Different kinds of situations evoke maximal reaction from different persons. (p. 309)

Friedman and Rosenman (1974b) have described the pattern as "an action-emotion complex" in which coronary prone subjects become engaged in a "chronic, incessant struggle to achieve more and more in less and less time," and frequently "against the opposing efforts of other things or persons" (p. 67). Type B persons are the opposite of Pattern A individuals and are generally described as unhurried, easy-going, relaxed, and more self-satisfied.

Jenkins (1975, p. 6) stated that it is very important to distinguish Pattern A behavior from stress per se. He noted that Pattern A behavior differs in concept from

stress in that it is a "style of overt" behavior that involves a "challenge" to the Type A individual whether the situation be pleasant or otherwise. The Pattern A behavior should also be distinguished from "anxiety, depression and neurosis" as cited by Jenkins (1975, p. 7) since these factors correlate only minimally. Depression, anxiety, and neurosis are significant in terms of recovery from a heart attack and as related to angina but are not significant in describing Pattern A behavior.

Glass (1977) reported that Pattern A individuals "exert greater effort" to succeed, to control perceived threats to their environment, and that they "suppress subjective states (like fatigue) that might interfere with task performance" (p. 72). He stated also that Pattern A individuals tend to pace rapidly their activities and often become hostile if interrupted from completing a task (p. 72).

In a series of clinical experiments, Glass (1977) found that Pattern A subjects try to control their environment. They will initially respond in a "hyperresponsive" manner to a challenge. However, Glass noted that as they perceive a challenge to be uncontrollable, Pattern As become "hypo-responsive," more so than Pattern B subjects at this point (pp. 72-92).

Pattern A subjects were found to be more stressed than Pattern B subjects by a loss of control and exhibited greater "learned helplessness" when faced with an uncontrollable stressor (Glass, 1977, p. 127). Glass speculated that catecholamine release resulting from the increased response to stress of Pattern A subjects may be the mechanism that promotes coronary disease (p. 127). He also noted that results of these studies need replicating and have some methodological weaknesses (p. 168).

The Glass experiments are significant not only as they contribute to the definition of Pattern A behavior, but also because they provide true clinical experiments aimed at providing a conceptual framework of Pattern A behavior. Glass (1977) has emphasized that the coronary-prone behavior pattern is not the same as the coronary personality. He based this distinction on the fact that "personality traits do not lead to behavioral or physiological responses by some invariant process" (p. 24). Pattern A is an overt response that results "from the interaction of a specific set of predispositions with appropriate eliciting situations" (p. 24). The key to this distinction lies in the overt response of Pattern A subjects to a perceived challenge. Personality traits alone may not necessarily evoke behavioral or physiological responses.

One final distinction must be made in regard to the definition of Pattern A behavior. A very similar pattern, termed the "Sisyphus pattern" by Adsett, Bruhn, Paredes, and Wolf (1974) exists in the literature. The Sisyphus pattern is characterized like Pattern A by extreme "effort" orientation and "striving against odds but with little sense of accomplishment or gratification" (p. 187). The difference these authors make between the Sisyphus reaction and Pattern A behavior is that the Sisyphus reaction includes the "absence of a reward or emotional fulfillment" (p. 187). Adsett et al. (1974) in a 10 year perspective study of 65 subjects with well documented myocardial infarctions and 65 control subjects found a close correlation between the Sisyphus pattern and Pattern A behavior and also found that both were "predictive of a poor prognosis in those subjects who had suffered a myocardial infarction in the past (p. 187).

Etiology of Pattern A Behavior

As previously discussed, the most affluent industrialized nations of the Western world exhibit the highest rates of coronary artery disease. Also, the greatest increase in coronary heart disease has occurred essentially within the last 40 to 50 years in industrialized nations. Social and cultural factors related to stress

theory have been implicated. Indeed, the Western world promotes competition, achievement, time-consciousness, aggressiveness, and rapid performance.

A classic example of the effects of social and environmental effects on the incidence of heart disease are documented by Bruhn and Wolf in The Rosetto Story (1979). The inhabitants of the Italian community of Rosetto, Pennsylvania were noted to have unusually low rates of heart disease despite significant incidence of obesity, smoking, and other classic cardiac risk factors. As the community became modernized and cultural traditions and closeness were replaced by modern technology and stresses, the incidence of coronary heart disease significantly increased. Bruhn and Wolf attribute this result to "life stresses" and "Americanization" (p. 143).

Boyer (1974) has suggested that risk factors for heart disease may be present in children and pre-teens. He noted that the "applicability of adult coronary risk factors as predictive indexes in children have not been substantiated in long term studies" (p. 785). He did not speak in his article to stress or behavior patterns as factors. However, Russek (1974) has suggested that children are subjected to stresses for 12 to 20 years in educational systems which promote competition, demanding assignments, and

exceptional achievement with little attention or allowance for "living" (p. 122).

In regard to student behavior, Parfenbarger, Wolf, Notkin, and Thorne (cited in Verghese, 1971) found that:

College students who had a history of non-participation in athletics, early parental death, only child status and sociopsychological exhaustion at times of stress in college showed a tendency to get coronary heart disease later in life. (p. 11)

Glass (1977) reported that there is little data regarding the possibility of genetic predisposition to Pattern A behavior and little data on how the pattern actually emerges psychosocially (p. 141). Glass (1977) reviewed numerous studies as well as results of his clinical experiments in regard to possible antecedent precursors of Pattern A behavior. He concluded that theory for development of Pattern A behavior is speculative at this point. Data suggest that although the personality may not be genetically transmitted, perhaps high-energy states may be (p. 150). Further, some data suggest that certain child-rearing practices or modeling may produce Pattern A behavior (p. 162). These conclusions, again, are highly speculative in this early stage of study.

Incidence and Prevalence

Friedman and Rosenman (1974b) pointed out that behaviors tend to run together. That is, individuals possess some Pattern A behavior and some Pattern B behavior, but are generally easily identifiable as one or the other. They have found that most "urban Americans" are Type A or B and that Type A individuals predominate, or "represent over half of all those in the open samples" they have tested (p. 68). Of these, Friedman and Rosenman estimated that 40% are Type B. Mixed A and B patterns account for about 10% (p. 68). Jenkins (1975) stated that Types A and B "represent the extremes of a bipolar continuum that is probably normally distributed in the U.S." (pp. 18-19).

Friedman and Rosenman (1974) have also documented the increased prevalence of coronary heart disease in both males and females (p. 272). Prior to 1969 little data existed in regard to personality much less Type A behavior in females (Finn, Hickey, Mulcahy, & O'Doherty, 1969, p. 339). A study of Type A behavior and hypertension among "inner-city" black females showed "reliability of the A-B classification for the sample and demonstrated a general congruence with Type A behaviors reported in previous (mostly white) samples" (Smyth, Call, Hansell, Sparacino, & Strodbeck, 1978, pp. 30-35).

Jenkins (1975) has stated that "the ratio of Type A risk to Type B risk of coronary disease is highest among younger persons and decreases with age" (p. 19). The standard cardiac risk factors of cholesterol and cigarette smoking follow this same trend.

Jenkins (1975) has also stated that after age 65 no published data exist as to the significance of Type A behavior as a risk factor (p. 19). Friedman and Rosenman (1974b) note that coronary heart disease rarely occurs prior to age 70 "regardless of fatty foods eaten, the cigarettes smoked, or the lack of exercise, unless Type A behavior exists" which is associated with the incidence of coronary heart disease in individuals in their 30s and 40s (p. ix).

Psychological Characteristics

Psychological manifestations of Pattern A subjects are extensively documented in the literature. Jenkins (1975) has emphasized that this overt behavior pattern is best seen when the Pattern A subject is engaged in a challenging or frustrating activity and may be difficult to assess in the Pattern A subject who is "bored" or who has "disengaged himself from interacting with his surroundings" (p. 11). Friedman and Rosenman (1974a) emphasized Pattern A "does not 'solely' stem from an individual's

personality but emerges when challenges or conditions of the milieu" arise to illicit the behavior (p. 271). Pattern A could "lessen or even disappear" in the absence of such a "challenge" (p. 271).

Type A individuals exhibit "enhanced personality traits" such as aggressiveness, ambition, time urgency, chronic impatience, a strong-work orientation, and often a preoccupation with deadlines (Friedman & Rosenman, 1974a, p. 270). They are usually quite competitive and may exhibit hostility if hampered in their efforts.

Pattern A subjects often become involved in stressful work conditions which "put pressure on them to be productive" and are "over-burdened with responsibility" (Mahapatra, 1972, pp. 568-569). They frequently display restlessness and impatience as well as "intense dedication" to their jobs (Kenigsberg, Zyzanski, Jenkins, Wardwell, & Licciardello, 1974, p. 344).

Friedman and Rosenman (1974b) stated that Type A persons often "indulge in polyphasic thought or performance," may be self-centered, feel guilty when relaxing or doing nothing, often fail to be aware of "interesting or lovely objects" in their milieu, are preoccupied with "having" rather than "being" and constantly evaluate themselves and others in terms of "numbers" (pp. 83-85).

Jenkins (1975) described the "values" of Type A subjects:

1. The Type A subject is conscientious, and responsible, in an intense, inflexible way.
2. The Type A individual prefers being respected for what he does, whereas the B usually prefers to be loved for who he is. This leads to the Type A having to maintain productivity in order to maintain his feeling of self-worth.
3. The Type A individual has more obvious cravings for recognition and power than the Type B but may deny this when asked directly.
4. The Type A individual is compulsively attracted to competition and challenges. He will often compete against himself when no one else is available, trying to better his own "record" either in quality or speed. (pp. 11-12)

In regard to "interpersonal" relations, Jenkins (1975) has further described the Type A as "self-centered" and a "poor listener" who may display anger quickly and become more quickly frustrated, particularly in a work situation, than Type Bs (p. 13). Jenkins (1975) stated that Type A subjects often express "overt bravado" and an air of superiority. However, when faced with failure, the Type A may "reveal the insecurity and feelings of inferiority that lie beneath the surface" (p. 15). The Type A individual may be "more sexually aggressive than the Type B, but it is the chase that he loves most rather than the consumation" (p. 13).

Physical and Motor Characteristics

The literature reveals few physical characteristics of the Type A individual other than those previously documented in Chapter 1. Type A subjects tend to be short, stocky males who are often obese, have elevated cholesterol and triglyceride levels, and frequently demonstrate abnormal glucose tolerance (Netter & Yonkman, 1971, p. 217). Friedman and Rosenman (1974b) in describing their coronary patients have pointed out that they have rarely seen coronary artery disease in "a tall (over six feet), very lean or almost gaunt individual" under age 65 and weighing less than 160 pounds (p. 138). They noted that men who are "better muscled (the anthropologists label such heavily muscled persons 'mesomorphs')" and who "probably do possess a heavier bone structure" appear prone to coronary heart disease "taken as a whole" (p. 139). Specific physiological characteristics will be presented later in this chapter.

A number of motor and speech characteristics have been documented in Type A subjects. These overt characteristics are strongly relied upon in the interview technique developed by Friedman and Rosenman (1974b) to determine Type A behavior. The manner of response of Type A subjects more than the content of their responses is considered diagnostic.

Mahapatra (1972) summarized these motor and speech characteristics as "forceful, rapid and often unduly explosive emphatic speech, restlessness and sudden gestures of fist-clenching or taut facial mannerisms" (p. 569). Type A individuals are often described as hyperalert. They display "brisk and impatient body movements" and fist-clenching in ordinary conversation. Type A persons seldom relax (Friedman & Rosenman, 1974a, p. 271).

Jenkins (1975, p. 15) noted that a Type A subject displays "abrupt movements," has a "strong hand shake" and may "strike a table" with his fist to make a point. Type As will "motorize" or fidget in a "rapid, rhythmic, and repeated" manner which may include foot tapping or knuckle drumming for example (p. 15).

The facial expressions of Type A subjects are closely monitored during the interview assessment of Type A behavior. Type As are noted to flash "tense smiles marked by a lateral pull of the mouth with a tight horizontal lip line" (Jenkins, 1975, p. 15). Type A subjects often reveal a "grimace" that Jenkins (1975) described as "almost a tic" in extremely developed Type A subjects (p. 15). They may reveal tense jaw muscles or gritting of teeth while emphasizing points (p. 15).

Type A subjects are noted to sigh heavily and to take frequent deep breaths during conversations in an impatient

or restless manner. Jenkins (1975) pointed out that this "sigh characteristic" is easily overlooked and that it was originally determined from review of taped interviews of Type A subjects (p. 16).

Clipped and hurried speech patterns have also been noted in Type A persons. The Type A person emphasizes his points with volume and certainty; he/she speaks "to the point" and is impatient with slow conversations. The Type A will hurry slow speakers along by head nodding and other gestures (Jenkins, 1975, p. 14).

Jenkins (1975) has stated that determination of Type A behavior depends on "an intuitive summing of the number of characteristics possessed and their intensity" (p. 16). There are extremes of response and moderate modes of response of Type A subjects. The Type A subject will probably not possess all the characteristics herein listed. However, the individual characteristics of the coronary-prone behavior taken "as a whole" have been shown "to be predictive" (p. 16).

Physiological Manifestations of Type A Behavior

Type A behavior according to Friedman and Rosenman (1974b) is "the major cause of coronary heart disease" (p. 53). The following description details the physiological processes produced by Pattern A behavior as described

in the book, Type A Behavior and Your Heart (Friedman & Rosenman, 1974b, pp. 172-179).

Emotions received in the brain via the neocortex and limbic systems "signal" the hypothalamus which responds by sending out systemic messages based on the type of emotion perceived. Anger, for example, results in the stimulation of the sympathetic nervous system which in turn releases catecholamines, adrenalin and noradrenalin. In addition, the hypothalamus signals the pituitary gland, "the master of all endocrine glands" to release its "exclusively" produced hormones (i.e., growth hormone) (Friedman & Rosenman, 1974b, p. 174). The thyroid, adrenals, sex glands, and pancreas are in turn signaled and release their hormones.

Friedman and Rosenman (1974b) have stated that Type A behavior results in a "chronic excess discharge of these various hormones" (p. 175). The Type A subject has been found to discharge more catecholamines and more adrenocorticotrophic hormone (ACTH) produced by overstimulation of the pituitary gland. Adrenocorticotrophic hormone (ACTH) stimulates the adrenals which then discharge cortisol, growth hormone and other hormones (p. 175). Type A subjects also demonstrate more circulating insulin and abnormal metabolism of fat and sugar (p. 175).

The net result of these changes is that Type A subjects exhibit:

1. An increased blood level of cholesterol and fat
2. A marked lag in ridding their blood of cholesterol added to it by the food ingested
3. A pre-diabetic state
4. An increased tendency for the clotting elements of the blood (the platelets and fibrinogen) to precipitate out. (Friedman & Rosenman, 1974b, p. 175)

The means by which damage to the coronary arteries occurs from these processes is speculative. Friedman and Rosenman (1974b) have shown that Type A subjects take longer to rid their blood of dietary ingested cholesterol. Therefore, the arteries in Type A subjects are exposed to the detrimental effects of cholesterol continuously.

Another possibility is that the chronic excess amounts of catecholamines that are known to increase the "intra-vascular deposition of clotting elements of the blood" may produce enlargement of existing plaques within the artery wall (Friedman & Rosenman, 1974b, p. 175).

A third possibility suggested by Friedman and Rosenman (1974b) is that the excess circulating catecholamines of Type A subjects may result in "serious narrowing of coronary blood vessels and the plaques appended thereto" (p. 177). The result would diminish blood flow to the coronary arteries and "would threaten the viability of the internal areas" of the existing plaques (p. 177).

Fourthly, Friedman and Rosenman (1974b) have postulated that "because of the hypothalamus-induced overstimulation of

the sympathetic nervous system" excess circulating insulin is present (p. 178). The combined presence of increased insulin as well as abnormal metabolism of fat and sugar is known to cause damage to arteries (p. 178).

