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UNIVERSITY OF OKLAHOMA

GRADUATE COLLEGE

THE RELATIONSHIPS AMONG DEPRESSION, MARITAL SATISFACTION, AND CARDIAC RECOVERY IN THE DAYS AND MONTHS POST HOSPITALIZATION FOR MYOCARDIAL INFARCTION AND UNSTABLE ANGINA

A Dissertation

SUBMITTED TO THE GRADUATE FACULTY

in partial fulfillment of the requirements for the

degree of

Doctor of Philosophy

By

JIM R. KELLER, M.ED. 1997

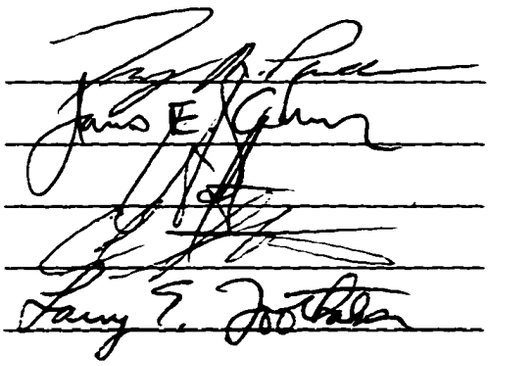
Norman, Oklahoma

THE RELATIONSHIPS AMONG DEPRESSION, MARITAL SATISFACTION, AND CARDIAC RECOVERY IN THE DAYS AND MONTHS POST HOSPITALIZATION FOR MYOCARDIAL INFARCTION AND UNSTABLE ANGINA

A Dissertation APPROVED FOR THE

DEPARTMENT OF EDUCATIONAL PSYCHOLOGY

BY



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Abstract

The current study sought to build upon existing literature by examining the relationships among depression, marital satisfaction, and cardiac recovery from the time of hospitalization to 5-8 months post-discharge for myocardial infarction and unstable angina. To better illustrate the theoretical linkages between depression, marital satisfaction and cardiac functioning, a *causal pathway model* was created as a framework to aid research and clinical practice. A self-report instrument (*Myocardial Infarction Risk Factor Inventory: MIRFI*) was also devised to screen for five of the more recognized psychosocial risk factors linked to heart disease and impeded recovery (depression, marital satisfaction, Type A, hostility, and social support). To assess recovery, the *Myocardial Infarction Recovery Index-Short Form* was also created by the author as an adaptation from a measure first proposed by Utz and Beach (1988). Unlike previous research, both myocardial infarction and unstable angina patients were studied, and special attention was paid to the interaction of depression and marital functioning as it relates to cardiac recovery.

Fifty-three male myocardial infarction and unstable angina patients hospitalized in a V.A. Medical Center were assessed for depression, marital satisfaction, and several other psychosocial risk factors while in the hospital. Thirty patients were again assessed between five and eight months post-discharge. Results indicated that myocardial infarction and unstable angina patients did not differ statistically on any of the psychosocial or recovery measures, and, therefore, appear comparable in terms of psychosocial functioning. Among the baseline sample (N=53), the incidence of major depression was nearly six times greater than the general adult male population. Findings indicated that higher degrees of depression at baseline and at follow-up were associated with poorer recovery. Social support at baseline was also found to be significantly correlated with the recovery measure. Preliminary analysis suggested that the MIRFI Marital Satisfaction and Depression subscales related significantly to their parent measures. Proposed explanations for the findings, as well as implications for clinical practice and future research are discussed.

CHAPTER I: INTRODUCTION

Background of the Problem

Context and Rationale

Many argue that coronary heart disease (CHD) is the most pressing health care issue facing this country today in terms of loss of life, human suffering and social cost. Indeed, the numbers seem to support this claim. Of the 1.5 million Americans that suffer an initial or recurrent myocardial infarction (MI), or heart attack, each year, over 500,000 die (American Heart Association, 1989). Of the survivors, a significant proportion will experience a substantial loss of wages or be unable to return to work at all. Most will require additional medical treatment and/or rehabilitation services, and a frightening number will experience recurrent cardiac events. Most staggering is that an estimated 5 million people in the United States have medically relevant CHD, of which 2.5 million experience some form of disability from the disease (American Heart Association, 1989). These figures translate into a $40 billion price tag for CHD via medical expenses, lost wages and productivity, not to mention the costs stemming from the emotional and physical impact of the disease on loved ones and associates. In sum, CHD burdens a great number of Americans...a large segment directly through the traumatic and debilitating disease process itself, and society as a whole via lost productivity and increased health care costs. Hence, the effective prevention of CHD, as well as the improved treatment and rehabilitation of the CHD patient seems a pressing challenge warranting substantial multidisciplinary attention.

A significant proportion of this country's 500,000 annual cardiac deaths are thought to be associated with *recurrent* cardiac events, that is to say the occurrence of myocardial infarction, ventricular fibrillation and cardiac arrest subsequent to a patient's first heart attack. In an attempt to reduce total cardiac deaths in this country, increased attention has been afforded not only to those factors that may enhance CHD onset

and/or progression, but those that may impede post MI recovery as well (American Heart Association, 1988). As will be detailed in the pages to follow, researchers have, for several decades, sought to identify factors related to CHD that transcend the purely physiological mechanisms (i.e., cholesterol, vascular disease etc.). It was clear many years ago that heart disease and cardiac recovery entailed far more than solely biological forces. Accordingly, the focus has shifted toward a biopsychosocial model of CHD and factors that involve the interaction of psychological, social and biological realms of human functioning. From these efforts, several biopsychosocial factors thought to contribute to CHD and cardiac recovery have been identified. Four of the more prominent are: (1) marital distress; (2) depression; (3) hostility; and (4) Type A behavior pattern. These and additional CHD factors will be discussed in greater detail within the literature review section.

Context & Duration of the Study

Why Target the Unstable Angina & Post-MI Patients 5 to 8 Months After Hospitalization?