In summary, Friedman and Rosenman (1974b) have described four major physiologic changes that are known to result from Type A behavior. Of these, they postulated that the chronic excess of circulating catecholamines probably is most significant (p. 177). The actual means by which the physiologic alterations actually "cause" arterial degeneration is speculative. Four possibilities have been presented as described by Friedman and Rosenman (1974b).

Methods to Identify Pattern A Behavior

The two major criticisms related to research of Pattern A behavior have been few prospective studies and the lack of a valid tool to measure the behavior. Numerous standard tools have been used to determine psychological factors related to coronary heart disease such as the Cattell 16 PF questionnaire (Finn, Hickey, Mulcahy, & O'Doherty, 1969), the Minnesota Multiphasic Personality Inventory (MMPI) (Ruskin, Stein, Shelsky, Bailey, Jefferson Braverman, & Zatzkin, 1970), and the Thurstone Temperament Schedule (Brozek, Keys, & Blackburn, 1966).

In the past, efforts have been made by different investigators to develop new tools to identify psychological variables associated with coronary heart disease and to determine Type A behavior (Friedman & Rosenman, 1974b; Jenkins, 1975). There now exist three standard means for determining Type A behavior that are generally recognized and that have been validated in different populations.

The primary and best means recognized by experts to determine Type A behavior is the standard interview technique developed by Friedman and Rosenman (1974b, pp. 80-81). Overt behavior that includes speech patterns, motor characteristics such as fist clenching and facial expressions as well as the subjects' responses to specific questions are analyzed. It is generally known that the manner in which a Type A subject responds is far more significant than his actual response to the question content of the interview (Glass, 1977, pp. 25-27, 177-178).

The reliability of the interview technique has been determined in several major studies. A complete review of studies that have shown the reliability of the interview is provided by Glass (1977, pp. 25-26, 176).

The Jenkins Activity Survey was developed as a paper-pencil tool to determine Type A behavior and has undergone numerous revisions since it was developed in the mid-1960s.

It is currently considered highly reliable as an instrument to measure Type A behavior and has been shown to closely correlate with results of the interview technique (Rustin, Dramaix, Kittel, Degré, Kornitzer, Thilly, & de Backer, 1976). Glass (1977) provided an in-depth review of the major studies that have documented the reliability of the Jenkins Activity Survey (pp. 177-178).

A third technique to measure Type A behavior has been developed by Bortner. Originally, Bortner devised a performance battery that consisted of a series of cognitive and psychomotor tests to determine Pattern A behavior (Bortner & Rosenman, 1967, pp. 525-533). He later developed a paper-pencil tool that consisted of 14 self-rating scales. The 14 items were composed of two adjectives separated by a 1.5 inch line. Subjects rated themselves by marking a point on the lines. Bortner (1969) found a "significant correlation between the interview classification and the self ratings" (p. 89).

In a major Belgian study (Rustin et al., 1976), the Bortner scale and the Jenkins Activity Survey were compared with the interview technique to determine Type A behavior. The Jenkins Activity Survey and the Bortner Scale were found to be concurrently predictive of Type A behavior. The Bortner Scale correlated significantly with the

interview technique at $p < .001$. Further, the Bortner Scale correctly classified 78% of the subjects tested as Type A. These results are significant for documenting the Bortner Scale reliable a second time against the interview technique, as equivalent to the Jenkins Activity Survey in determining Pattern A, and by replication of results in a different population.

Significance of Type A Behavior as
a Cardiac Risk Factor

Type A behavior remains a controversial cardiac risk factor. The major cause of the controversy can be primarily attributed to the lack of valid means to determine the Pattern until the late 60s and early 70s. Prior to this time numerous standard measures of personality such as the MMPI, for example, had been used to isolate certain characteristics of the coronary personality in a general manner. The advent of the interview technique provided a means for Pattern A to be reliably measured. However, interviewers had to be trained in the techniques and this meant that wide-scale determination of Pattern A was limited. The Jenkins Activity Survey and the Bortner Scale were developed in this general time span and provided "paper-pencil" means to determine Type A behavior. These tools have each undergone several revisions and required

testing in different populations for reliability. The Jenkins Activity Survey is still in revision and a version for students has been developed only recently (Glass, 1977, p. 28). A Children's Activity Survey has also been developed (Glass, 1977, pp. 154-155). Essentially, the means to measure Type A behavior have reached a "prime" only since the early and probably more accurately, the mid 1970s.

The second major criticism of research related to Pattern A behavior has been the lack of prospective studies. The major prospective studies of Type A behavior have emerged since the mid 1960s and are few in number.

The following presentation will provide a review of literature which reflects the significance of Type A behavior as a risk factor. The review is limited specifically to Pattern A behavior and does not encompass other aspects of the coronary prone personality, such as life dissatisfactions or stress in general.

Friedman and Rosenman (1974b) have stated Type A behavior is the major cause of coronary heart disease (p. 53). They also recognized that there are other factors "known to cause" coronary heart disease which include Diabetes Mellitus, hypertension, heredity, hypercholesterolemia, and hypothyroidism (pp. 100-107). Of these factors, they speculated that hypertension may actually be a result of Type A behavior.

Mahapatra (1972) has stated that a clear relationship between psychological factors and coronary artery disease has not been established (p. 568). The issue of Pattern A behavior as a causative variable in coronary artery disease or as one of a multiplicity of factors is widely debated.

Friedman and Rosenman (1974a) have contributed most to the documentation of Type A behavior as a significant risk factor. In a major review article, they summarized results of the major studies related to Type A behavior. Review of the major Friedman and Rosenman results are presented first.

Friedman and Rosenman (1974a) have found Type A behavior to be present in "most patients with coronary heart disease under the age of 60 years" (p. 272). Pattern A behavior has also been associated with increased incidence of coronary heart disease in both males and females (p. 272).

The most significant study and the first large scale prospective study of Pattern A behavior was instituted by Friedman and Rosenman in 1960-1961. The Western Collaborative Group Study (WCGS) included 3500 males, aged 39 to 59 years at intake. "Medical, and socioeconomic histories; dietary, drinking and smoking habits; blood pressure; serum cholesterol; triglycerides and lipoproteins; blood clotting

studies; anthropometric measurements" and behavior pattern were obtained (Friedman & Rosenman, 1974a, p. 272). At 8.5 years of follow-up, initially assessed Type A subjects were found to be more than twice as prone to the development of coronary heart disease than the Type B subjects (Friedman & Rosenman, 1974a, p. 272; Rosenman, Brand, Sholtz, & Friedman, 1976, pp. 903-909). Even more significant, Type A subjects were found to be "five times more prone to have a second myocardial infarct" than the Type B subjects (Friedman & Rosenman, 1974a, p. 272).

Further, it was found that:

While the presence of Type A Behavior Pattern in men with other risk factors (e.g., hypertension, hypercholesterolemia, positive family history) further increased the incidence of coronary heart disease, nevertheless, Pattern A alone and independently appeared to exert a strong pathogenic force. (Friedman & Rosenman, 1974a, p. 272)

From 1960 to 1967, 80 subjects enrolled in the Western Collaborative Group Study died. Of these, 25 died of coronary heart disease. Friedman and Rosenman (1974a) were able to study 51 of these 80 subjects. They found that of the 25 coronary heart disease deaths, 88% of the subjects were known to be Type A. Overall, six times more Type A subjects had died of coronary heart disease than Type B subjects. They also found that coronary atherosclerosis was almost twice greater in Type A than in

Type B subjects, whether the Type A subjects had died of a heart attack, or by accident or other illness (p. 272).

From a physiological standpoint, Friedman and Rosenman (1974b) have also documented changes in blood clotting times and increased cholesterol levels in a group of 42 accountants faced with "occupational deadlines" (p. 59). In other studies, they have found that Type A subjects, male and female, demonstrate higher serum cholesterol levels than Type Bs, and that Type As "have not only a significantly greater fasting but also a higher post-prandial serum triglyceride" level than Type B subjects (Friedman & Rosenman, 1974a, p. 273). Serum cholesterol levels have also been shown to "vary directly with the intensity" of Type A behavior (Friedman & Rosenman, 1974b, p. 59).

Other investigators have also documented Type A behavior as a significant risk factor. Catecholamine and platelet aggregation in coronary prone men have been studied by Simpson, Olewine, Jenkins, Ramsey, Zyzanski, Thomas, and Hanes (1974). They concluded that Type B subjects "who are easy-going with regard to time and flexible in their personal reactions are notably less likely to have blood platelets easily stimulated to the kind of release reactions that result in irreversible clumping of platelets" (p. 485).

Lower levels of growth hormone concentrations were found in Type A than in Type B subjects. The significance of this finding is inconclusive, as the difference found was not apparently "fixed" (Friedman, Byers, Rosenman, & Neuman, 1971, p. 929). Plasma growth hormone is probably related to the maintenance of normal cholesterol concentrations and this finding of lower growth hormone concentrations in Type A subjects is interesting since they are often hypercholesterolemic also.

Adsett et al. (1974) studied 130 subjects, 65 of which were known to have had a "well documented infarction," and 65 control subjects over a 10 year period. The purpose of this prospective study was to make predictions of the likelihood of myocardial infarction or sudden death based on results of the Sisyphus pattern, extreme Type A behavior, and depression. Results of this study showed a close correlation between the Sisyphus pattern and Pattern A. In addition, "both were found to be predictive of a poor prognosis in these myocardial infarction subjects" (pp. 187-191).

Jenkins, Zyzanski, Rosenman, and Cleveland (1971) studied 3000 subjects involved in the Western Collaborative Group Study. Groups of subjects with recent and distant myocardial infarctions, subjects with recurrent

myocardial infarction, and a group of subjects with fatal coronary heart disease were studied using scores obtained from the Jenkins Activity Survey. A major purpose of the study was to test the hypothesis that subjects tested closer to the initial myocardial infarction would "manifest enhanced Type A characteristics" (p. 609). They found that subjects with recent and those with distant myocardial infarctions, "one studied retrospectively and one studied prospectively," both scored highly on the Jenkins Activity Survey (p. 611). Subjects who had "two CHD incidents" scored higher than those with a single event. They also found the Jenkins Activity Survey reliable in discrimination of "recent cases" but found it could not be "relied upon to predict individual cases or even to discriminate between small groups of cases and non-cases, particularly if a CHD episode is more than 5 yr in the past" (p. 611).

Kenigsberg, Zyzanski, Jenkins, Wardwell, and Licciardello (1974) studied a group of male and female patients hospitalized with myocardial infarction along with a group of control subjects. Results of the study showed that coronary heart disease patients, both male and female, scored higher on the Jenkins Activity Survey used in this study to determine Type A behavior than did the control subjects (p. 345).

Efforts to Modify Type A Behavior

The lack of prospective studies and complete definition of Type A behavior as well as controversy over its significance as a cardiac risk factor have resulted in few attempts to modify the behavior. Most efforts have been directed toward refining valid tools for measurement of the behavior and clinical and field research to further clarify the Pattern.

Friedman and Rosenman (1974b) devoted a chapter of their book to guidelines for modifying Pattern A behavior designed for the lay-person (pp. 180-205). The American Heart Association does not specifically mention coronary-prone behavior, but lists stress as a risk factor which "may contribute to cardiovascular disease" (Heart Facts, 1979, p. 16). Specific means to alter stress are not identified.

To date, few specific programs to deal with Pattern A behavior exist. Cardiac rehabilitation programs that include psychological support generally promote modification of stress as a risk factor. A few studies have begun utilizing group therapy that primarily involves behavior modification techniques to alter Pattern A. Rahe, O'Neil, Hagan, and Ranson (1975) utilized "brief group therapy" in a study that involved 60 postmyocardial infarction subjects and found fewer cardiac complications in these subjects.

This study did not include modification of Type A behavior but dealt with educational and emotional support of the subjects.

Other methods that are now being considered or studied to modify Pattern A behavior include relaxation and biofeedback techniques (Patel, 1976). These techniques have been widely used to "manage stress" by numerous investigators (Benson, Marzotta, & Rosner, 1974, pp. 293-301; Kehoe, 1974, pp. 247-262; Shapiro, Schwartz, & Benson, 1974, pp. 279-292). Pharmacologic means to modify Type A behavior have also been suggested that have included primarily the use of psychotropic drugs (Sigg, 1974, pp. 263-276). This approach has met with poor response. Fuller and Eliot (1974, pp. 311-323) have described the use of exercise to modify stress. The literature does not reflect specific modification of Type A behavior using exercise.

The literature primarily deals with means to modify stress and not Pattern A behavior per se. Glass (1977) has noted that "in the long run" Pattern A is "probably maladaptive and entails considerable risk to health" (p. 173). Research is needed to specifically determine appropriate and safe means to modify Pattern A behavior.

Major Selected Cardiac Risk Factors

The major selected cardiac risk factors include age, sex, familial history of premature coronary heart disease, hypertension, elevated serum cholesterol and/or triglycerides, cigarette smoking, glucose intolerance, obesity, and lack of exercise/sedentary living. The following summary defines the singular significance of each risk factor. However, it is well known that the number of risk factors and the severity of each places an individual at greater risk (Heart Facts, 1979, p. 15). In fact, the combination of certain of these risks is often more than simply additive.

For example, cigarette smoking is associated with a three to five fold increase in relative coronary risk and a cholesterol level above 275 mg/dl with a three to fivefold greater risk than a cholesterol level less than 225 mg/dl. When these two risk factors are present in the same individual the coronary risk becomes fourteen to sixteen times (instead of six to nine times) greater than an individual free from these two factors. (DiGirolamo & Schlant, 1974, p. 992)

Results of the Framingham and other major epidemiological studies have shown the "synergistic" effect of these major factors. The Coronary Risk Handbook prepared by the American Heart Association, based on the Framingham Study data, provides tables for predicting the risk of singular and combinations of factors based on age and sex (American Heart Association, 1973).

The National Cooperative Pooling Project sponsored by the American Heart Association combined data of eight long term prospective studies of white males (Stamler, 1971). These subjects were free of coronary heart disease at the beginning of the study. Over a 10 year period, cholesterol, diastolic blood pressure and cigarette smoking were studied in regard to development of coronary heart disease and CHD mortality. Results showed that the presence of a single risk factor contributed to a 2.4 or 140% probability of subjects having a major coronary event within 10 years. The risk of death in that same period with a single risk factor was double. In the presence of two risk factors, a "four fold" risk for a major coronary event was found and a "threefold" risk for death. Subjects with all three risk factors were found to have an "eightfold" risk of development of a major coronary event and a "fivefold" risk for death (Stamler, 1971, pp. 44-53).

Dolder and Oliver (1975) studied 240 survivors of myocardial infarction aged 40 years or less in nine countries. Risk factors varies in developed as opposed to underdeveloped countries. In the seven developed countries studied, including a center in Los Angeles, "a high prevalence of risk factors, particularly hyperlipidemia and cigarette smoking" were found (p. 493).

The individual significance of the nine selected major cardiac risk factors are presented in terms of their significance. The purpose of this literature review is to summarize the generally accepted criteria of their importance.

Age

Atherosclerosis progresses with age. As previously documented, heart attack death rates increase with age (Metropolitan Life Insurance Co., 1979, p. 3). Heart Facts Reference Sheet (1979) published by the American Heart Association stated that almost one-fourth of all heart attack deaths occur before age 65. Age is a "non-modifiable" risk factor, according to DiGirolamo & Schlant (1974, p. 990). The extensiveness of atherosclerosis in regard to age is significantly related to the presence and number of other cardiac risk factors.

Sex

Men demonstrate more coronary heart disease than do women of child-bearing age (DiGirolamo & Schlant, 1974, p. 990). Hormonal changes in women after menopause produce significant increases in the incidence of coronary heart disease in women, "but never reaches that of men" (Heart Facts, 1979, p. 15). DiGirolamo and Schlant (1974) stated that the differences between sexes appears to be more marked in the white than the Negro population.

Of the many reasons presented for a sex difference in susceptibility to atherosclerosis, the protective effect of estrogen, the differences in blood lipids and hematocrit, the reduced risk of cigarette smoking, and a more sheltered mode of life have been proposed. There is no proof, however, for any of these, except for a modest effect of estrogen on the β - and α - lipoproteins. (DiGirolamo & Schlant, 1974, p. 990)

Family History of Premature Coronary Heart Disease

Sokolow and McIlroy (1977) stated that a positive family history may include genetic tendencies toward hypertension, hyperlipidemia, diabetes and also environmental and life style variables (p. 124). The importance of a positive family history is probably underestimated as genetic research in this regard is still in its infancy.

There is no evidence that atherosclerosis or resultant coronary artery disease is hereditary although a "tendency" towards such can be inherited (Heart Facts, 1979, p. 15). Certain families frequently demonstrate a predisposition or a "tendency" to increased susceptibility to coronary heart disease. DiGirolamo and Schlant (1974) stated that "even though a familial tendency may be influenced by genetic transmission," the effects of other risk factors and environmental and socioeconomic influences related to a genetic tendency are unknown as well as the mechanism of transmission and if such a tendency is modifiable (p. 990).

Individuals whose parents or siblings are affected by heart disease prior to age 50 are at greater risk themselves to "develop coronary atherosclerosis at a younger age" (DiGirolamo & Schlant, 1974, p. 990). For some individuals the risk may be as high as five to one (Fredrickson & Levy, 1972). Slack (1969) has found that the coronary death rate is three times normal in males below 55 years who had a "first-degree" male relative who died of coronary heart disease before age 55 (p. 1380). Similarly, the risk is five times normal in women below age 65 who had a first-degree male relative who died of coronary heart disease before age 55 (Slack, 1969, p. 1380).

Hypertension

The most universally accepted cardiac risk factor is hypertension. Heart Facts (1979) indicated that one in four adults demonstrates elevated blood pressure and that hypertension has been documented in children as young as four years (p. 2). More than 34,290,000 adults are known to have hypertension in the United States (Heart Facts, 1979, p. 24). Resultant strokes accounted for 181,934 deaths in 1977 and the Heart Association estimates that 1,820,000 Americans "are afflicted" by stroke (Heart Facts, 1979, p. 10).

Hypertension is generally defined as pressures in excess of 150/90 mm Hg (Orgain & Gunnels, 1974, p. 1229). Some increases in blood pressure are expected with age. However, Orgain and Gunnels stated that regardless of age or sex, there is almost general agreement that diastolic blood pressures above 100 mm Hg are abnormal.

Hypertension has been vigorously attacked as a cardiac risk factor by all-out efforts of the American Heart Association that have included massive public education and screening programs. Statistics indicate a significant decline has occurred in the incidence of hypertension as well as stroke and coronary heart disease (Heart Facts, 1979, p. 14). However, hypertension ranks second in incidence only to coronary heart disease and according to the American Heart Association, occurs in one of every four adults (p. 24).

Eknoyan and Jackson (1978) summarized the results of a Department of Health, Education, and Welfare study in 1962 that shows the 10 year mortality rates at different blood pressures. The authors report that the following numbers "have not changed much" since 1962:

When the diastolic blood pressure is 85 to 94 mm Hg, the mortality rate is 60 percent higher than that for subjects who have diastolic pressures of less than 85 mm Hg. When the diastolic pressure ranges from

95 to 104 mm Hg, the mortality rate is 60 percent higher than the rate of those people with diastolic pressures of 85 mm Hg or less. The major impact occurs when the diastolic pressure is over 105 mm Hg, at which level the mortality rate is three times normal. (Eknoyan & Jackson, 1978, p. 138)

Hypertension is twice as prevalent in blacks than in whites and tends to be "twice" as severe in blacks (Eknoyan & Jackson, 1978, p. 138). The cause of this variation is not clear (Tuttle, 1974, p. 1162). In general, males are affected more, possibly due to their higher rates of coronary artery disease (Sokolow & McIlroy, 1977). Mortality rates are higher among younger hypertensives (Sokolow & McIlroy, 1977). Overall, the mortality rates for hypertension are "several times normal" (DiGirolamo & Schlant, 1974, p. 991).

Elevated Cholesterol/Triglycerides

Cholesterol and triglycerides are present in humans via dietary intake and biosynthesis (Lipids, 1973, p. 3). The plasma lipids which consist mainly of "cholesterol, triglyceride, phospholipid, and free fatty acids" are insoluble in water and are carried via lipoprotein molecules of which there are four major classes (DiGirolamo & Schlant, 1974, pp. 993-994). Of the lipids, cholesterol has generally received more attention simply because more is known about it to date (Netter & Yonkman, 1971, p. 217).

Elevation of cholesterol is generally considered when in excess of 220 mg/100 ml and triglycerides when in excess of 140 mg/100 ml (DiGirolamo & Schlant, 1974, p. 991). The Fredrickson-Lees method classifies five major types and sub-types of hyperlipoproteinemias (DiGirolamo & Schlant, 1974, pp. 990-991, 993-996; Fredrickson & Lees, 1965, pp. 321-327). This widely used method provides norms for specific age ranges as well as description of the component combinations of the hyperlipoproteinemias. Most hyperlipoproteinemias are "acquired" whereas some are considered truly genetic in origin (Hurst & Logue, 1974, p. 1052).

High fat consumption may increase cholesterol and triglyceride levels. In fact, premature coronary heart disease is more prevalent in the more affluent countries where diets are high in fat content (Stamler, 1972, p. 65). The American Heart Association (Heart Facts, 1979) reports that "a man with a cholesterol of 250 or more has about three times the risk of heart attack and stroke of a man with a cholesterol of 194" (p. 24). Tibblin, Wilhelmsen, and Werko (1975) in a study of risk factors in a Swedish population noted that cholesterol was a significant risk factor in the development of ischemic heart disease, and they also found that an elevated cholesterol was even "more strongly associated with fatal ischemic heart disease" (p. 521).

Cigarette Smoking

Persons who smoke have twice the risk of non-smokers for heart attack (Heart Facts, 1979). DiGirolamo & Schlant (1974) noted that the risk of developing "CAHD or the risk of death from CAHD is two to six times higher in smokers than nonsmokers, and the risk appears to be proportionate to the number of cigarettes smoked per day" (p. 991). Persons who smoke 20 or more cigarettes per day are at three times greater risk than non-smokers, cigar or pipe smokers to have a myocardial infarction (Doyle, 1974, p. 1566). "The likelihood of coronary heart disease developing is doubled when smoking is associated with any other risk factor" (Lough, 1975, p. 917). Sudden death, a significant risk to smokers, occurs five times more frequently in persons who smoke greater than 20 cigarettes per day (considered heavy smokers) than non-smokers (Sokolow & McIlroy, 1977, p. 124).

International data summaries according to Stamler (1972) indicated correlations of coronary heart disease are higher in women than in men in the populations studied (p. 81). Recent data regarding cigarette smoking showed that teenage smoking is significant because teenagers begin smoking at an early age, usually about 12 years, and they smoke more (McIntosh, Entman, Evans, Martin & Jackson, 1978, p. 145).

The 1973 Surgeon General's Report best summarized the risks and effects of smoking as follows:

Cigarette smoking acts independently of and synergistically with the other CHD risk factors to greatly increase the risk of developing coronary heart disease. The risk of developing CHD for pipe and cigar smokers is much less than it is for non-smokers.

Autopsy studies have demonstrated that aortic and coronary atherosclerosis are more common and severe, and myocardial arteriole wall thickness is greater, in cigarette smokers than in non-smokers.

Experimental studies in humans and animals suggest that cigarette smoking may contribute to the development of CHD through the action of several independent or complimentary mechanisms: The formation of significant levels of carboxyhemoglobin, the release of catecholamines, inadequate myocardial oxygenation which may contribute to acute thrombus formation. There is evidence that cigarette smoking may accelerate the pathophysiological changes of pre-existing coronary heart disease and therefore contribute to sudden death from CHD. (Surgeon General's Report, 1973, p. vii)

Doyle (1974) stated that in addition to catecholamine release, free fatty acids are mobilized with cigarette smoking and platelet adhesiveness is enhanced (p. 1564). Animal experiments have produced rises in blood glucose after injection of nicotine. This finding however is not concrete as yet in human research (Doyle, 1974, p. 1564). McIntosh et al. (1978) noted that the effects of the synthetic material added to cigarettes since the early 70s is totally unknown in regard to cardiovascular disease or cancer.

The literature varies as to the effects upon risk by those who quit smoking. Generally, cessation of smoking

results in decreased risk (Doyle, 1974, p. 1565; Surgeon General's Report, 1973).

Glucose Intolerance (Diabetes Mellitus)

The American Heart Association identified Diabetes Mellitus as a major cardiac risk factor. Diabetes or a family tendency towards diabetes places an individual at increased risk for heart attack and other cerebral and peripheral vascular diseases (Heart Facts, 1979, p. 15). The singular significance of diabetes is difficult to assess since diabetics frequently demonstrate obesity, hypertension and hyperlipidemia.

Persons with diabetes generally present with more extensive coronary disease and at an earlier age than non-diabetics (Epstein, 1967, p. 609; Netter & Yonkman, 1971, p. 217). Nondiabetic females are less susceptible to coronary disease than nondiabetic males. Diabetic females, however, lose this advantage (Arteriosclerosis, 1971, p. 1). Interestingly, an increased incidence of "abnormal glucose tolerance and elevated blood sugar levels" have been noted in patients with coronary heart disease (Arteriosclerosis, 1971, p. 13).

Obesity

Obesity according to Hurst and Logue (1974) "does not contribute directly to atherosclerosis" (p. 1055). Obese

persons are prone to the development of Diabetes Mellitus, hypertension and hyperlipemia. Gotto, Nichols, Scott, Foreyt, and Jackson (1978) found that obesity has a direct effect upon hyperlipemia and hypertension and "may also be related to diabetes mellitus, exercise, stress, personality type and cigarette smoking" (p. 132).

Obesity is defined as weight 10% above the standard for persons of the same age, sex and race according to Hurst and Logue (1974, pp. 1055-1056). Numerous large scale studies by insurance companies have established norms for weight as well as the degree of risk for obesity. The Metropolitan Life Insurance Company's (1969) Table of Desirable Weights is generally accepted and appears in the Coronary Risk Handbook published by the American Heart Association (1973) for determination of obesity as a risk factor (pp. 32-33).

Lack of Exercise/Sedentary Living

Heart Facts (1979) stated that persons who lead sedentary life styles are at greater risk of heart attack than persons who regularly exercise (p. 16). Sedentary life styles contribute to other negative effects such as "chronic caloric imbalance and obesity--20% among teenagers and 50% among middle-aged adults in the United States" (Stamler, 1972, p. 67).

Proponents of exercise speculate that regular exercise promotes collateral circulation and improved myocardial oxygenation (Netter & Yonkman, 1971, p. 217). Alexander, Fred, Wright, Turell, Jackson, and Jackson (1978) stated that regular exercise

improves cardiac function, lowers blood pressure, reduces fasting and postprandial hyperlipidemia, decreases blood glucose values, increases fibrinolytic activity, retards platelet aggregation, and results in weight loss if caloric intake remains constant. (p. 141)

There is no specific evidence that exercise "prevents or delays" coronary heart disease (Alexander et al., 1978, p. 141). Many studies regarding exercise to date have been criticized because of lack of control of other risk factors and generalizations made from study populations for dissimilar populations (Alexander et al., 1978, p. 142). Cooper (1972), who is a noted leader in aerobic exercise research, has stated that "most" studies relating exercise and heart disease have been retrospective and that efforts to "quantify" the effects of exercise are only recently possible (pp. 61-63).

Exercise is a controversial risk factor as are several of the other factors. Although the merits of exercise are debated, it is generally agreed that a sedentary life style produces negative cardiac effects.

Summary

The latest facts related to the decreased incidence of coronary heart disease have been presented. An in-depth review of the literature related to Type A behavior has been presented in terms of a historical review, definition, discussion of etiology as well as incidence and prevalence. Psychological, physiological, physical and motor characteristics common to Type A persons have been described. Methods to identify Type A behavior as well as its significance as a cardiac risk factor have been reviewed. The selected major cardiac risk factors have been presented in terms of their significance as risk factors.

CHAPTER 3

PROCEDURE FOR COLLECTION AND TREATMENT OF DATA

Chapter 3 provides discussion of the standard components used for collection and treatment of data which include the research design, the study setting, the population, sample, and methods used to protect human subjects. The Cardiac Risk Factor Assessment Tool (CRAT) and the Bortner Self-Rating Scale to determine Type A behavior are presented and documentation of appropriate validity and reliability is discussed. Sequential steps used to collect the data and problems encountered therein are covered as well as the statistical approaches appropriate to the treatment of data.

This study was nonexperimental in nature (Polit & Hungler, 1978, pp. 179-182) and was descriptive correlational in design (Polit & Hungler, 1978, pp. 185-186). The study was designed to determine the association between Type A behavior and the standard cardiac risk factors.

Setting

Permission was obtained from three private general hospitals in the southwestern United States in a city of approximately 844,000 people. The bed capacity of the

hospitals utilized averages from 300 to 450. These hospitals all have facilities to care for cardiac patients.

Population and Sample

Thirteen convenience selected postmyocardial infarction subjects were studied. The sample contained patients over 18 years of age of both sexes who were at least 8 days postmyocardial infarction. Patients with known psychological disturbances diagnosed by a physician were excluded from the study. Further, patients were not studied if they were: (a) not fully alert and cooperative, (b) experiencing any pain or distress, (c) being monitored, (d) requiring oxygen, an IV or any other prophylactic or supportive device, or (e) if they were not ambulatory.

Protection of Human Subjects

The study was approved by the Texas Woman's University Human Research Review Committee and the Texas Woman's University Graduate School (Appendix D). Permission was obtained from the three participating agencies and cardiologists or primary internists as appropriate (Appendix D). In addition, the investigator obtained permission from the participants' primary internists in cases where the cardiologists were serving as consultants and not the primary physicians of the participants. Participants were given an

oral description of the study and completed the written consent form prior to participation (Appendix B).

All participants were asked to sign this consent form which included explanation of their rights. Participants were given a brief explanation of the study along with instructions. The investigator was available to answer any questions. Participants were informed that they were free to withdraw from the study at any time and that their participation was voluntary.

The anonymity of participants was explained. Anonymity was assured in that the participants' (patients, cardiologists or, where applicable, primary internists, and hospitals) names were in no way used. Permission forms were kept in a locked file. Code numbers were used to label the forms 1 through 50, and letters A, B, and C were used to replace the names of the hospitals used. This was done only to allow the investigator to correlate the various parts of the tool and to assure anonymity of the hospitals participating in the study.

Instruments

Two instruments were used: The Bortner Self-Rating Scale, a standardized instrument, and the Cardiac Risk Factor Assessment Tool (CRAT), designed by the researcher. The CRAT consists of the Investigator's Check List,

Part A which is completed by the subjects' cardiologists, and Part B that is completed by the subjects themselves.

The Bortner Self-Rating Scale

A self-rating scale developed by Bortner was used to determine the Type A coronary-prone behavior pattern (Appendix A). The Bortner Scale used in this study is believed to be the last revision completed by Bortner. Correspondence (included in Appendix A, March 18, 1976, July 1, 1976, and July 12, 1976) indicated a conversion of the previously used linear scale used in the 1969 study to the current 0-9 point numerical scale. Methods to score the scale are also provided in this correspondence.

The Bortner Scale is considered highly significant in terms of validity and reliability as a paper-pencil tool, and has been tested against the interview technique which is considered the optimal means for determining Type A behavior (Bortner, 1969; Appendix A, Bortner correspondence, July 12, 1976; Rustin, Dramaix, Kittel, Degré, Kornitzer, Thilly, & de Backer, 1976). In Bortner's study a significant correlation between the interview classification and the Bortner Self-Rating Scale was demonstrated with $r = 0.53$, significant at $p < .01$ (Bortner, 1969). The interview technique, however, is highly impractical for large scale clinical use.