About 1.5 million people in this country suffer a first or recurrent myocardial infarction per year, and of these over a third die (American Heart Association, 1988). Since peaking in the 1960's, however, there has been a steady 3% decrease in both heart attacks and cardiac death in the past few decades. Many factors have been linked to this decline including more efficient emergency care and greater sophistication of in-hospital medical treatment.

It is widely held that the reduction in cardiac deaths is due mainly to the growing emphasis on primary care, or, more specifically, the prevention of CHD. The medical community's actions of late seem to reflect this trend toward prevention, for the bulk of its CHD research and public awareness campaigns focus almost exclusively on risk factors such as diet, smoking, exercise and stress management (Smith & Leon,

1992). Third-party payers and even the U.S. government now seem to actively endorse the old notion. "an ounce of prevention is worth a pound of cure".

There is little argument that it is far more cost effective to avoid heart disease altogether than to treat advanced CHD or rehabilitate the MI patient. But. should primary prevention be our *only* concern? Many of this nation's 1.5 million heart attacks occur in the weeks and months following hospitalization for unstable angina (persistent chest pain that marks the presence of severely advanced CHD and impending infarction) and a myocardial infarction. Certainly. the prevention of CHD is ideal. but the data suggest that these efforts should not overshadow the needs of the patient who has already succumbed to unstable angina or a heart attack. If the overall objective is the reduction of cardiac death. it is of questionable logic to exert *all* resources to forestalling the development of CHD. while allowing an avoidable first or second MI to incapacitate or kill. To truly have an impact on the annual cardiac-related deaths in this country. energy must be expended in the prevention of *any cardiac event that threatens life*...be it a first or a fifth.

To maximize preventive efforts. it seems logical that energy be channeled toward periods when the risk of an MI occurrence is greatest. Both theory and epidemiologic data strongly suggest that risk of an acute cardiac event and premature death is most pronounced during the period following hospitalization for unstable angina or an initial myocardial infarction (Kannel. et al.. 1979: Moss & Benhorin. 1990: Norris et al.. 1984: Smith & Leon. 1992). Moss and Benhorin (1990) in a review of nine studies utilizing a combined sample of over 6000 post MI patients. report that in *100% of the studies* the majority of recurrent cardiac events (nonfatal reinfarction or cardiac death) occurred within six months after hospital discharge. While type of infarct. the amount of tissue damage and medical intervention are by far the best predictors of prognosis (Ahnve et al.. 1988). level of improvement within this

recovery phase tends to be a relatively good indicator of longer-term prognosis (Moss & Benhorn, 1990). For these reasons, the recovery phase 6 to 9 months post MI appears to be one of the most appropriate contexts in which to target a wide array of MI risk factors, and an ideal context for exploring biopsychosocial correlates of CHD.

Due to the similarities between hospitalized unstable angina and post-MI patients in terms of disease severity, risk of infarction, and the 6 to 9 month post-hospitalization critical period (an issue to be discussed in greater detail in a later section), the current study seeks to investigate the role of psychosocial variables in the recovery of hospitalized unstable angina and post myocardial infarction patients.

Objectives and Overview of the Study

Objectives

Several factors including Type A Behavior Pattern (TABP) and hostility have received much empirical attention in recent years within the cardiology literature. Marital distress and depression have also been examined as correlates of CHD and cardiac rehabilitation. However, relative to Type A and hostility, these studies are relatively few in number and lack replication. Moreover, no study has been found that incorporates *both* marital distress and depression in a single design as is proposed here. Therefore, the current study seeks to break new ground by addressing marital distress and depression as biopsychosocial mediators of heart disease, with special emphasis on the interaction between the two within the five to eight months post hospitalization for unstable angina and myocardial infarction. In sum, this study is thought not only to augment the dearth of data concerning marital distress and depression as *independent* mediators of cardiac recovery, but to provide the first investigation into the interplay among the variables as well.

A second objective of the project is to investigate the main and/or interactive effects of a handful of other biopsychosocial factors that the literature suggests may be

linked to cardiac rehabilitation as well (referred to as *collateral and MIRFI* variables in later sections). Specifically. Type A. hostility and social support will be assessed along with marital distress and depression through the use of an original assessment device that was designed by the principal investigator to screen for these factors without requiring the participant to complete a separate instrument for each variable. Use of the *Myocardial Infarction Risk Factor Inventory* (MIRFI) will serve two purposes: (1) to tap these additional CHD variables for statistical analysis: and (2) to serve as a pilot study exploring the ability of a brief (30-item) self-report measure to screen for these elements. It is hoped that the MIRFI will evolve into a screening tool for general medical practitioners. cardiologists. health psychologists. and allied health professionals. With the dawning of managed care. medical professionals will shoulder more of the responsibility for identifying relevant psychological issues and making appropriate mental health referrals. Hence. reliable screening measures are needed. In addition. those working specifically with the cardiology patient will be under greater pressure to more efficiently conceptualize a case and prevent rehospitalization. The MIRFI might offer utility in both capacities.

A third goal of the current study emerged quite by accident. While struggling to clarify the key links between marital distress. depression and coronary heart disease. the authors found themselves confused by the lack of conceptual cohesion binding the many biological. psychological and environmental pieces together. Therefore. a conceptual framework seemed necessary to bring order to a fragmented and voluminous literature base such that a cogent review and research problem could be presented. As a result. a model was created delineating an overarching causal system of coronary heart disease (See Appendix D). As one will see. such a model allows for a more coherent discussion of marital distress and depression as central psychosocial factors of CHD in that their respective roles in the disease process are plotted on a

causal pathway. A conceptual road map of sorts, the *Causal Pathway Model of CHD* delineates the various trails from a psychosocial variable (i.e., depression, Type A, marital distress, hostility) to its impact on cardiac functioning. Since marital distress and depression are but two variables in this complex interactive system, several of the major biopsychosocial factors of CHD are also included in the causal pathway model which serves as the framework for the Relevant *Literature* section.