A 1976 Belgian study evaluated the Jenkins Activity Survey (JAS) and the Bortner Scale in relation to the interview technique for classifying Type A behavior. The results showed that both the JAS and the Bortner Scale were concurrently predictable of Type A behavior. The Bortner Scale correlated significantly with the interview technique at $p < .001$ and was found to correctly classify 78% of the subjects tested as Type A behavior (Rustin et al., 1976). The results of the Belgian study are highly significant ($p < .001$) for validating the Bortner Scale a second time against the interview technique and in a different populus.

Jenkins developed and has been refining for several years the Jenkins Activity Survey (JAS). The JAS consists of an interview-questionnaire format and is considered the most promising tool available to measure Type A behavior. The JAS, however, is still in a developmental phase of revision (Appendix A). Jenkins recommended the use of the Bortner Scale for the current research (Appendix A, Jenkins correspondence, October 30, 1975).

Participant instructions for use of the Bortner Scale were simplified by the investigator from those provided in the latest scale developed by Bortner. This was necessary for clarity and because the latest form provided by Bortner

included other data not relevant in this study (Appendix A). The Bortner Scale items as designated by Bortner were not altered (Appendix A).

Scoring the Bortner Scale was done according to directions provided by Bortner (Appendix A). The higher scores indicate more Type A behavior on the 0 to 9 point self-rating scale developed by Bortner.

Cardiac Risk Factor Assessment Tool (CRAT)

In addition to the Bortner Scale to measure the Type A behavior pattern, the Cardiac Risk Factor Assessment Tool (CRAT) was developed to determine the standard cardiac risk factors as well as the basic demographic data of the post-myocardial infarction patients. The primary goal of the investigator in designing the CRAT was to develop a tool to determine the standard cardiac risk factors which include: (a) age, (b) sex, (c) familial history of premature coronary heart disease, (d) hypertension, (e) elevated serum cholesterol and/or triglycerides, (f) cigarette smoking, (g) glucose intolerance (diabetes), (h) obesity, and (i) lack of exercise/sedentary living. In addition, the tool was structured to determine basic demographic data which includes items such as race, occupation, and marital status.

The CRAT consists of the Investigator's Check List used to maintain uniformity in data collection, an introductory page of instructions, Part A completed by the participant's cardiologist or, when applicable, the primary internist, and Part B completed by the participant (Appendix B, CRAT: Part B). Part A of the CRAT has the subjects' cardiologists rate their patients in terms of past and/or present history of hypertension, elevated cholesterol or elevated triglyceride levels. The cardiologists were also asked to specify how many myocardial infarctions, including the patient's current one, had the participant experienced. This information was included to obtain a better description of the sample and to benefit possible future replication of this study.

Cholesterol and triglyceride determinations are often controversial. Therefore, the cardiologists or primary internists, as appropriate, were of great assistance in this research by making available their expert judgment in regard to lipids as risk factors in their patients as they were familiar with their patients historically and at the time of the study.

The second part of the CRAT, Part B, was rated entirely by the participants. These questions were designed to cover the standard cardiac risk factors and also basic demographic data.

Collection of Data

Permission was obtained from the three private general hospitals and from the cardiologists or, where applicable, the primary internists to contact their postmyocardial infarction patients. The cardiologists were asked to participate in the research by completing several ratings of their patients in regard to the cardiac risk factors. Permission was obtained from those patients who agreed to participate in the study prior to administration of the questionnaire.

Participating cardiologists or primary internists were given uniform information and instruction in regard to completion of the Cardiac Risk Factor Assessment Tool (CRAT, see Appendix B). The investigator asked that the participating cardiologists or primary internists abide by the following plan:

1. The investigator placed a copy of Part A of the CRAT in the charts of possible subjects of the participating cardiologists or primary internists. They were asked to sign Part A of the CRAT to signify to the investigator approval to approach the patient to participate in the study and to document the information as valid. If the possible subject did not meet the criteria, the cardiologists or primary internists as appropriate did not complete

this form. Completed forms were placed at the front of the patients' charts for the investigator to collect.

2. The cardiologists or primary internists, where applicable, were asked not to offer information regarding the research to participants other than to explain that the study was to determine factors which may or may not contribute to heart disease if the topic arose.

The questionnaires were administered on a one-to-one basis by the investigator. No time limit was placed on participants to complete the questionnaire, and the average completion time was 10 to 20 minutes.

The Investigator Criteria Check List, a research aid developed by the investigator, was utilized to maintain uniformity in the process of data collection (Appendix B). After completing the Investigator's Check List, the investigator then entered each patient's room in a white lab coat and introduced herself as a Texas Woman's University graduate nursing student. Each patient was asked to participate for the purpose of furthering the knowledge about heart disease. It was explained that the questionnaire consists of items which may or may not contribute to heart disease. The anonymity of each patient was assured and explained according to information listed in the Participant Written Permission Form (Appendix B). Those subjects who agreed

to participate in the research were asked to read, complete, and sign this form. The written permission forms were collected by the investigator before the participants were given the questionnaire. A pen, the questionnaire consisting of Part B of the CRAT and the Bortner Scale (Appendix B) were given to the participants. A comfortable setting was provided. The participants were asked to read the instructions provided and to complete the questionnaire to the best of their knowledge. During the administration of the questionnaire, clarification of instructions or the questionnaire was provided as necessary.

Completed questionnaires were then collected and checked to be sure that no names were on the forms. The body frame of each subject was then noted (i.e., small, medium, large) on each questionnaire. The weights recorded by the subjects on Part B of the CRAT were compared with the estimated frame size to the Metropolitan Life Insurance Company's Tables of Desired Weights (1969, p. 12) to determine normal weights and obesity.

All data were coded, placed on a computer sheet, and then analyzed via computer as appropriate. The cardiologists and hospitals were informed when the study had ceased.

Treatment of Data

The extent that selected standard cardiac risk factors and Type A coronary prone behavior were present in post-myocardial infarction patients was determined by data analysis. The data were analyzed utilizing statistical measures including frequency counts, modes, medians, means, standard deviations, percentages, and cross-classification tables as appropriate in graphic and tabular forms. The association between selected standard cardiac risk factors of age (hypothesis 1), obesity (hypothesis 8) (considered interval/ratio data), and Type A coronary prone behavior pattern in postmyocardial infarction patients was analyzed using the Kendall Tau statistic. The Kendall Tau was used in lieu of the Pearson Product Moment Statistic because of the small sample size and the skewed distribution which dictated the use of this nonparametric technique.

Association between the coronary prone behavior and the remaining risk factors was analyzed using the Fisher Exact test and the Phi Coefficient (Nie, Hull, Jenkins, Steinbrenner, & Bent, 1975, p. 224). These later risk factors--sex (hypothesis 2), familial history of premature coronary heart disease (hypothesis 3), hypertension (hypothesis 4), elevated serum cholesterol and/or triglycerides (hypothesis 5), cigarette smoking (hypothesis 6),

glucose intolerance (Diabetes Mellitus) (hypothesis 7), and lack of exercise/sedentary living (hypothesis 9)--are considered nominal data and were correlated with Bortner Scale scores by categorizing the Bortner scores as high or low. Since the frequencies were placed in a 2 x 2 contingency table (Bortner Scores--High and Low--and present or absent risk factors), and since the smallest expected frequency was less than 5, the Fisher Exact test was the appropriate substitute for the Chi Square test. When the sample size is less than 20, as in the case of this sample population, the Fisher Exact test should always be used in lieu of the Chi Square test. The Phi Coefficient was computed from the results obtained via the 2 x 2 contingency table.

Since all items on the Bortner Scale were not applicable, the Bortner Scale was analyzed as follows. Ratings were summed and divided by the number of items applicable to each subject in order to get an average rating. The average rating was then classified as high or low. Higher scores indicated by Bortner represent more Type A behavior. The average ratings were routinely rounded to the next highest number. High scores were rated as 5 to 9 and low as 0 to 4. The determination of which cardiac risk factor or set of factors most highly associated with

the Type A coronary prone behavior pattern in postmyocardial infarction patients using stepwise regression was not completed since the sample size was too small and the assumption of a normal distribution was violated. Null hypotheses were tested at $p < .05$.

CHAPTER 4

ANALYSIS OF DATA

Chapter 4 provides a quantitative description of the postmyocardial infarction subjects studied. The first two purposes of this study were to determine the extent that the selected standard cardiac risk factors and Type A coronary-prone behavior were present in postmyocardial infarction patients. A third purpose was to determine the association between selected standard cardiac risk factors and Type A behavior in the postmyocardial infarction subjects. The fourth research purpose was to determine which selected standard cardiac risk factors or set of factors best predicted the Type A coronary prone behavior pattern.

Description of Sample

Data obtained from this study of 13 postmyocardial infarction patients were subjected to statistical analysis. This section presents the descriptive analysis of these findings. Medians in place of means are reported when the distributions are skewed. The tables, A through JJ, shown in parentheses indicate those that appear in Appendix E.

The 13 subjects studied were evenly distributed among three participating hospitals (Table A). The majority of

the subjects, 10 (76.9%), were male and 3 (23.1%) were female (Table B). Male and female subjects ranged in age from 31 to 68 years (Table C). The mean age of the subjects was 50.8 years and the mode was 61.0 years. Five (38.5%) of the subjects were in the 31 to 39 years age range, and five (38.5%) were in the 61 to 70 years age range. The remaining three (23%) subjects fell into the 51 to 60 years age range (Tables C and D). Of the subjects, 12 (92.3%) were white and 1 (7.7%) was black (Table E). The majority of subjects, 8 (61.5%), were married, 4 (38.8%) were divorced, 1 (7.7%) was widowed, and none were single (Table F).

The mean height of the sample was 67.4 inches, the mode was 71.0 inches, and the median was 68.5 inches; thus the majority, 12 (92.3%) of the subjects were below six feet (72 inches) in height (Table G). Subjects ranged in weight from 136 to 270 pounds. The median weight was 170.5 pounds (Table H). The heights and weights were compared to the Metropolitan Life Insurance Company's Table of Desired Weight (1969, p. 12) to determine the pounds overweight, obesity. Eight (61.5%) subjects were overweight and five (38.5%) were normal weight (Table I). The range of obesity varied from 7 to 71 pounds. The median pounds overweight were 36.5.

The majority of the 10 male subjects studied were "white collar" workers. Two of these male subjects were retired. Occupations of all subjects included minister, engineers, attorney, architect, housewife, manager, laborer, and one subject had no occupation. Of the three female subjects, one had no occupation, one was a shipping clerk, and one was a housewife. Cross-classification of occupation with the average Bortner Scale scores showed that 10 subjects scored high on the scale and 2 scored low. Of the two low scorers on the Bortner Scale, one was a clerk (female) and one was retired (male).

Only four (30.8%) subjects had a past or known history of hypertension (Table J); one (7.7%) subject had a past history of elevated cholesterol (Table K), and three (23.1%) had a past history of elevated triglycerides (Table L). One subject (7.7%) was found to be currently hypertensive (Table M). Two subjects (15.4%) were found to have elevated cholesterol, but no data were available on two subjects (15.4%) in this category (Table N). Of the subjects, five (38.5%) were found to have elevated triglycerides and five (38.5%) did not. No data were available on three subjects in the category which therefore makes this category more difficult to assess. A bimodal distribution is seen in this category (Table O).

It is descriptively significant that the majority of subjects did not have a history of hypertension (9; 69.2%; Table J), elevated cholesterol (12; 92.3%; Table K), or triglycerides (10; 76.9%; Table L). The majority of subjects were found not to be hypertensive (12; 92.3%; Table M) and not to have elevated cholesterol (9; 69.2%, no data on 2 subjects; Table N). Data are difficult to assess in regard to elevated triglycerides as no data were available on three subjects. However, a "possible trend" was seen for this category since five (28.5%) of the subjects did have currently elevated triglycerides (Table O).

For 11 (84.8%) of the subjects, the present myocardial infarction (MI) was their first. For two subjects (15.4%), the current MI was their second (Table P).

Six subjects (50%) were found to have a positive family history for premature heart disease and six (50%) did not. No data were available on one subject (Table Q). A positive family history of hypertension was indicated by 11 (84.6%) of the subjects (Table R), and only 2 subjects had a family history of diabetes. One (7.7%) subject was found to have diabetes (Table S).

A majority of subjects, 8 (92.3%), were cigarette smokers (Table T). The range was found to be from one to four packs per day for those 8 subjects who smoked

cigarettes (Table U). The number of years those subjects had smoked ranged from 1 to 44 years with a mean of 25.4 years (Table V). A majority, 12 (66.7%), subjects were found not to exercise and experienced a sedentary lifestyle (Table W).

The number of selected standard cardiac risk factors in the sample is reflected in Figure 1. One subject (7.7%) had one cardiac risk factor; four (30.8%) subjects had two; four subjects (30.8%) had three; two subjects (15.4%) had four; one subject (7.7%) had five; and one subject (7.7%) had six cardiac risk factors.

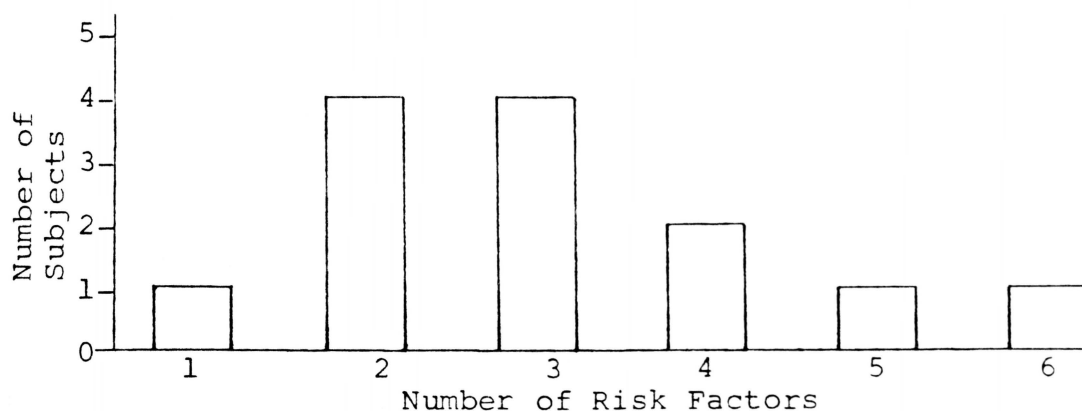


Figure 1. Frequency distribution of the selected standard cardiac risk factors in the sample.

The Bortner Self Rating Scale scores were summed and then divided by the number of applicable items to obtain the average Bortner Scale rating. The higher the score,

the more Type A behavior (Appendix A). These average scores were categorized as high (5-9) or low (0-4).

The range of average Bortner Scale scores in the sample is reflected in Table 1. Of the 13 subjects, 10 scored high on the Bortner Scale and 2 scored low.

Table 1
Range of Average Bortner Scale Scores

Average Bortner Scale Scores	Absolute Frequency	Relative Frequency (Percent)
High (5-9)	10	76.9
Low (0-4)	3	23.1
Total	13	100.0

The frequency distribution of the average Bortner Scale scores in the sample is provided in Table 2. Two subjects scored low on the scale. The lowest low score was at 3. Ten subjects scored high on the scale. The highest score obtained was at 8. The mean average Bortner Scale score was 5.3, the median 5.2, and the mode 5.0. The standard deviation was 1.31 reflecting little deviation and small variation of scores. The standard error was .365 and the variance was 1.71. A .95 confidence interval was

Table 2

Frequency Distribution of Average Bortner Scale Scores^a

Average Bortner Scale Score	Rating	Absolute Frequency	Relative Frequency (Percent)	Cumulative Frequency (Percent)
Low (0-4)	3	1	7.7	7.7
	4	2	15.4	23.1
	5	5	38.5	61.5
High (5-9)	6	3	23.1	84.6
	7	1	7.7	92.3
	8	1	7.7	100.0
Total		13	100.0	

^aMean 5.3, median 5.2, mode 5.0, standard deviation = 1.31, standard error = .365, variance = 1.71, .95 confidence interval at 4.513 to 6.103, data negatively skewed.