Finally, mainstream counseling psychology research has long been attacked for its lack of clinical relevance (Gelso, 1979). Moreover, few would argue that psychosocial theory has little enduring value if it spurs no practical utility. In this vein, the application of the biopsychosocial causal pathway model of CHD should apply not only to better etiological understanding, but to clinical application as well. The current study seeks not only to reinforce the importance of several key CHD contributors, but to assist in the formulation of psychosocial strategies aimed at their demise as well. In short, it is a goal of this project to apply both theory and resulting data to the actual treatment of the cardiac patient. To this end, the literature review section includes assessment and treatment implications from the perspective of the biopsychosocial causal pathway model of CHD. Included is a brief review of assessment and treatment literature of the leading psychosocial factors of CHD, including depression and marital distress. The section culminates with a proposed model for psychological intervention with the cardiac patient. While the section may travel beyond the scope of the formal study, it is felt that an analysis of how the project might impact clinical practice further justifies its implementation.

The Research Problem

An examination of the relationships among depression, marital distress and cardiac recovery following hospitalization for unstable angina or heart attack will be the primary focus of the current study. As such, discussions of *a priori* research

questions, hypotheses, designs and statistical analysis procedures will focus only on these central aspects of the study.

Included as a secondary venture will be the role of Type A, hostility and social support (via the Myocardial Infarction Risk Factor Inventory, MIRFI, to be discussed in a later section) as they relate to recovery, marital distress and depression.

The overriding research problem guiding the exploration of all aspects of the current study is to what extent do each of the above biopsychosocial factors impact cardiac recovery during the five to eight months post hospitalization. Of particular interest are any interactions among the variables that may account for notable trends in cardiac recovery.

Specific Areas of Interest

Several specific *a priori* investigations have been formulated to address the overarching research problem. The reader will note that the emphasis of the project is on the primary variables in question (marital satisfaction & depression). Analysis of the collateral variables (the MIRFI variables), therefore, is considered exploratory. The primary relationships under investigation include: (a) the association between depression and cardiac recovery; (b) the association between marital satisfaction and cardiac recovery; and (c) any interaction or interdependence between depression and marital satisfaction as they relate to cardiac recovery.

Secondary objectives of the current study include: (a) the impact of the collateral variables (Type A, hostility, social support, and treatment compliance) on recovery; (b) the relative strengths of each of the collateral variables, as well as depression and marital satisfaction, in predicting recovery; (c) the utility of the MIRFI as a screening instrument for depression and marital satisfaction; and (d) the utility of the MIRFI as a gauge of cardiac risk and predictor of recovery in the 5 to 8 months post-hospitalization.

Research Questions

The above explorations bring about several specific research questions.

(1) Do myocardial infarction and unstable angina patients have higher rates of depression than the general population?

(2) Are MI and UA patients statistically comparable on the key variables of interest?

(3) Is depression associated with poorer recovery in the months post-hospitalization?

(4) Is depression at Time 2 associated with poorer recovery?

(5) Is marital satisfaction associated with recovery post-hospitalization?

(6) Is marital satisfaction at Time 2 associated with recovery?

(7) Do depression and marital satisfaction somehow interact to impact recovery?

(8) Among the primary (depression and marital satisfaction) and secondary (MIRFI) variables. is there a model that is most predictive of recovery?

(9) Is the MIRFI an effective screening instrument for depression and marital distress?

Hypotheses

Based upon previous research and theory. several specific outcomes are predicted.

(1) Post-MI and UA patients in distressed marital relationships will experience poorer recovery than those entering the recovery period in non-distressed marriages.

(2) Participants who are clinically depressed post-hospitalization will experience poorer cardiac recovery during the first five to eight months of recovery.

(3) A positive association will emerge between marital distress and depression.

(4) A negative association will emerge between depression at baseline and cardiac recovery.

(5) A positive association will emerge between marital satisfaction and recovery.

CHAPTER II: RELATED LITERATURE

Biopsychosocial Factors of CHD within a Causal Pathway Model

Beyond a Solely Biological Focus

The federal government, medical and academic communities and the corporate world have long understood the relevance of heart disease, and accordingly, CHD is one of the most widely and thoroughly researched issues to date. Understandably, throughout the course of CHD's empirical history, much attention has been paid to the primary biomedical correlates of the disease (i.e., cholesterol, hypertension, circulatory disorders etc.). However, extending back to the early stages of contemporary investigations (circa 1950's), the research has consistently demonstrated the inadequacy of solely biomedical factors in explaining the incidence, development and exacerbation of CHD and acute cardiac events (i.e., MI and ventricular fibrillation). In response, the role of behavior (i.e., smoking, diet, exercise, work habits etc.) and psychosocial factors in the coronary artery disease (CAD) process have risen in priority (Carroll, 1992; Smith & Leon, 1992). Within the past three decades, medical research has increasingly examined psychosocial variables as potential mediators of illness. Stress has long been the focus of the vast majority of these efforts due its prevalence within American society, and its apparent impact on both physiology and emotional functioning. Until the late 1970's, no comprehensive paradigm existed from which to conceptualize the interplay between psychosocial and physiological functioning.

A Biopsychosocial Emphasis

Within the last two decades a *biopsychosocial model* has gained favor in medical and psychological camps (Engel, 1977). A biopsychosocial model, as the term implies, emphasizes the reciprocal interaction among biological, psychological, environmental and behavioral components in wellness and disease. As such, it offers a

comprehensive framework within which to conceptualize and empirically evaluate a host of medical and psychological disorders. In an apparent response to the shortcomings of etiological theory positing purely physiological bases of CHD, many leaders within traditional cardiac research have gradually aligned with a biopsychosocial (or an equivalent by another name) interactive model of heart disease (Carroll, 1992; Price, 1988; Smith & Leon, 1992; Waltz, 1986). Coronary heart disease seems especially well suited to a biopsychosocial perspective, for few other disease processes have amassed the impressive quantity of empirical research supporting a causal interaction among the *individual* (biology & psychology), the *environment* (social, economic and emotional resources) and *behavior* (diet, exercise, smoking etc.).