Figure 2. Frequency distribution of average Bortner Scale Scores.^a

^aMean 5.3, median 5.2, mode 5.0, standard deviation = 1.31, standard error = .365, variance = 1.71, 95 confidence interval at 4.513 to 6.103, data negatively skewed.

found at 4.513 to 6.103. Figure 2 graphically portrays these findings.

Findings

The hypotheses that no association would be found between Type A coronary-prone behavior and the following selected major cardiac risk factors: (1) age, (2) sex, (3) familial history of premature coronary heart disease, (4) hypertension, (5) elevated serum cholesterol and/or triglycerides, (6) cigarette smoking, (7) glucose intolerance (Diabetes Mellitus), (8) obesity, and (9) lack of exercise/sedentary living were not rejected at $p \leq .05$. Table 3 reflects the statistical techniques utilized and the significance levels obtained for each hypothesis.

The association between the selected standard cardiac risk factors age and obesity and Type A coronary prone behavior pattern in postmyocardial infarction patients (hypotheses 1 and 8) was analyzed using the Kendall Tau statistic. Results were found to be statistically non-significant at .420 and .78, respectively (Table 3)

The average Bortner Scale scores classified with obesity (pounds overweight) and normal weight are reflected in Table 4. Of the eight overweight subjects, six (75%) scored high on the Bortner Scale, and two (25%) subjects scored low. The four (80%) normal weight subjects scored

Table 3

Summary: Statistical Tests and Significance Levels
for Each Hypothesis

Risk Factors ^a with Average Bortner Scale Scores	Statistical Test	Significance Level
	<u>Kendall Tau</u>	
(1) Age	.0427	.420 <u>p</u> = <.05
(8) Obesity	-.41	0.78 <u>p</u> = <.05
	<u>Phi Coefficient</u>	<u>Fisher Exact Test</u>
(2) Sex	.562	.110 <u>p</u> = <.05
(3) Familial history of premature coronary heart disease	.192	.50 <u>p</u> = <.05
(4) Hypertension	.03043	.797 <u>p</u> = <.05
(5) Elevated cholesterol	.567	.231 <u>p</u> = <.05
(6) Elevated triglycerides	.333	.892 <u>p</u> = <.05
(7) Glucose intolerance (Diabetes Mellitus)	.158	.769 <u>p</u> = <.05
(9) Lack of exercise/ sedentary living	.000	.51 <u>p</u> = <.05

^aNote. Number in parentheses indicates related hypothesis.

high on the Bortner Scale and one normal weight subject scored low (Table 4).

Table 4

Cross Classification: Average Bortner Scale Scores with Obesity and Normal Weight

Average Bortner Scale Scores	Total Subjects	Obesity		Normal	
		Number	Percent	Number	Percent
High (5-9)	10	6	75	4	80
Low (0-4)	3	2	25	1	20
Total	13	8	100	5	100

Note. Obesity not significant at $p \leq .05$. More obese subjects scored lower on the Bortner Scale; an inverse correlation was found.

A breakdown of the actual number of pounds overweight and the Bortner Scale scores showed that the more obese the subjects, the lower they rated themselves on the scale (even though many were still in the designated High (5-9) range. These data are also reflected in the Comprehensive Data Table (Table X). Thus, a moderate inverse correlation was found when obesity was compared to the average Bortner Scale scores, though not significant at $p \leq .05$.

Although age cross-classified with the average Bortner Scale scores was found not significant at $p \leq .05$, it is descriptively significant that all five subjects in the

31-40 years age group scored high on the Bortner Scale.

These data are shown in Table 5.

Table 5

Cross Classification: Age with Average Bortner
Scale Scores

Average Bortner Scale Scores		31-40 Years		51-60 Years		61-70 Years		Total	
		Num- ber	Per- cent	Num- ber	Per- cent	Num- ber	Per- cent	Num- ber	Per- cent
Low (0-4)	3	0	0.0	1	7.7	0	0.0	1	7.7
	4	0	0.0	0	0.0	2	15.4	2	15.4

	5	3	23.1	1	7.7	1	7.7	5	38.5
High (5-9)	6	2	15.4	0	0.0	1	7.7	3	23.1
	7	0	0.0	1	7.7	0	0.0	1	7.7
	8	0	0.0	0	0.0	1	7.7	1	7.7

Totals		3		3		5		13	100.0

Note. $p \geq .05$, $n = 13$. No significance seen for age and score.

The association between the Type A coronary-prone behavior pattern and the remaining selected standard risk factors, sex, familial history of premature coronary heart disease, hypertension, elevated cholesterol and/or triglycerides, cigarette smoking, glucose intolerance (Diabetes Mellitus), and lack of exercise (hypotheses 2, 3, 4, 5, 6,

7, and 9), was analyzed using the Phi coefficient. None of these factors were found to be significant at $p \leq .05$, as indicated in Table 6.

Table 6
Statistical Significance of Nominal Data

Risk Factor ^a	Fisher Exact Probability	Phi Coefficient	Significance
(2) Sex	$p = .110$	.567	none
(3) Familial history of premature heart disease	$p = .50$	.192	none
(4) History of hypertension	$p = .797$	.03043	none
(5) History of elevated cholesterol	$p = .231$	.527	none
(5) History of elevated triglycerides	$p = .892$	.333	none
(6) Cigarette smoking	$p = .764$	.000	none
(7) Glucose intolerance (Diabetes Mellitus)	$p = .769$	.158	none
(9) Exercise	$p = .51$	.000	none

Note. These risk factors are considered nominal data.

^aNumber in parentheses indicates related hypothesis.

Some inference may be drawn from this small sample. The single most descriptively significant factor in the study was the occurrence of Type A behavior in 10 (81.8%) of the subjects, all of whom scored in the high range on the scale. Three subjects ranked low on the scale and of these "low" scores, two scored 4 (Tables 2 and 3, Figure 2).

Table 7 describes the average Bortner Scale scores in regard to the number of myocardial infarctions (MIs). A high degree of relationship was seen between high Bortner Scores and MIs. For 11 subjects, the current MI was their first; 9 of these (81.8%) scored high on the Bortner Scale and 2 (18.2%) scored low. Of the subjects in which the current MI was their second, one scored high on the Bortner Scale and one scored low.

Table 7

Cross Classification: Average Bortner Scale Scores with
Number of Myocardial Infarctions

Average Bortner Scale Scores	1 MI		2 MIs		Total Subjects
	Number	Percent	Number	Percent	
High (5-9)	9	81.8	1	50.0	10
Low (0-4)	2	18.2	1	50.0	3
Totals	11	100.0	2	100.0	13

The most frequently occurring risks ranked according to their occurrence were the non-modifiable risks of age and sex. Of the modifiable risk factors, obesity, cigarette smoking, and lack of exercise or sedentary living were the most frequently occurring variables, even though they were not statistically significant. The relationship of the average Bortner Scale scores to the nine selected standard cardiac risk factors is presented as follows:

Age

More subjects in the 31-40 years age range demonstrated the highest Type A mean ratings than from the other age ranges. In fact, five subjects (100%) in the 31-40 years age range scored high on the Bortner Scale (refer to Table 5).

Sex

In regard to sex, the study sample was too small to make any conclusions. There were 3 females and 10 males in the study. Of the 10 males, 90% had high Bortner scores. The three females comprised 33% of the study sample. Of these, one had a high Bortner Score and two (67%) had low scores (Table Y).

Familial History of Premature Heart Disease

A positive family history of premature heart disease was found in six subjects (50%) of the sample (no data were available on one subject). Of these six subjects, four scored high on the Bortner Scale and two scored low, as shown in Table 8.

Table 8

Cross Classification: Average Bortner Scale Scores with Family History of Premature Heart Disease

Average Bortner Scale Scores	Yes		No	
	Number	Percent	Number	Percent
High (5-9)	4	66.7	5	83.3
Low (0-4)	2	33.3	1	16.7
Totals ^a	6	100.0	6	100.0

^aN = 12, no data from one respondent.

Hypertension

Four subjects were found to have a past or known history of hypertension. Of these, three scored high on the Bortner Scale and one scored low (Table Z). Three subjects were found currently to have hypertension and of these, two scored high on the Bortner Scale and one scored low (Table AA). Of the sample 11 subjects (84.6%) had a positive family history of hypertension (Table R).

Elevated Cholesterol and/or Triglycerides

Only one subject had a past history of elevated cholesterol and he scored low on the Bortner Scale (Table BB). Three subjects (66.7%) had a past history of elevated triglycerides. Of these, two scored high on the Bortner Scale and one scored low (Table CC).

Cigarette Smoking

Two subjects were found currently to have elevated cholesterol. Of these, one scored high on the Bortner Scale and one scored low. No data were available on one subject (Table DD). Five subjects were found currently to have elevated triglycerides. Of these, three subjects scored high on the Bortner Scale and two scored low. Data were not available on one subject (Table EE).

Eight of the subjects (66.7%) smoked cigarettes an average of 25.4 years (Tables T, U, V, FF, and GG). Table 9 shows that six of these subjects scored high on the Bortner Scale scores and two scored low.

Glucose Intolerance/Diabetes Mellitus

Only one subject was diabetic. This subject scored high on the Bortner Scale (Table HH).

Table 9

Cross Classification: Average Bortner Scale Scores
with Cigarette Smoking

Average Bortner Scale Scores	Smoker		Nonsmoker	
	Number	Percent	Number	Percent
High (5-9)	6	75.0	3	75.0
Low (0-4)	2	25.0	1	25.0
Totals ^a	8	100.0	4	100.0

^a $\underline{N}$ = 12, no data from one respondent.

Obesity

Eight subjects (62%) of the sample were obese. They ranged from 7 to 71 pounds overweight. Of these, six subjects (75%) had a high Bortner Scale score and two low. The median weight was 39.5% overweight (Tables 4 and I).

Lack of Exercise/Sedentary Living

Twelve subjects reported information regarding exercise. Four subjects (33.3%) do exercise; eight (66.7%) of the subjects do not exercise, as indicated in Table 10 (Table W).

The relationship of obesity to cigarette smoking and Bortner Scale scores is shown in Table 11. Of the three obese individuals who smoked, two smoked and received high

Table 10

Cross Classification: Average Bortner Scale Scores
with Exercise

Average Bortner Scale Scores	<u>Exercise</u>		<u>No Exercise</u>	
	Number	Percent	Number	Percent
High (5-9)	3	75.0	6	75.0
Low (0-4)	1	25.0	2	25.0
Totals ^a	4	100.0	8	100.0

^a $\underline{N}$ = 12, no data from one respondent.

Bortner Scale scores and one smoked and received low
Bortner Scale scores.

Table 11

Cross Classification: Average Bortner Scale Scores with
Cigarette Smoking and Obesity

Average Bortner Scale Scores	<u>Smoker</u>	<u>Obesity</u>
	Number	Number
High (5-9)	4	2
Low (0-4)	1	1
Totals ^a	5	3

^a $\underline{N}$ = 8.

Other interesting relationships found in the scrutiny of data were that of the 12 white subjects, 75% scored high on the scale. The one black subject also scored high on the Bortner Scale (Table II).

The majority of subjects who scored high on the Bortner Scale were married, seven subjects (87.5%). Three subjects who scored high on the Bortner Scale were divorced (Table JJ).

Table 12 reflects the number of risk factors per high and low Bortner Scale scores. The largest number of risks were associated with high Bortner Scale scores.

Table 12

Numbers of Risk Factors Occurring with Bortner
High and Low Scores

Average Bortner Scale Scores	Number of Risk Factors					
	1	2	3	4	5	6
High (5-9)	1	3	3	2	1	0
Low (0-4)	0	1	1	0	0	1

Summary of Findings

The hypotheses that no association would be found between Type A coronary-prone behavior pattern and the following selected major cardiac risk factors--(1) age,

(2) sex, (3) familial history of premature coronary heart disease, (4) hypertension, (5) elevated serum cholesterol and/or triglycerides, (6) cigarette smoking, (7) glucose intolerance (Diabetes Mellitus), (8) obesity, and (9) lack of exercise/sedentary living, in the postmyocardial infarction subjects studied--were not supported.

CHAPTER 5

SUMMARY OF THE STUDY

Chapter 5 presents a review of the study. The discussion of findings summarizes significant research related to the investigator's findings. A recommendation was made that further research related to the topic of this study be undertaken.

Summary

A review of the major research related to Type A coronary-prone behavior was completed. The selected standard cardiac risk factors were reviewed in terms of their significance as risk factors. The literature revealed that Type A coronary-prone behavior is significant as a cardiac risk factor and has been found predictive independently of other factors for myocardial infarction and particularly for recurrence of myocardial infarction (Jenkins, 1975, p. 17). It is further significant that the major cardiac risk factors "probably explain no more than 25% of the variance in the incidence of coronary disease" (Jenkins, 1975, p. 33).

The first two purposes of this study were to determine the extent that selected standard cardiac risk factors and

Type A coronary-prone behavior was present in postmyocardial infarction patients. The third purpose was to determine the association between selected standard cardiac risk factors and Type A behavior pattern in postmyocardial infarction patients. The fourth purpose was to determine which selected standard cardiac risk factor or set of factors best predicted Type A coronary-prone behavior pattern. The hypotheses of the study were that no association would be found between Type A coronary-prone behavior and the selected major cardiac risk factors: (1) age, (2) sex, (3) familial history of premature coronary heart disease, (4) hypertension, (5) elevated serum cholesterol and/or triglycerides, (6) cigarette smoking, (7) glucose intolerance (Diabetes Mellitus), (8) obesity, and (9) lack of exercise/sedentary living in the postmyocardial infarction subjects.

The design of this study was nonexperimental and was descriptive and correlational in nature. The sample consisted of 13 postmyocardial infarction subjects studied at least eight days postmyocardial infarction. The sample was obtained by convenience selection from three private general hospitals in the southwestern United States in a city of approximately 844,000 people. Eleven cardiologists and primary internists participated in the study by completing a part of the Cardiac Risk Factor Assessment Tool

(CRAT). The CRAT, consisting of three parts--the Investigator's Check List, Part A, and Part B, was used to determine the standard cardiac risk factors. The Investigator's Check List was used as a research aide to maintain uniformity in data collection. Part A of the CRAT was completed by participating cardiologists or primary internists. Part B of the CRAT was completed by the subjects. The Bortner Self-Rating Tool was used to measure Type A coronary prone behavior.

The hypotheses that no association would be found between Type A coronary-prone behavior pattern and the following selected major cardiac risk factors--(1) age, (2) sex, (3) familial history of premature coronary heart disease, (4) hypertension, (5) elevated serum cholesterol and/or triglycerides, (6) cigarette smoking, (7) glucose intolerance (Diabetes Mellitus), (8) obesity, and (9) lack of exercise/sedentary living in the postmyocardial infarction subjects studied--were not supported. The Kendal Tau and Phi Coefficient statistics were used to test these hypotheses. Determination of the factor or set of factors that best predicted Type A behavior could not be determined secondary to the small sample and because the assumptions of a normal distribution were violated.

Discussion of Findings

No statistically significant relationship was found between Type A coronary-prone behavior and the standard cardiac risk factors. Because the sample size was so small the findings of this study were effected. A factor or set of risk factors that best predicted Type A behavior could not be obtained. The stepwise regression technique was not appropriate because the distribution of risk factors was skewed.

The outstanding factor in the study was the incidence of Type A coronary-prone behavior. Even though it could not be found statistically significant, it was definitely descriptively significant. Of the 13 subjects, 10 (76.9%) scored high on the Bortner Scale. Three subjects scored low: However, two of these scores were "4s". The mean for the Bortner Scale was 5.3, the median 5.2, and the mode 5.0 (average Bortner Scale scores were: High, 5 to 9, and Low, 0 to 4). The standard deviation was 1.31, the standard error was .365. The 95% confidence interval was 4.513 to 6.103. The Bortner scores were found to be independent of all other risk factors. The finding supported the literature as Friedman and Rosenman in the Western Collaborative Study (1974b) found Type A behavior to be independently significant also.

Although this study was not prospective, the data have potential usefulness as it has been shown that Type A behavior is significant for recurrence of myocardial infarction (Jenkins, 1975, p. 17). Type A behavior has also been associated with a poor prognosis postmyocardial infarction (Adsett et al., 1974, pp. 187-191).