It is for the above reasons that the underpinnings of this study, both theoretical and empirical, stem from a biopsychosocial view of coronary heart disease.

Psychosocial Factors as Correlates of CHD

From the study of biopsychosocial processes, a multitude of *psychosocial constructs* thought to be central to CHD have emerged. The term *construct* is used to reflect the merging of the three major domains of human functioning, the person, the world and behavior, (a process Bandura termed *triadic reciprocality*; 1986) such that a factor (or construct) emerges. In the case of coronary heart disease, several of these constructs have been identified as contributors to the disease process and acute cardiac events. Consistent with a biopsychosocial tradition, they are not pristine, unidimensional causal variables, but rather complex byproducts of the interactive nature of human existence. A handful of these psychosocial constructs and their relationship to cardiac recovery will be the primary focus of the following literature review and form the conceptual basis for the current study. They are: (1) marital distress; (2) depression; (3) hostility; and (4) Type A Behavior Pattern; and (5) Social Support.

Extending the Biopsychosocial Foundation: Toward a Comprehensive Model of CHD

Much of the theoretical and empirical CHD research has been channeled toward the identification of the psychosocial correlates of CHD and poor cardiac recovery. This work has been essential in building a foundation for a biopsychosocial understanding of CHD in that it has spawned a host of psychosocial contributors, including those mentioned in the preceding paragraph. While several authors have demonstrated such constructs to be correlates of poor cardiac functioning, relatively few have sought to explain just how one factor might relate to another, or how a phenomena at the psychosocial level might influence cardiac physiology. When reviewed in the literature, psychosocial factors of CHD are typically presented in a very segregated, compartmentalized manner. For example, a prototypical article or book chapter focusing on the biopsychosocial perspective of CHD will often provide a general explanation of the link between Biopsychosocial Factor A and CHD and Psychosocial Factor B and CHD (independent of one another). The *big picture*...or just how Factor A and Factor B fit into the overall disease process, however, is typically not delineated.

The author felt that this lack of theoretical cohesion contradicts the most central tenets of a biopsychosocial perspective...that is the ongoing cybernetic, bidirectional interaction of variables such that a system is maintained. To fully appreciate the role Factor A has in fostering CHD, one must, in my opinion, have a sense of its place in the overarching system. For, Factor A may only have an effect on coronary function in the presence of Factor B, or they may share causal properties. Moreover, without a road map of the system, determining appropriate inroads and targets for psychological intervention would be difficult. Hence, it is posited here that research must address the *systemic interaction* of the many biopsychosocial factors of CHD if relevant theory and efficient clinical applications are to emerge.

Toward this end, a comprehensive *Causal Pathway Model of CHD* (see

Appendix A) is proposed so as to provide some theoretical order to the currently chaotic biopsychosocial research of heart disease. Specifically, the model has been formulated, based on current literature, to suggest an overarching system within which many variables and processes interact to bring about CHD and cardiac events (i.e., heart attack). While the purpose of the proposed study is not to validate such a model, it seems that a conceptual framework is necessary to fully appreciate where in the causal system each of the CHD psychosocial factors in question reside. Moreover, it is hoped that the model relays, both visually and conceptually, a stronger appreciation for the rationale and hypotheses of the current project, as well as setting the stage for analysis of additional CHD factors in future research. As we will see, the model may also offer utility in guiding both the assessment and treatment of psychosocial correlates of coronary heart disease as well.

In the following section, the use of causal pathway modeling in CHD research and clinical practice is reviewed. Included will be a discussion of the causal factors of CHD per the literature, including those psychosocial factors that have been identified as contributors to CHD and critical cardiac events. The section concludes with an examination of the potential applications of causal pathway modeling in psychological treatment of the cardiac patient, thus bolstering the argument for continued research into psychosocial aspects of coronary heart disease.

Causal Pathway Modeling in CHD

The term *causal pathway* has many possible connotations and definitions. For the purposes of this exploration, however, a causal pathway is seen as a conceptual roadway comprised of many interacting factors such that, when viewed from a distance, a logical progression is conveyed. Some of these pathways may be more analogous to a one-way street in which travel (causation) is generally unidirectional. Injury or trauma could be one example of a more linear causal pathway. That is, a

triggering context (the poison) travels in a generally unidirectional fashion eliciting mediating *factors* (e.g., pulmonary distress, convulsions, cardiac arrhythmia) which lead to the onset of a *critical event* (e.g., heart attack, cardiac arrest) and possibly resulting in a true *endpoint* (i.e., death).

Most disease processes, however, are more consistent with an interstate highway. These pathways often involve multiple *lanes of traffic* traveling both directions, with various detours, and many on/off ramps leading to multiple potential destinations. In the case of such complex causal pathways, variables possess bidirectional influence such that causality is not purely linear. Coronary heart disease may best be described as such a pathway. To reflect this biopsychosocial highway of CHD etiology, the *Causal Pathway Model of CHD* is promulgated within the framework of Bandura's (1986) *Triadic Reciprocality* theory (See Appendix D, Fig. 1). Specifically, this model is designed to emphasize the interactions among the environment, the individual and behavior in the CHD process, and to serve as the conceptual basis for the delineation of more specific causal pathways within CHD (e.g., mapping the path from depression to ventricular fibrillation).

To lay the foundation for later discussion of the specific pathways between psychosocial factors and the physiology of CHD, an overview of the pathophysiology of heart disease is necessary and will be included below. Each component is briefly defined, and its suggested place in the overarching causal pathway of CHD is presented. It should be noted that the terminology used to define and describe the various aspects of the CHD pathway is an integration of that found in the literature and this author's conceptualization of a pathway model.

Several Key Biopsychosocial Factors
Within the Context of a Causal Pathway Model of CHD

As mentioned above, a causal pathway represents an interaction among

triggering contexts. mediating variables (primary. secondary. tertiary. etc.) and critical events. The following examines the literature pertaining to a host of central aspects of CHD within this framework. including those featured as foci of the current study.

Cardiac Pathophysiology:

Critical Cardiac Events in the CHD Causal Pathway

A *critical event* within a causal pathway framework represents the culmination of the many mediating processes (primary. secondary. etc.) along the pathway such that a physiological expression of an advanced disease state is evidenced. While death is the only true endpoint in the CHD etiological highway. critical events serve as a type of *weigh station* from which the subsequent course of the disease is determined. Two key critical cardiac events that have received much play in the biopsychosocial literature are *myocardial infarction* and *cardiac arrest.* The pathophysiology of these critical cardiac events follows so as to illustrate the possible entryways for mediator (psychosocial. biological or biobehavioral) influence.

Myocardial Infarction

The heart is a pump that relies upon the repeated contraction of muscle. the *myocardium*. to force the flow of oxygenated blood throughout the body and spent blood to the lungs. Like all muscle. the myocardium requires oxygen to survive. In a healthy person. oxygenated blood flows freely through the branches of the *aorta* (the body's chief artery) such that the heart can extract the oxygen it needs and send the remainder on its way. Without the ability to store oxygen. the heart must meet increased demand (e.g.. during vigorous exercise) by increasing the number of contractions. When demands are not met. the myocardium may run short of oxygen. a process called *ischemia*. If ischemia persists for 1-3 hours. myocardial cell death (necrosis) may occur resulting in a heart attack. or *myocardial infarction* (MI). The severity of an MI depends largely on the amount and location of the myocardial

necrosis (or infarct). Less than 90% of all infarcts lead to death if the patient reaches a hospital (Thom. et al.. 1985).

Within a CHD causal pathway model. MI can be viewed as a *critical event* for heart attack and may lead to cardiac death. further cardiac damage or serve as a complication of rehabilitation. Nevertheless. MI is a traumatic and powerful determinant of a patient's fate. For. it can be a turning point (i.e.. alerting the individual of the need for treatment and improved behavior). or a turn for the worse.

Cardiac Arrest

Cardiac arrest (CA) differs from myocardial infarction in that tissue damage is not a prerequisite for interrupted cardiac function. Arrest literally describes a state in which the heart ceases to contract. A variety of factors have been implicated in cardiac arrest including ischemia. arrhythmia as well as many externally induced triggers such as electrocution. toxins and systemic shock.

Cardiac Pathophysiology:

Predisposing Cardiac Events

When CHD is severely advanced. dramatic impairment of cardiac functioning results. Due to these significant impairments. critical cardiac events. if left untreated. are considered inevitable. It is for this reason that a category has been created within the causal pathway model of CHD identifying cardiac changes that. while not critical (life threatening) in themselves. predispose the patient to critical cardiac events such as myocardial infarction and cardiac arrest. Unstable angina is a primary example of such a *predisposing cardiac event.*

Unstable angina (sometimes referred to as *crescendo angina*) is a diagnosis referring to a pattern of persistent chest pain that is the result of significant arterial blockage and ischemic episodes. Unstable angina differs from *angina pectoris* (literally. "pain in the chest) in that angina merely denotes the presence of chest pain

thought to be linked to impaired cardiac functioning. The diagnosis of angina pectoris refers to chest pain that emerges only when notable strain is placed upon the cardiovascular system resulting in ischemia and associated chest pain. This pain is usually predictable in that patients learn that strenuous activity (i.e., exercise, walking stairs, doing yard work etc.) is usually the trigger. Therefore, the pain is often relieved by simply avoiding or reducing the level of physical activity. In contrast, the qualifier *unstable* is usually applied in cases where severe pain arises *without* significant strain on the cardiovascular system. In fact, unstable angina patients have such severe blockage that typically no movement is required to bring about chest pain. These patients suffer pain while at rest or even while sleeping, and medication such as nitroglycerine is usually necessary to reduce the symptoms.

Unstable angina patients are, by definition, those who have such severely advanced CHD that their arterial blockage brings about ischemic episodes even in the absence of added strain on the system. Due to the severity of the disease, physicians often consider unstable angina as a state of *impending infarction*. In other words, unstable angina is really a heart attack in the making: the arterial blockage is there, the ischemia is there, the pain is there, yet death of myocardial muscle has not occurred.

Due to the graveness of the unstable angina patient's condition, he is typically treated in the same way as the patient having suffered an actual heart attack. Specifically, hospitalization is often necessary. While in the hospital, the unstable angina patient is placed on a heart monitor, and commonly undergoes similar (or the same) medical interventions. Anti-thrombolytic medications, heart catheterization, angioplasty, and bypass surgery are often utilized in treating both the unstable angina and post-myocardial infarction patient.

In addition, many of the same psychosocial factors affecting the heart attack victim are also experienced by those suffering from unstable angina. Both deal with

almost daily pain. have their lives interrupted by the need for limited physical activity and stress. and both are thought to have similar fears of death and debilitation. Accordingly. it has been proposed that the families of unstable angina and heart attack patients share many of the same concerns and hardships. Due to the proposed physiological and psychosocial similarities between these two types of cardiac patients. the current study will combine them in the majority of analyses regarding their recovery of 6 to 9 months post-hospitalization.

Unfortunately. few empirical studies have compared the unstable angina and heart attack patient on specific psychosocial variables. The current study seeks to explore (and possibly confirm) the statistical and practical similarities between these two closely linked patient populations.