The data obtained in this study must be viewed cautiously because of the small sample size and also because of its retrospective nature. The limitations of the study must also be considered. Physiological factors may have influenced behavior ratings obtained in this study. It is also possible that medications may have altered responses of the subjects studied. Some subjects may have responded inaccurately because of a lack of knowledge of their health histories or problems. Further, it is possible that some subjects may have been incapable of accurate self-ratings of their behavior. The extent that hospitalization may affect behavior is undetermined.

Conclusions and Implications

Because of the small sample size, no conclusions can be drawn from the findings of this study. Therefore, no implications can be made from this study.

Recommendations

As a result of this study, the following recommendation is presented. Due to the inordinately small sample size obtained in this study, replication of the study with a larger sample should be done.

APPENDIX A

CORRESPONDENCE

October 18, 1975

Dr. C. David Jenkins
Dept. of Epidemiology
Boston University

Dear Dr. Jenkins:

I am a graduate nursing student at Texas Woman's University, Dallas, Texas. I am presently working on my thesis which involves correlating the coronary prone behavior pattern with the other cardiac risk factors. I have done a great deal of literature research in the area of the coronary prone behavior personality and have not found a complete form of the Jenkins Activity Survey. The literature I have searched so far clearly indicates your form to be the most appropriate. However, I have not found a complete copy of the tool, instructions for use or scoring methods.

I would like to use the Jenkins Activity survey in my research. May I have your permission to use the form? Also, could you direct me to appropriate sources to obtain a copy of the tool? I will gladly forward the cost for a copy of the tool or any additional information you might suggest. I would greatly appreciate your assistance in this matter.

Departmental Chairman, Faculty Advisor
Beth C. Vaughn-Wrobel
School of Nursing
Texas Woman's University
Dallas, Texas

Sincerely yours,

Deborah Nickell
Deborah Nickell



Boston University Medical Center

School of Medicine
720 Harrison Avenue
Boston, Massachusetts 02118

Division of Psychiatry
Department of Behavioral Epidemiology

October 30, 1975

Ms. Beborah Nickell
4101 W. 45th Street
Apt. 1304
Amarillo, Texas 79109

Dear Ms. Nickell:

In response to your request for the use of the Jenkins Activity Survey (JAS) as an adjunct to your protocol for studying cardiac risk factors, we would like to clarify several pertinent issues. First, JAS scores have not been found to correlate with standard risk factors in several large groups already studied. In one such group, over 3,000 men were studied prospectively for eight years with similar negative findings. Thus, the Activity scores appear to be independent of the standard risk factors measured so far.

Secondly, the JAS in its present form still misclassifies too many individuals to allow it to be used in the usual clinical setting for evaluating coronary risk among individuals or small groups. The Activity Survey is presently undergoing development and is in the process of being substantially changed. These new scales will not be generally available until a thorough evaluation and cross-validation have been completed.

Thirdly, a different short test was developed by Dr. Ray W. Bortner and published in the Journal of Chronic Diseases, Volume 22, pages 87-91, 1969. The article is entitled "A short rating scale as a potential measure of Pattern A behavior". This is a semantic differential type instrument based upon fourteen items. It agrees with the standard behavior type interview to the same degree as does the JAS. It seems most likely from your letter that this short rating scale might better fit your needs.

We do thank you for your interest in our work and wish you the best of luck in your project.

Sincerely,

C. David Jenkins, Ph.D.
Director
Department of Behavioral Epidemiology

March 1, 1976

Dr. Rayman W. Bortner
College of Human Development
Pennsylvania State University

Dear Dr. Bortner:

Presently, I am working towards my M.S. in Nursing at Texas Woman's University, Dallas, Texas. My thesis involves correlating the coronary prone behavior pattern with the other cardiac risk factors. I am having great difficulty in finding an appropriate tool to measure the coronary prone behavior pattern. I wrote to Dr. David Jenkins in October asking permission to use the Jenkins Activity Survey. Dr. Jenkins answered my request saying that the scale was being revised at this time. He suggested using the Bortner scale.

I have utilized the computer information retrieval services at two medical schools searching for the latest and best means to measure the coronary prone behavior pattern. It appears that the Bortner scale is the most appropriate. I am very concerned that my research be worthwhile and that my data is current. Do you recommend the Bortner scale as current? If so, may I have your permission to utilize the scale in my research? Also, how do I go about getting a copy of the tool and instructions for use? The last article I can find dealing with the scale is in the Journal of Chronic Diseases, volume 22, pp. 87-91, 1969. Do you recommend a later source?

I would greatly appreciate your assistance in this matter. I will also be happy to forward payment for a copy of the tool, instructions for its' use, or postage.

Departmental Chairman, Faculty Advisor
Beth C. Vaughn-Wroebe
Texas Woman's University
Dallas, Texas

Sincerely yours,

Deborah Nickell

Deborah Nickell R.N.

P.O. Box 233
Geles Ferry, Ct. 06335
March 18, 1976

Ms. Deborah Nickell
4101 West 45th Street #2304
Amarillo, Texas 79109

Dear Ms. Nickell,

I am enclosing a copy of the Pattern A Rating Scale currently in use. In addition, Items 31-39 are also under additional study.

I am on sabbatical and have not kept track of the users of the Rating Scale since it is only recently that I have turned my attention to it again. Currently, I have been using the rating scale in conjunction with Sterns personality and environment measures (People in Context, Syracuse University Press, 1970). It appears that both personality and environment tend to stimulate and re-inforce Pattern A Behavior as measured by the Rating Scale.

The Rating Scale is being used on a heterogeneous sample ranging in age from 14 to 61 and containing both males and females. There are 156 respondents. In that sample, as you can see, the rating scale has been converted from a set of two opposing characteristics which the respondent uses to gauge his relative position between to the two extremes to a simple 10 point (0-9) rating scale. Rather than weighting these scores, the simple sum has been used. For this sample, this procedure yields a Mean of 58.21, S.D. 10.91 with a range from 27 to 90. Higher scores represent more Pattern A Behavior.

While the original scoring and technique may be more accurate, the results of the present study suggest that the simpler approach may be as effective and is somewhat easier for respondents to complete.

I would be interested in hearing about the results of your study. Good luck?

Sincerely,

Rayman W. Bortner
Rayman W. Bortner

*Sorry that I don't have any response
here with me. Lutz*

Background Information and Self Ratings - 2

These self ratings are designed to look at similarities and differences between parents and children in terms of hopes and fears, some aspects of life style and values. Almost all of these ratings are to be made on a zero to nine point scale. The extremes of each scale are defined by the numbers and descriptions in parentheses. Each of us belongs somewhere in between usually. For example, most of us are neither the most competitive nor the least competitive people we know. What we would like you to do is pick a number that would best describe the way you think of yourself between the two points. Put your rating in the rating column. Don't spend a great deal of time; we want your first impressions. Skip any item that doesn't seem to apply to you.

Rating

1. My personality is, mostly, (0 - very different; 9 - quite like) my mother's. _____
2. My personality, to me, is (0 - very different; 9 - quite like) my father's. _____
3. The way I live, stress and strain is (0 - easy to avoid; 9 - almost impossible to avoid). _____
4. Changing my way of doing things might (0 - improve my chances; 9 - interfere with my chances) of getting ahead. _____
5. I'm usually (0 - never late; 9 - casual) about keeping appointments on time. _____
6. For me, 'on time' means (0-5 or 10 minutes one way or the other; 9 - a couple of minutes before or 'on the dot' at the latest). _____
7. Usually I (0 - can wait patiently; 9 - get impatient if kept waiting). _____
8. Most of the deadlines I have to meet (0 - I set for myself; 9 - are 'built into' my job so that they can't be chanced easily). _____
9. Forced to make a choice, it is more important a good job I've done (0 - be recognized by other people; 9 - has satisfied my own standards). _____
10. In comparison with other people I'm (0 - not competitive; 9 - very competitive). _____
11. When I'm interested in something, I work (0 - at "all out" pace; 9 - fairly casually). _____
12. I'd describe myself as (0 - forceful in speech; 9 - a slow, deliberate talker). _____
13. My close friends would say that I was (0 - hard driving and ambitious; 9 - easy going). _____

Father
 A
 Father
 Scale
 (all
 part
 of)

Self Ratings (Cont'd)

Rating

14. In general, how satisfied are you with the progress you've made in your life in the last 5 years? (0 - Very dissatisfied; 9 - very satisfied). _____
15. In the economic area, specifically, how satisfied are you with the increase in the amount of money you've gotten over the last five years? (0 - very dissatisfied, 5 - very satisfied). _____
16. Would you say that the economic gains made by (other) ^{white} ~~white~~ collar workers have been (0 - very much worse; 9 - very much better) than your own? _____
17. What about the economic gains of other blue collar workers? (0 - very much worse; 9 - very much better) than your own? _____
18. In comparison with your own economic gains, are the gains of other blacks (0 - very much worse; 9 - very much better) than your own? _____
19. When you compare your economic gains with those of others in your own age group, are you (0 - very satisfied, 9 - very dissatisfied)? _____
20. How about comparing your gains with those of (other) whites? (0 - very satisfied; 9 - very dissatisfied)? _____
21. How about when you compare your gains in the last five years with those of the other (older or younger) generation? Are you (0 - very satisfied; 9 - very dissatisfied). _____
22. How do your economic gains compare with (other) professionals (0 - very much worse; 9 - very much better). _____
23. How do your economic gains compare with (other) unskilled white workers? (0 - very much worse; 9 - very much better). _____
24. It's hardly fair to bring a child into the world with the way things look for the future. (0 - Strongly disagree; 9 - strongly agree). _____
25. In spite of what some people say, the condition of the average man is getting worse. (0 - strongly disagree; strongly agree). _____
26. These days a person doesn't really know whom he can count on. (0 - strongly disagree; 9 - strongly agree). _____
27. Most people in public office are not really interested in the problems of the average man. (0 - strongly disagree; 9 - strongly agree). _____
28. Most people really don't care what happens to the next fellow. (0 - strongly disagree; 9 - strongly agree). _____
29. To make money there are no right and wrong ways any more, only easy and hard ways. (0 - Strongly disagree; 9 - strongly agree). _____

Self Ratings (Cont'd)

Rating

30. Nowadays a person has to live pretty much for today and let tomorrow take care of itself. (0 - strongly disagree; 9 - strongly agree). _____
31. I (0 - usually; 9 - almost never) have time enough to complete a job to my satisfaction. _____
32. Most of the things I do from day to day, I do with the feeling that it is because I (0 - want to do them; 9 - ought to do them.) _____
33. In comparison with others who do my kind of work, I judge myself (0 - very much less successful; 9 - very much more successful). _____
34. In my work the standards for judging success are (0 - very vague; 9 - clear cut). _____
35. In my work I have to consider what (0 - only one or two people expect of me; 9 - what a great many people expect of me). _____
36. Considering the number of things I have to do (0 - I have time enough to do them to my satisfaction; 9 - I almost never have time enough to do them to my satisfaction). _____
37. Farmers have 'sequential deadlines. There are all kinds of things a farmer has to do but it usually doesn't matter whether he finishes a particular job at 7:32 or 7:35. By contrast, chemists, commuters and assembly line workers have 'segmental' deadlines; if something isn't done at precisely the right time, the rest of the day or the whole process might be fouled up. Most of my deadlines are (0 - sequential; 9 - segmental). _____
38. In trying to reach a deadline I (0 - often feel plagued by interruptions and distractions; 9 - can usually 'brush aside' interruptions and distractions). _____
39. Most of the time I feel that (0 there isn't very much; 9 - there is a great deal) I can do for myself to make my life easier and happier. _____

*Additional
of time
N/A
Slightly*

Fill in the blanks and then do the next three ratings.

The best possible life I could imagine would be _____

The worst possible life I could imagine would be _____

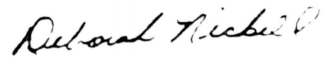
July 1, 1976

Dr. Rayman W. Bortner
Geles Ferry
Connecticut, 06335

Dear Dr. Bortner:

Thank you for clarifying for me that items 31-39 of the Bortner scale have been validated. I will appreciate any information you can send me in regard to scoring of the scale. I am also particularly interested in which numbers on the 0 to 9 point self-rating scale are most indicative of Type A behavior. That is, what number or range of numbers on this scale would indicate that a person is not Type A?

Sincerely yours,

A handwritten signature in cursive script, appearing to read "Deborah Nickell".

Deborah Nickell R.N.

P. O. Box 233
Gales Ferry, Ct. 06335
July 12, 1976

Ms. Deborah Nickell
4101 West 45th Street #1304
Amarillo, Texas 79109

Dear Miss Nickell,

I have just returned from Penn State where I was taking back some materials in preparation for the end of my Sabbatical. Inadvertently I also included most of the materials on Pattern A Behavior.

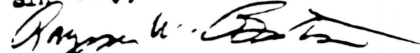
All but two of the items proved to be indicative of Pattern A when scored in a positive direction. That is, the higher the rating, the more Pattern A Behavior. These two items, which require reverse scoring, can be identified by the difference in sign from the 1969 article.

The simplest way to score the scale is to reverse the score on those two items (i.e., subtract the rating that the subject gave from 9) and then to add the ratings over all of the items. Then the higher the total score, the more intense the Pattern A Behavior. This method is 'quick and dirty' it does not permit a translation into the A1, A2, B3, B4 classifications used by Rosenman and Friedman and does not make use of the differential weights for the items.

The other method which overcomes these advantages is to use the regression weights as it was done in the J. Chronic Diseases article. First, ~~if~~ multiply the rating by 2.666. This converts the 9 point scale into the equivalent of the units used in the original study. Multiply the resulting scores by the regression weights given in the original article. Since these weights have/ signs it is not necessary to do any reverse scoring in this version. The algebraic sum of the item scores is then added to or subtracted from the constant given in the table. This should result in scores which are comparable to Rosenman and Friedman's interview version of the assessment of Pattern A Behavior.

I am sorry for the delay in responding but your note did not reach me prior to my leaving for Pennsylvania.

Sincerely,


Rayman W. Bortner

APPENDIX B

QUESTIONNAIRE PACKET

CONSENT FORM

TEXAS WOMAN'S UNIVERSITY
COLLEGE OF NURSING

Consent to Act as a Subject for Research and Investigation:

1. I hereby authorize Deborah Jean Nickell, R.N., B.S.N. to perform the following investigation:

The American Heart Association has identified numerous factors which may or may not contribute to heart problems in certain individuals. These risk factors include such items as high blood pressure, obesity, smoking, and others. The purpose of this study will be to see if you have any of these factors present and if so how they may or may not be of consequence.

The study consists only of your filling in a questionnaire. You will in no way now or in the future be identified by name. Please do not write your name on the questionnaire. Completed questionnaires will be seen by the investigator only and will be kept in a locked file.

Your participation is strictly voluntary. You are free to withdraw from this study at any time. Your participation or nonparticipation will in no way affect your current or future treatment. No known physiological risks other than possible anxiety exists from participating in this study.

2. The procedure or investigation listed in Paragraph 1 has been explained to me by Deborah Jean Nickell.
3. (a) I understand that the procedures or investigations described in Paragraph 1 involve the following possible risks or discomforts:

3. (a) 1. It is possible that you may feel some anxiety when completing the questionnaires. However, the following steps have been taken to protect you from feeling overly anxious: (a) You can withdraw from the study at any time. (b) Your anonymity will be maintained; your name will in no way be identified with this study. Results of this study will be reported as group rather than individual results. (c) The investigator will be happy to answer any questions you may have about the study or the questionnaire. (d) Although your doctor has given his permission for you to participate in this study, you will not be identified in any way with your questionnaire and your present or future treatment will in no way be affected. All results of this study will be reported as group data rather than individual data.
3. (b) I understand that the procedures and investigations described in Paragraph 1 have the following potential benefits to myself and/or others:
- Potential benefits of this study will be to increase knowledge about cardiac risk factors, and determining their significance in contributing to heart disease. This information may contribute to better prediction of heart disease.
- (c) I understand that--No medical service or compensation is provided to subjects by the university as a result of injury from participation in research.
4. An offer to answer all of my questions regarding the study has been made. If alternative procedures are more advantageous to me, they have been explained. I understand that I may terminate my participation in the study at any time.