Cardiac Pathophysiology:

Primary Mediators in the CHD Causal Pathway

As the title and model imply. primary mediators are processes that *most directly* contribute to critical cardiac events. While influenced by psychosocial and behavioral factors. primary mediators are expressed in the physiological domain which accounts for their proximity to critical cardiac events such as MI and CA.

Atherosclerosis

The most common cause of ischemia. and eventual MI. is the narrowing of significant arteries. or a CAD process called *atherosclerosis*. Simply put. fatty substances (cholesterol) in the blood latch on to artery walls. gradually blocking blood flow (*occlusion*). Over time. calcium deposits may coat cholesterol plaque. hardening the artery and further restricting blood flow. The occlusion of a major artery or several medium sized arteries reduces the blood flow to the heart. and in turn. limits the oxygen to the myocardium and other key mechanisms. As stated above. if occlusions prolong ischemia. among other things. MI may result.

Because of the direct role atherosclerosis plays in CHD and major cardiac events, it is often termed a *primary causal agent* in the etiological pathway to CHD, MI and cardiac death. As we will see, several psychosocial factors have been implicated in the enhancement of atherosclerosis.

Arrhythmia and Ventricular Fibrillation

A second *primary mediator* in the pathway to CHD, MI and cardiac death is *arrhythmia, or in the extreme, ventricular fibrillation* (VF). Within the literature, VF has been regarded as both critical event and casual agent depending on one's perspective. To this author its distinction is really a matter of semantics. Therefore, VF will be outlined here as a primary mediator of CHD. VF is considered primary because it can contribute directly to myocardial infarction or sudden cardiac death.

Normally, an electrical impulse (originating from the *atria*) travels across the ventricles triggering the orderly contractions of the myocardial fibers. The systematic fiber contractions insure an effective ventricular pumping action. When contractions are irregular, the heart beats in an abnormal and often less efficient manner (*arrhythmia*). VF is an extreme form of arrhythmia in which the muscle fibers contract in total disharmony resulting in an unproductive ventricular spasm. During such a spasm, little or no circulation is accomplished, and vital organs are rapidly denied oxygen. There may be three outcomes of VF: (1) normal contractions and systemic circulation are restored via CPR and/or electrical shock (defibrillation); (2) poor circulation leads to extended ischemia and MI; or (3) cardiac arrest ensues resulting in sudden cardiac death and/or brain death.

The causes of VF are debated by researchers. Ischemia is considered a contributor to the disorganization of the electrical conduction mechanism causing arrhythmia, including ventricular fibrillation (Lown et al., 1980). This process can occur independent of, or in association with MI. As will be discussed more fully in a

following section. severe emotional-physiological arousal and even psychopathology (i.e.. depression and anxiety) have been implicated in VF onset (Katz et al.. 1985: Lown. 1987: Lown et al.. 1980: Orth-Gomer et al.. 1980: Tavazzi et al.. 1986).

Cardiac Pathophysiology:

Secondary Mediators in the Causal Pathway of CHD

Three biopsychosocial phenomena that seem to be expressed most potently at the biological level are promulgated here as *secondary* mediators of CAD development and exacerbation. They are: hypertension. hypercholesterolemia and immunity. The term *secondary* is used to denote a less direct impact on CHD onset and critical cardiac events relative to the primary mediators of CHD. In general. these processes also represent a link in the causal pathway that is in closer proximity to psychosocial mediators than the primary mediators (i.e.. atherosclerosis & ventricular fibrillation). As such. these factors are frequent targets of psychological intervention...a topic that will be discussed more in the pages to follow.

Hypertension

Hypertension describes a pattern of chronic elevation in blood pressure (BP). Above normal BP is commonly recognized by readings exceeding 140 mm Hg systolic and/or 90 mm Hg diastolic (Smith & Leon. 1992). Others suggest a mean diastolic blood pressure (DBP) $\geq$100 mm Hg. regardless of evidence of tissue/organ damage. warrants clinical intervention (Irvine & Logan. 1991: Joint National Committee on Detection. Evaluation. and Treatment of High Blood Pressure. 1984: Logan. 1984). It should be noted that even mild hypertension places an individual at increase risk for CHD and acute cardiac events. and therefore should not be overlooked (Blackburn. 1987: Leon. 1987).

Hypertension is thought to impact the development and course of CHD in three general ways. First. the added force created by blood under increased pressure

hastens damage to the endothelial lining of artery walls. Specifically, this force, which is maximized at major bends and branches of the artery, creates microscopic lesions in the lining that then trap free-floating fatty substances (i.e., cholesterol). This process represents the beginning of atherosclerosis and, as has been discussed above, the road to artery occlusion or thrombus, ischemia and potentially myocardial infarction.

Second, high blood pressure inherently increases the oxygen demands of the myocardium through increased intensity and rate of ventricular contractions. This demand heightens the risk of ischemia (especially in the presence of preexisting CAD) and, in turn, ups the chance of MI or ventricular fibrillation.

Thirdly, through the above process, long-standing hypertension may result in the hypertrophy, or the enlargement of the left ventricle. Hypertrophy also increases the myocardial oxygen demand, thus fostering ischemia and related CHD events.

In sum, hypertension can contribute to the onset *and* exacerbation of two identified primary mediators of CHD...atherosclerosis and ventricular fibrillation. Blood pressure, then, is a central component of the CHD treatment and prevention puzzle.

Hypercholesterolemia

Cholesterol is a fatty substance (often originating from ingestion of animal fat) that flows within the blood stream. Lipoproteins are thought to be the carriers of cholesterol, with *low density lipoproteins* (LDL) carrying most of the body's total cholesterol, and *high density lipoproteins* (HDL) carrying the balance. Interestingly, LDL has been identified as the primary transporter of cholesterol to the endothelial lining, while HDL has been linked to resistance of atherosclerosis (Leon, 1987). Since LDL carries the vast majority of the blood's cholesterol, total cholesterol has been utilized as a marker of the relative LDL levels. Total average cholesterol level among middle-age men in the U.S. is approximately 210 mg/dl. Experts feel that ideal levels.

however. are below 200 mg/dl (or 5.2 mmol/litre) to promote reduction in CHD risk (Caroll. 1992: The Expert Panel. 1988 from Smith & Leon. 1992). Hypercholesterolemia refers to the presence of a total serum cholesterol above these recommended norms.