Subject's Signature

Date

INVESTIGATOR'S CHECK LIST

Participant Criteria

- _____ 1) Permission from hospital
- _____ 2) Patient selection. Must meet all criteria.
 - A) Diagnosis: Myocardial infarction
 - B) Minimum age: 18 years
 - C) Minimum requirements: 8 days postmyocardial infarction
 - D) On regular unit
 - E) No known psychological or mental disturbances
 - F) Patients will be studied if:
 - a) Fully alert and cooperative
 - b) ~~Ex~~periencing no pain or distress
 - c) Not being monitored
 - d) Not requiring oxygen, an IV or any other prophylactic or supportive device
 - e) Is ambulatory
- _____ 3) Cardiologist has completed Part A of the Cardiac Risk Factor Assessment Tool

Research Criteria

- _____ 1) Participant Criteria Check List complete
- _____ 2) Purpose of research explained
- _____ 3) Anonymity explained
- _____ 4) Written permission form obtained
- _____ 5) Provide pen, questionnaire, and comfortable setting
- _____ 6) Ask participant to read the provided instructions; answer any questions

Instructions to Participating Cardiologists:

Please complete the following questions and place at the front of the participant's chart. To be studied, participants must meet all the following criteria:

- A. Diagnosis: Myocardial infarction
- B. Minimum age: 18 years
- C. Minimum requirement: 8 days postmyocardial infarction
- D. Be located on a regular hospital unit (no participants will be studied while in a critical care area)
- E. No known psychological or mental disturbances
- F. Patients will be studied if:
 - a. Fully alert and cooperative
 - b. Experiencing no pain or distress
 - c. Not being monitored
 - d. Not requiring oxygen, an IV, or any other prophylactic or supportive device
 - e. Is ambulatory

Once you have completed the questionnaire and placed it at the front of the chart, I will contact the patient to complete Part B of the study which consists of two self-rating questionnaires.

Thank you for your participation in this research effort.

Deborah Nickell R.N.

Deborah Nickell, R.N.
Texas Woman's University
Graduate Nursing Student

PART A: CRAT

RESEARCH INFORMATION

TO BE COMPLETED BY THE CARDIOLOGIST:

- | | <u>Yes</u> | <u>No</u> |
|---|------------|-----------|
| 1. Does this patient have a history of | | |
| A. Hypertension | _____ | _____ |
| B. Elevated cholesterol | _____ | _____ |
| C. Elevated triglycerides | _____ | _____ |
| 2. Presently, does the patient have: | | |
| A. Hypertension | _____ | _____ |
| B. Elevated cholesterol | _____ | _____ |
| C. Elevated triglycerides | _____ | _____ |
| 3. How many myocardial infarctions, including
the present one, has this patient been
diagnosed as having had? _____ | | |

Dr. _____
 (Cardiologist, please sign to designate this
 questionnaire is valid. Your anonymity will
 be maintained by the investigator.)

Date _____

Dr. _____
 (Primary Internist if not Cardiologist.)

Date _____

The following questionnaire has been developed to study factors which may or may not be related to heart disease. The questionnaire consists of two parts.

The researcher will in no way use your name now or in the future. No time limit will be imposed.

Please be absolutely truthful in your answers. Do not discuss your answers or the questionnaire with anyone while you are completing it. Read all instructions carefully.

The researcher will return and collect your questionnaire when you have completed it.

If you have any problem completing your questionnaire, push your call button and ask that Miss Nickell be sent to your room.

I greatly appreciate your interest and contribution to this research effort.

Deborah Nickell R.N.
Deborah Nickell, R.N.
Graduate Nursing Student
Texas Woman's University

B: CRAT

PART ONE: Instructions: Complete the following to the best of your knowledge. Place an X in the appropriate blank space.

Age: _____ Sex: Male _____ Female _____

Height: _____ ft. _____ in. Weight: _____ lbs.

Race: White _____ Mexican-American _____
 Black _____ Other (specify) _____

Occupation: _____

Marital Status: Single _____ Divorced _____ Widowed _____
 Married _____ Separated _____

Yes No

1. Have you been diagnosed by your doctor as having had a heart attack? _____
2. In your family, is there a history of premature heart disease? (That is, any blood relative who has had a heart attack prior to age 50?) _____
3. In your family is there a history among any of your blood relatives of diabetes? _____
4. In your family is there a history among any of your blood relatives of high blood pressure and/or strokes? _____
5. Has your doctor ever told you that you have high blood pressure? _____
6. Has your doctor ever told you that your cholesterol level is elevated? _____
 If so, are you being treated for this? _____
7. Has your doctor ever told you that you have diabetes? _____
8. Do you smoke a pipe or cigars? _____

Yes No9. Do you smoke cigarettes? If so, how many packs per day? If so, how long? mos. yrs.If you do not smoke cigarettes,
have you ever smoked? How many packs per day did you smoke
before you quit smoking? packs per day for yearsHow much time has it been since you quit
smoking? mos. yrs.10. Do you exercise? (That is, do you regularly
follow any planned regimen of exercise for
physical fitness or relaxation other than
your work?)

PART TWO: Instructions: Please complete the following rating scale to the best of your ability. Pick the number from 0 to 9 that would best describe the way you think of yourself. Put that number in the rating column. Don't spend a great deal of time; we want your first impression. Skip any item that doesn't seem to apply to you.

- | | <u>Rating</u> |
|---|---------------|
| 1. The way I live, stress and strain is (0--easy to avoid; 9--almost impossible to avoid). | _____ |
| 2. Changing my way of doing things might (0--improve my chances; 9--interfere with my chances) of getting ahead. | _____ |
| 3. I'm usually (0--never late; 9--casual) about keeping appointments on time. | _____ |
| 4. For me, "on time" means (0--5 or 10 minutes one way or the other; 9--a couple of minutes before or "on the dot" at the latest). | _____ |
| 5. Usually I (0--can wait patiently; 9--get impatient if kept waiting). | _____ |
| 6. Most of the deadlines I have to meet (0--I set for myself; 9--are "built into" my job so that they can't be changed easily). | _____ |
| 7. Forced to make a choice, it is more important that a good job I've done (0--is to be recognized by other people; 9--has satisfied my own standards). | _____ |
| 8. In comparison with other people I'm (0--not competitive; 9--very competitive). | _____ |
| 9. When I'm interested in something, I work (0--at an "all out" pace; 9--fairly casually). | _____ |
| 10. I'd describe myself as (0--forceful in speech; 9--a low, deliberate talker). | _____ |
| 11. My close friends would say that I was (0--hard driving and ambitious; 9--easy going). | _____ |
| 12. I (0--usually; 9--almost never) have time enough to complete a job to my satisfaction. | _____ |

Rating

13. Most of the things I do from day to day, I do with the feeling that it is because I (0--want to do them; 9--ought to do them). _____
14. In comparison with others who do my kind of work, I judge myself (0--very much less successful; 9--very much more successful). _____
15. In my work the standards for judging success are (0--very vague; 9--clear cut). _____
16. In my work I have to consider what (0--only one or two people expect of me; 9--what a great many people expect of me). _____
17. Considering the number of things I have to do (0--I have time enough to do them to my satisfaction; 9--I almost never have time enough to do them to my satisfaction). _____
18. Farmers have 'sequential' deadlines. There are all kinds of things a farmer has to do but it usually doesn't matter whether he finishes a particular job at 7:32 or 7:35. By contrast, chemists, commuters, and assembly line workers have 'segmental' deadlines; if something isn't done at precisely the right time, the rest of the day or the whole process might be fouled up. Most of my deadlines are (0--sequential; 9--segmental). _____
19. In trying to reach a deadline I (0--often feel plagued by interruptions and distractions; 9--can usually 'brush aside' interruptions and distractions). _____
20. Most of the time I feel that (0--there isn't very much; 9--there is a great deal) I can do for myself to make my life easier and happier. _____

APPENDIX C

LIPOPROTEIN DATA

THE INTERPRETATION OF LIPOPROTEIN DATA

NORMAL RANGES

Age, yr.	Cholesterol, mean and 90 percent limits	Triglyceride mean and 90 percent limits
0-19	175 (120-230)	65 (10-140)
20-29	180 (120-240)	70 (10-140)
30-39	205 (140-270)	75 (10-150)
40-49	225 (150-310)	85 (10-160)
50-59	245 (160-330)	95 (10-190)

Lipoprotein Summary

Type I Plasma: clear with creamCholesterol N, Triglycerides $\uparrow$ LP Electrophoresis: chylomicrons $\uparrow$, β N, pre β N
(Fat sensitive)Type II A Plasma: clearCholesterol $\uparrow$, Triglycerides NLP Electrophoresis: no chylo, β $\uparrow$, pre β N
(Fat sensitive)Type II B Plasma: turbidCholesterol $\uparrow$, Triglycerides $\uparrow$ LP Electrophoresis: no chylo, β $\uparrow$, pre β $\uparrow$
(Fat sensitive)Type III Plasma: usually turbidCholesterol $\uparrow$, Triglycerides $\uparrow$ LP Electrophoresis: no chylo, "Broad β " band
(Carbohydrate and fat sensitive)Type IV Plasma: usually turbidCholesterol $\uparrow$ or N, Triglycerides $\uparrow$ LP Electrophoresis: no chylo, β N, pre β $\uparrow$
(Carbohydrate sensitive)Type V Plasma: turbid with creamCholesterol $\uparrow$, Triglycerides $\uparrow$ LP Electrophoresis: chylomicrons $\uparrow$, β N, pre β $\uparrow$
(Carbohydrate and fat sensitive)Hypo-Beta Lipoproteinemia Plasma: clearCholesterol $\downarrow$, Triglycerides $\downarrow$ LP Electrophoresis: no chylo, β $\downarrow$, pre β $\downarrow$ Tangier Disease Plasma: clearCholesterol $\downarrow$, Triglycerides NLP Electrophoresis: very low to absent α
Orange tonsils

Source: University of Texas Medical Branch, Biochemistry
Lab, Galveston, Texas, 1980.

APPENDIX D

HUMAN RESEARCH REVIEW COMMITTEE APPROVAL AND
HOSPITAL PERMISSION FORMS

TEXAS WOMAN'S UNIVERSITY

Human Research Committee

Name of Investigator: Deborah J. Nickell Center: Dallas

Address: 8008 Seawall Blvd. #133 Date: 12/13/79
Galveston, Texas 77550

Dear Ms. Nickell:

Your study entitled Type A Coronary-Prone Behavior Correlated
With Cardiac Risk Factors

has been reviewed by a committee of the Human Research
 Review Committee and it appears to meet our requirements
 in regard to protection of the individual's rights.

Please be reminded that both the University and the
 Department of Health, Education and Welfare regulations
 require that written consents must be obtained from all
 human subjects in your studies. These forms must be
 kept on file by you.

Furthermore, should your project change, another
 review by the Committee is required, according to DHEW
 regulations.

Sincerely,

Estelle D. Kunt

Chairman, Human Research
 Review Committee

at Dallas.

TEXAS WOMAN'S UNIVERSITY
COLLEGE OF NURSING

AGENCY PERMISSION FOR CONDUCTING STUDY*

THE _____

GRANTS TO Deborah Jean Nickell R.N.

a student enrolled in a program of nursing leading to a Master's Degree at Texas Woman's University, the privilege of its facilities in order to study the following problem.

Type A Coronary-Prone Behavior Correlated With Cardiac Risk Factors

The conditions mutually agreed upon are as follows:

1. The agency (may) (~~may not~~) be identified in the final report.
2. The names of consultative or administrative personnel in the agency (may) (~~may not~~) be identified in the final report.
3. The agency (~~wants~~) (does not want) a conference with the student when the report is completed.
4. The agency is (willing) (~~unwilling~~) to allow the completed report to be circulated through interlibrary loan.
5. Other ----- Hospital requests the right to review
and approve any material that may be published as a
result of the study.

Date: 2/13/80

Deborah Nickell R.N.
Signature of Student

Signature of Agency Personnel

Beth C. Vaughan-Winkel, Ph.D.
Signature of Faculty Advisor

*Fill out & sign three copies to be distributed as follows:
Original - Student; First copy - Agency; Second copy - TWU
College of Nursing.

TEXAS WOMAN'S UNIVERSITY
COLLEGE OF NURSING

AGENCY PERMISSION FOR CONDUCTING STUDY*

THE _____

GRANTS TO Deborah Jean Nickell R.N.

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2. The names of consultative or administrative personnel in the agency (may) (may not) be identified in the final report.
3. The agency (wants) (does not want) a conference with the student when the report is completed.
4. The agency is (willing) (unwilling) to allow the completed report to be circulated through interlibrary loan.
5. Other _____

Date: 1/21/80

Signature of Agency Personnel

Deborah Nickell R.N.
Signature of Student

Bert C. Vaughan - M.D., R.N., Ed. D.
Signature of Faculty Advisor

*Fill out & sign three copies to be distributed as follows:
Original - Student; First copy - Agency; Second copy - TWU College of Nursing.

TEXAS WOMAN'S UNIVERSITY
COLLEGE OF NURSING

AGENCY PERMISSION FOR CONDUCTING STUDY*

THE _____

GRANTS TO Deborah Jean Nickell R.N.

a student enrolled in a program of nursing leading to a Master's Degree at Texas Woman's University, the privilege of its facilities in order to study the following problem.

Type A Coronary-Prone Behavior Correlated With Cardiac Risk Factors

The conditions mutually agreed upon are as follows:

1. The agency (~~may~~) (may not) be identified in the final report.
2. The names of consultative or administrative personnel in the agency (~~may~~) (may not) be identified in the final report.
3. The agency (wants) (~~does not want~~) a conference with the student when the report is completed.
4. The agency is (willing) (~~unwilling~~) to allow the completed report to be circulated through interlibrary loan.
5. Other _____

Date: 1/4/80

Signature of Agency Personnel _____

Deborah J. Nickell R.N.
Signature of Student

Beth C. Vaughan - M.Ed., R.N., Ed.D.
Signature of Faculty Advisor

*Fill out & sign three copies to be distributed as follows:
Original - Student; First copy - Agency; Second copy - TWU College of Nursing.

APPENDIX E

SUPPLEMENTARY TABLES

Table A

Frequency Counts: Subjects Per Hospitals Utilized

Hospitals	Absolute Frequency	Relative Frequency (Percent)
Hospital A	4	30.8
Hospital B	4	30.8
Hospital C	5	38.5
Totals	13	100.0

Note. Mode: Sample evenly distributed among hospitals.

Table B

Frequency Counts: Sex

Sex	Absolute Frequency	Relative Frequency (Percent)
Male	10	76.9
Female	3	23.1
Totals	13	100.0

Table C

Frequency Counts: Ages of Subjects

Age (years)	Absolute Frequency	Relative Frequency (Percent)	Cumulative Frequency (Percent)
31	1	7.7	7.7
34	1	7.7	15.4
37	1	7.0	23.1
38	1	7.7	30.8
39	1	7.7	38.5
51	1	7.7	46.2
54	1	7.7	53.8
58	1	7.7	61.5
61	2	15.4	76.9
64	1	7.7	84.6
65	1	7.7	92.3
68	1	7.7	100.0
Totals	13	100.0	

Table D

Age Ranges

Age Range	Number	Percent
31-40 years	5	38.5
51-60 years	3	23.0
61-70 years	5	38.5
Totals	13	100.0

Note. Mean 50.8, Variance 175.8, age data are skewed and show negative

Table E

Frequency Counts: Race

Race	Absolute Frequency	Relative Frequency (Percent)
White	12	92.3
Black	1	7.7
Totals	13	100.0

Table F

Frequency Counts: Marital Status

Marital Status	Absolute Frequency	Relative Frequency (Percent)	Cumulative Frequency (Percent)
Single	0	0.0	0.0
Married	8	61.5	61.5
Divorced	4	30.8	92.3
Widowed	1	7.7	100.0
Totals	13	100.0	

Note. Mode: Married, positively skewed.

Table G

Frequency Counts: Height

Height (inches)	Absolute Frequency	Relative Frequency (Percent)	Cumulative Frequency (Percent)
60.0	2	15.4	15.4
64.0	1	7.7	23.1
66.0	2	15.4	38.5
66.5	1	7.7	46.2
68.5	1	7.7	53.8
69.0	2	15.4	69.2
71.0	3	23.1	92.3
75.0	1	7.7	100.0
Totals	13	100.0	

Note. Mean 67.4, Median 68.5, Variance 19.06,
Mode 71.0, negatively skewed.

Table H

Frequency Counts: Weight

Weight (pounds)	Absolute Frequency	Relative Frequency (Percent)	Cumulative Frequency (Percent)
136	1	7.7	7.7
145	1	7.7	15.4
150	1	7.7	23.1
160	1	7.7	30.8
166	1	7.7	38.5
170	2	15.3	53.8
172	1	7.7	61.5
175	1	7.7	69.2
179	1	7.7	76.9
192	1	7.7	84.6
230	1	7.7	92.3
270	1	7.7	100.0
Totals	13	100.0	

Note. Median 170.5, Kurtosis is highly peaked, skewed to right at 2.872.

Table I

Frequency Counts: Pounds Overweight

Pounds Overweight	Absolute Frequency	Relative Frequency (Percent)	Adjusted Frequency (Percent)	Cumulative Frequency (Percent)
7	1	7.7	12.5	12.5
13	1	7.7	12.5	25.0
19	1	7.7	12.5	37.5
34	1	7.7	12.5	50.0
44	1	7.7	12.5	62.5
66	1	7.7	12.5	75.0
71	2	15.4	25.0	100.0
88 (not overweight)	5	38.5	N/A	
Totals	13	100.0		

Note. Median 36.5 pounds overweight, range 7 to 71 pounds overweight; 8 subjects were overweight, 5 were normal weight. Data negatively skewed, shows flatter kurtosis at -1.96.

Table J

Frequency Counts: Past History of Hypertension

Answer	Absolute Frequency	Relative Frequency (Percent)
Yes	4	30.8
No	9	69.2
Totals	13	100.0

Note. Mode: No past history of hypertension, negatively skewed.

Table K

Frequency Counts: Past History of Elevated Cholesterol

Answer	Absolute Frequency	Relative Frequency (Percent)
Yes	1	7.7
No	12	92.3
Totals	13	100.0

Note. Mode: No past history of elevated cholesterol, negatively skewed.

Table L

Frequency Counts: Past History of Elevated Triglycerides

Answer	Absolute Frequency	Relative Frequency (Percent)
Yes	3	23.1
No	10	76.9
Totals	13	100.0

Note. Mode: No past history of elevated triglycerides, negatively skewed.

Table M

Frequency Counts: Present History of Hypertension

Answer	Absolute Frequency	Relative Frequency (Percent)	Cumulative Frequency (Percent)
Yes	1	7.7	7.7
No	12	92.3	100.0
Totals	13	100.0	

Note. Mode: No present history of hypertension.

Table N

Frequency Counts: Present History of Elevated Cholesterol

Answer	Absolute Frequency	Adjusted Frequency (Percent)	Cumulative Frequency (Percent)
Yes	2	15.4	15.4
No	9	69.2	84.6
No data	2	15.4	100.0
Totals	13	100.0	

Note. Mode: No present history of elevated cholesterol.

Table O

Frequency Counts: Present History of Elevated Triglycerides

Answer	Absolute Frequency	Adjusted Frequency (Percent)	Cumulative Frequency (Percent)
Yes	5	38.5	38.5
No	5	38.5	76.9
No data	3	23.1	100.0
Totals	13	100.0	

Note. Mode: Possible trend for present history of elevated triglycerides, bimodal distribution.

Table P

Frequency Counts: Number of Myocardial Infarctions

Number	Absolute Frequency	Relative Frequency (Percent)
One	11	84.6
Two	2	15.4
Totals	13	100.0

Note. Mode: One myocardial infarction

Table Q

Frequency Counts: Family History of Premature Heart Disease

Answer	Absolute Frequency	Adjusted Frequency (Percent)
Yes	6	50.0
No	6	50.0
No Data	1	----
Totals	13	100.0

Note. Bimodal distribution except one with no data, negatively skewed.

Table R

Frequency Counts: Family History of Hypertension

Answer	Absolute Frequency	Relative Frequency (Percent)
Yes	11	84.6
No	2	15.4
Totals	13	100.0

Table S
Frequency Counts: Diabetics

Answer	Absolute Frequency	Relative Frequency (Percent)
Yes	1	7.7
No	12	92.3
Totals	13	100.0

Note. Mode: Majority of subjects not diabetic, negatively skewed.

Table T
Frequency Counts: Cigarette Smoking

Answer	Absolute Frequency	Adjusted Frequency (Percent)
Yes	8	66.7
No	4	33.3
No data	1	----
Totals	13	100.0

Note. Mode: Most subjects smoke, data negatively skewed.

Table U

Frequency Counts: Packs of Cigarettes Smoked

Number of Packs	Frequency	Percent
1	4	50.0
2	2	25.0
3	1	12.5
4	1	12.5
Totals	8	100.0

Note. Eight subjects smoked cigarettes.

Table V

Frequency Counts: Years Smoked

Number of Years Smoked	Absolute Frequency	Adjusted Frequency (Percent)	Cumulative Frequency (Percent)
1	1	12.5	12.5
5	1	12.5	25.0
12	1	12.5	37.5
15	1	12.5	50.0
38	2	25.0	75.0
40	1	12.5	87.8
44	1	12.5	100.0
88 (not applicable)	4		
99 (no data)	1		
Totals	13	100.0	

Note. Mean 25.4.

Table W
Frequency Counts: Exercise

Answer	Number	Percent
Yes	4	33.3
No	8	66.7
No data	1	----
Totals	13	100.0

Note. N = 12. Mode: No exercise, positively skewed data.

Table X
Comprehensive Data Sheet

Range of Scores	Risk Factors	Subjects	Past History of Hypertension	Past History of Elevated Cholesterol	Past History of Elevated Triglycerides	Family History of Premature Heart Disease	Diabetes	Cigarette Smoking	Exercise	Pounds Overweight	Actual Age (Years)	Actual Score	Range of Scores	
													High	Low
Low	6	1 Female	0	1	1	1	0	1	No	34	58	3	0	1
		Row	0	0	100.0	100.0	0	100.0		100.0	100.0	100.0	0	100.0
		Col	0	100.0	33.3	16.7	0	12.5		100.0	100.0	100.0	0	33.3
		Tot	0	7.7	7.7	8.3	0	8.3		12.5	7.7	7.7	0	7.7
High	5	2 Male	0	0	1	1	0	1	No	71	31	5	1	0
		Row	0	0	100.0	100.0	0	100.0		100.0	100.0	100.0	100.0	0
		Col	0	0	33.3	16.7	0	12.5		50.0	100.0	20.0	10.0	0
		Tot	0	0	7.7	8.3	0	8.3		12.5	7.7	7.7	7.7	0
High	3	3 Male	1	0	0	0	1	0	No	7	54	7	1	0
		Row	100.0	0	0	0	100.0	0		100.0	100.0	100.0	100.0	0
		Col	25.0	0	0	0	100.0	0		100.0	100.0	100.0	10.0	0
		Tot	7.7	0	0	0	7.7	0		12.5	7.7	7.7	7.7	0
High	4	4 Female	0	0	0	1	0	1	No	44	64	5	1	0
		Row	0	0	0	100.0	0	100.0		100.0	100.0	100.0	100.0	0
		Col	0	0	0	16.7	0	12.5		100.0	100.0	20.0	10.0	0
		Tot	0	0	0	8.3	0	8.3		12.5	7.7	7.7	7.7	0
High	2	5 Male	0	0	0	No Data	0	1	No	19	37	5	1	0
		Row	0	0	0		0	100.0		100.0	100.0	100.0	100.0	0
		Col	0	0	0		0	12.5		100.0	100.0	20.0	10.0	0
		Tot	0	0	0		0	8.3		12.5	7.7	7.7	7.7	0
Low	3	6 Female	0	0	0	1	0	0	No	66	65	4	0	1
		Row	0	0	0	100.0	0	0		100.0	100.0	100.0	0	100.0
		Col	0	0	0	16.7	0	0		100.0	100.0	50.0	0	33.3
		Tot	0	0	0	8.3	0	0		12.5	7.7	7.7	0	7.7
Low	2	7 Male	1	0	0	0	0	1	No	None	61	4	0	1
		Row	100.0	0	0	0	0	100.0			100.0	100.0	0	100.0
		Col	25.0	0	0	0	0	12.5			50.0	50.0	0	33.3
		Tot	7.7	0	0	0	0	8.3			7.7	7.7	0	7.7
High	4	8 Male	0	0	1	0	0	1	No	71	51	5	1	0
		Row	0	0	100.0	0	0	100.0		100.0	100.0	100.0	100.0	0
		Col	0	0	33.3	0	0	12.5		50.0	100.0	20.0	10.0	0
		Tot	0	0	7.7	0	0	8.3		12.5	7.7	7.7	7.7	0
High	2	9 Male	0	0	0	0	0	1	No	None	34	5	1	0
		Row	0	0	0	0	0	100.0			100.0	100.0	100.0	0
		Col	0	0	0	0	0	12.5			100.0	20.0	10.0	0
		Tot	0	0	0	0	0	8.3			7.7	7.7	7.7	0
High	1	10 Male	0	0	0	1	0	0	No	None	61	6	1	0
		Row	0	0	0	100.0	0	0			100.0	100.0	100.0	0
		Col	0	0	0	16.7	0	0			50.0	33.3	10.0	0
		Tot	0	0	0	8.3	0	0			7.7	7.7	7.7	0
High	3	11 Male	1	0	0	0	0	0	No	13	38	6	1	0
		Row	100.0	0	0	0	0	0		100.0	100.0	100.0	100.0	0
		Col	25.0	0	0	0	0	0		100.0	100.0	33.3	10.0	0
		Tot	7.7	0	0	0	0	0		12.5	7.7	7.7	7.7	0
High	3	12 Male	0	0	0	1	0	1	No	None	39	6	1	0
		Row	0	0	0	100.0	0	100.0			100.0	100.0	100.0	0
		Col	0	0	0	16.7	0	12.5			100.0	33.3	10.0	0
		Tot	0	0	0	8.3	0	8.3			7.7	7.7	7.7	0
High	2	13 Male	1	0	0	0	0	No Data	No	None	68	8	1	0
		Row	100.0	0	0	0	0				100.0	100.0	100.0	0
		Col	25.0	0	0	0	0				100.0	100.0	10.0	0
		Tot	7.7	0	0	0	0				7.7	7.7	7.7	0
X = 2.7 Column			4	1	3	6	1	8	9				10	3
Total			30.8	7.7	23.1	50.0	7.7	66.7	69.2				76.9	23.1

Table Y

Cross Tabulations: Average Bortner Scale Scores with Sex

Average Bortner Scale Scores	Male		Female	
	Number	Percent	Number	Percent
High (5-9)	9	90.0	1	33.3
Low (0-4)	1	10.0	2	66.7
Totals	10	100.0	3	100.0

Note. N = 13.

Table Z

Cross Tabulations: Average Bortner Scale Scores with Past History of Hypertension

Average Bortner Scale Scores	Yes		No	
	Number	Percent	Number	Percent
High (5-9)	3	75.0	7	77.8
Low (0-4)	1	25.0	2	22.2
Totals	4	100.0	9	100.0

Note. N = 13.

Table AA

Cross Tabulations: Average Bortner Scale Scores with
Current Hypertension

Average Bortner Scale Scores	Yes		No	
	Number	Percent	Number	Percent
High (5-9)	2	66.7	8	80.0
Low (0-4)	1	33.3	2	20.0
Totals	3	100.0	10	100.0

Note. N = 13.

Table BB

Cross Tabulations: Average Bortner Scale Scores with
Past History of Elevated Cholesterol

Average Bortner Scale Scores	Yes		No	
	Number	Percent	Number	Percent
High (5-9)	0	0	10	83.3
Low (0-4)	1	100.0	2	16.7
Totals	1	100.0	12	100.0

Note. N = 13

Table CC

Cross Tabulations: Average Bortner Scale Scores with
Past History of Elevated Triglycerides

Average Bortner Scale Scores	Yes		No	
	Number	Percent	Number	Percent
High (5-9)	2	66.7	8	80.0
Low (0-4)	1	33.3	2	20.0
Totals	3	100.0	10	100.0

Note. N = 13.

Table DD

Cross Tabulations: Average Bortner Scale Scores with
Currently Elevated Cholesterol

Average Bortner Scale Scores	Yes		No		No Data	
	Number	Percent	Number	Percent	Number	Percent
High (5-9)	1	50.0	7	77.8	2	100.0
Low (0-4)	1	50.0	2	22.2	0	0
Totals	2	100.0	9	100.0	2	100.0

Note. N = 12, no data from one respondent.

Table EE

Cross Tabulations: Average Bortner Scale Scores with
Currently Elevated Triglycerides

Average Bortner Scale Scores	Yes		No		No Data	
	Number	Percent	Number	Percent	Number	Percent
High (5-9)	3	60.0	4	80.0	3	100.0
Low (0-4)	2	40.0	1	20.0	0	0
Totals	5	100.0	5	100.0	3	0

Note. N = 12, no data from one respondent.

Table FF

Cross Tabulations: Average Bortner Scale Scores with
Packs of Cigarettes Smoked per Day

Average Bortner Scale Scores	Packs							
	1		2		3		4	
	Num- ber	Per- cent	Num- ber	Per- cent	Num- ber	Per- cent	Num- ber	Per- cent
High (5-9)	2	50.0	2	100.0	1	100.0	1	100.0
Low (0-4)	2	50.0	0	0	0	0	0	0
Totals	4	100.0	2	100.0	1	100.0	1	100.0

Note. N = 8, no data from four respondents.

Table GG

Cross Tabulations: Average Bortner Scale Scores with Years Smoked

Average Bortner Scale Scores		Years									
		1	5	12	15	38	40	44			
Num- ber	Per- cent	Num- ber	Per- cent	Num- ber	Per- cent	Num- ber	Per- cent	Num- ber	Per- cent	Num- ber	Per- cent
High (5-9)	1	100.0	1	100.0	1	100.0	2	100.0	0	0	0
Low (0-4)	0	0	0	0	0	0	0	1	100.0	1	100.0
Totals	1	100.0	1	100.0	1	100.0	2	100.0	1	100.0	1

Note. N = 8.

Table HH

Cross Tabulations: Average Bortner Scale Scores with Diabetes

Average Bortner Scale Scores	Yes		No	
	Number	Percent	Number	Percent
High (5-9)	1	100.0	9	75.0
Low (0-4)	0	0.0	3	25.0
Totals	1	100.0	12	100.0

Note. N = 13.

Table II

Cross Tabulations: Average Bortner Scale Scores with Race

Average Bortner Scale Scores	White		Black	
	Number	Percent	Number	Percent
High (5-9)	9	75.0	1	100.0
Low (0-4)	3	25.0	0	0
Totals	12	100.0	1	100.0

Note. N = 13.

Table JJ

Cross Tabulations: Average Bortner Scale Scores with
Marital Status

Average Bortner Scale Scores	<u>Single</u>		<u>Married</u>		<u>Divorced</u>		<u>Widowed</u>	
	Num- ber	Per- cent	Num- ber	Per- cent	Num- ber	Per- cent	Num- ber	Per- cent
High (5-9)	0	0	7	87.5	3	75.0	0	0
Low (0-4)	0	0	1	33.3	1	25.0	1	100.0
Totals	0	0	8	100.0	4	100.0	1	100.0

Note. N = 12, no data from one respondent.

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