The primary link between cholesterol and heart disease has already been implied. for it is the very substance that accumulates on the interior arterial walls in the process known as atherosclerosis. It is known that nearly all people have some measure of plaque build-up (even men in their early twenties). but only a segment of the population (albeit a large one) will suffer significant blockage such that restricted blood flow impairs oxygenation of central mechanisms of the heart. One determining factor in the level of plaque deposition is the level of cholesterol available in the blood. Moreover. the amount of HDL in relation to LDL offers some predictive value as to the level of risk for atherosclerosis.

As will be discussed in the following section on treatment of CHD mediators. cholesterol levels may be influenced by multiple variables including those within the psychosocial domain such as stress. Therefore. serum cholesterol serves as a secondary mediating factor of CHD by serving as a contributor to the primary mediator of atherosclerosis.

Immunity

While the topic of immunity appears often in the literature of such disease states as cancer. arthritis. hepatitis. and AIDS. few authors within the field of CHD seem to include the immune system in their discussions of causal agents and mediators of the disease process. This trend is somewhat confusing since the body's ability to defend itself from pathogen and to repair its tissue seems inseparable from the body's ability to rebound from acute cardiac events. Moreover. an empirical link has been established between the psychosocial realm (i.e.. stress. depression. hostility) and immune

functioning. Therefore, while the literature does not formally posit immunity as a central factor in CAD, CHD or recovery from acute cardiac events, I would like to include it as a hypothesized mediating variable from a purely theoretical standpoint.

More specifically, it is hypothesized here that lowered immunity (resulting possibly from chronic stress) may serve as a secondary mediator of CHD in two ways: (1) by inhibiting the body's ability to heal itself (i.e., damaged myocardial tissue and/or conduction mechanisms); and (2) by increasing the risk of secondary infection/disease which might further impair healing potential and/or threaten adaptive behaviors essential to prevention/recovery (i.e., exercise, proper diet etc.).

Per the *psychoneuroimmunology* literature (Maier et al., 1994), significant injury or infection triggers a systemic immune response, or *acute phase response*, such that the battle against pathogens and restoration of tissue occurs both locally (at the sight of the injury) and systemically (involving the entire organism). This response involves many processes including: (a) the delivering of necessary *building blocks* (i.e., amino acids) to damaged tissue; (b) the production of leukocytes (white blood cells) within bone marrow to combat infection; (c) the initiation of enzymatic reactions to thwart pathogen formation resulting in fever; (d) mechanisms are activated to insure that fever remains so as to provide optimal heat for vital enzymatic responses (i.e., shivering & peripheral vasoconstriction); and (e) pain is broadcast to announce the presence and locale of the injury. As one can see even from this oversimplified model, the body requires much from itself in the face of physiological insult. Indeed, the *acute phase response* requires a great deal of *systemic energy* in order to produce and maintain the optimal environment in which healing can occur.

It has been theorized that the physiological responses in times of severe distress (i.e., fight-or-flight) may directly contradict those activated during the acute response phase (Maier et al., 1994). In short, both response sets exact great amounts of energy

to accomplish seemingly diametrically opposed ends. While one works hard to shut the body down to limit unnecessary movement, promote fever via shivering etc., and communicate pain, the other struggles to channel energy reserves to ready the muscles and brain for attack or retreat, while secreting endorphins to mask pain and maximize defense (Maier et al., 1994). The result is a body in chaos, with vital healing energy being depleted in a biochemical civil war. It is my hypothesis that a casualty of this battle may be an already weakened cardiovascular system as evidenced by poor recovery and/or recurrent cardiac events. In this way, immunity may serve as a secondary mediator of CHD and critical cardiac events.

Cardiac Pathophysiology: Biopsychosocial Factors as Mediators in a CHD Causal Pathway Model

Now that a conceptual causal pathway and underlying pathophysiology has been outlined, focus can shift to the elements of CHD of greatest import to the mental health practitioner. A handful of psychosocial factors have been strongly implicated in the literature as contributors to CAD onset and progression and key variables in acute cardiac events and subsequent recovery. Stress, Depression, Emotional Support within the Marital Relationship, Type A Personality/Behavior, and Hostility will each be presented in terms of their respective places in a causal pathway of CHD.

Stress as the Biopsychosocial Superhighway of CHD

Any discussion of the psychosocial aspects of CAD and CHD must begin with a review of the biopsychosocial model of stress. For, stress serves not only as an independent mediator of CHD, but as the central gateway through which many psychosocial factors gain access to the physiological domain. Because of its critical role in the physiological expression of so many psychological processes, the biopsychosocial model of stress (and underlying physiology) will be outlined in relative detail.

Biopsychosocial Model of Stress

While many models of stress have been formulated, an aggregate of those presented by Selye (1956, 1993) and Lazarus & Folkman (1984) is a fair representation of the biopsychosocial perspective. Selye pioneered the concept of the general *adaptation syndrome* (GAS) which is comprised of three stages: (1) the *alarm stage* in which cognitive and physiological arousal occurs; (2) the *resistance stage* in which the alarm fades and resistance to the stressor emerges; and (3) *exhaustion* ensues if the stressor continues or if a new stressor arises. Lazarus and Folkman (1984) refined Selye's model by incorporating the role of cognitive appraisal in the determination of what is deemed by the individual as a stressor. In this model, a stimulus travels through a three-part appraisal process. The *primary appraisal* involves the determination as to whether the stimulus is a threat, while a *secondary appraisal* is an evaluation as to the manageability of the threat (one's perceived ability to cope). After coping strategies have been selected and employed, an analysis of the outcome of the encounter is initiated and serves as the *third stage* of appraisal. The level of psychological and physiological arousal (stress) is a product of the perceived severity of the threat, coupled with one's sense of his/her ability to cope with the stimulus.

The Physiology of Stress

It is the physiological expression of the stress response that impacts cardiac functioning. A brief examination of this complex chain of events should serve to illuminate many of the key relationships and mechanisms by which psychological alarm is transformed into physical disease.

Initially, an *alarm phase*, or fight *or flight* response is experienced such that the body is preparing itself for attack, defense and or retreat (Cannon, 1929; Selye, 1956). To achieve this state of readiness, many biochemical processes fire in rapid succession. First, the sympathetic nervous system (SNS) diverts the physiological focus from *non-*

emergency systems to those that will be essential for immediate energy demands. sudden and sustained movement and organism survival. Specifically. stress hormones (catecholemines) are released and SNS mechanisms fire promoting increased heart rate. peripheral vasoconstriction (i.e.. limbs) upping systolic blood pressure. muscular vessels and lung bronchi dilation to increase blood flow and air intake. slowed digestion. and the conversion of energy reserves to usable fuel (Asterita. 1985a. 1985b: Caroll. 1992: Smith & Leon. 1992).

Soon. the hypothalamus releases cortiotropic releasing factor (CRF) which stimulates the pituitary gland to secrete adrenocorticotrophic (ACTH) hormone. the primary trigger of adrenal gland activation. resulting in the release of cortisol and adrenalin. Cortisol and adrenalin are central in the activation of the autonomic nervous system (ANS) and what we know as the *fight or flight* response. In the face of short-lived threats or challenges. cortisol and adrenalin secretion are wonderfully adaptive. However. the research suggests that when the stress response is activated repeatedly and for extended periods. the biochemicals that once served as life savers may actually contribute to tissue damage and diminished immunity (Kiecolt-Glaser et al.. 1986).

Stress as a Mediator of CHD

Chronic stress is thought to impact CHD development and progression in several ways including: (1) promotion of high blood pressure and heightened cardiac activity: (2) disturbance of electrical conduction fostering arrhythmia: (3) impairment of cholesterol metabolism: and (4) suppression of autoimmune functioning (Krantz et al.. 1988). Several psychosocial factors highlighted in this paper are mediators of CHD primarily because they lead to the triggering of the stress response. Accordingly. the psychophysiology of stress and CHD will be outlined in detail so that only a reference to the process will be necessary when discussing these psychosocial factors.

As we have seen. during the stress response. heart rate and intensity of

contractions increases to supply blood and oxygen to vital organs and prepare for sudden energy demands. Moreover, peripheral vasoconstriction makes this job even more taxing. The result is twofold: elevated blood pressure (BP) and increased demand for cardiac oxygen (i.e., myocardium, atria etc.). Per the atherosclerotic disease process outlined above, high BP contributes to lesions in the endothelial lining which is the basis for plaque build-up. Additionally, increased demand for oxygen may bolster the occurrence of ischemia, especially in a diseased heart and arterial system in which blood flow is already limiting oxygen supply (Bairey et al., 1990; Freeman et al., 1987; Rozanski et al. 1988). Hence, chronic stress may contribute to the onset and progression of CAD, as well as the occurrence of critical cardiac events (i.e., MI and VF) via greater risk of ischemia.

There is some evidence that stress may influence cardiac rhythm independent of ischemia. CNS activity has been implicated in the *electrophysiologic properties* of the heart such that the *threshold* for VF is decreased (Dalack & Roose, 1990; Lown et al., 1980). Simply put, consistent electrical conduction of ventricular contractions is typically not difficult for the heart to maintain even when faced with periodic arrhythmia. During times of severe stress, however, it may require far less arrhythmic activity to render the heart incapable of regulating electrical impulses such that chaos ensues. In the extreme, such chaos represents ventricular fibrillation.

Within the last twenty years, evidence has accumulated suggesting a relationship between blood lipids and stress (Bloom & Herd, 1983; Dimsdale & Herd, 1982). The reader will recall that, as part of the body's preparation for *fight or flight*, stored energy is made available for immediate and rapid use. An example of this process is the tapping of fat stores per the release of free *fatty acids* (FFA's) into the blood stream. FFA's are optimal fuel for a physically active body, however, when excursion does not occur, excess FFA's are allowed to circulate throughout the body.

Floating FFA's have been implicated in increased LDL (Bloom & Herd, 1983; Stoney et al., 1988) and impaired myocardial functioning (Smith & Leon, 1992). Therefore, stress may enhance CHD through onset and progression of atherosclerosis and/or a jeopardized myocardium.

Finally, a theoretical case has been formulated in a previous section for the role of weakened immunity in CHD and recovery from critical cardiac events. If valid, yet another link between stress and heart disease may exist. The association between stress and immunity is not new, for the area of *stress-induced immunosuppression* has been discussed by numerous authors for over 30 years (Daruna & Morgan, 1990; Maier et al., 1994; Stanford & Salmon, 1993). In fact, many different types of stressors, ranging from marital disruption (Kiecolt-Glaser et al., 1987) to unemployment (Arnetz et al., 1987), have been shown to suppress some aspect of immunity. Specific components of immune function thought to be negatively influenced by stress include a reduced number and activity level of T-helper lymphocytes and NK cells (Kiecolt-Glaser et al., 1987). Hence, stress may serve as a mediating factor of CHD through its destructive impact on immunity.

In sum, stress is a widely researched biopsychosocial phenomena that contributes to CAD and CHD onset and progression in many ways. To date, the evidence seems clear that the stress response is, indeed, a superhighway connecting the psychosocial and physiological realms. As we will see, many other psychosocial constructs rely on this pathway to adversely impact cardiovascular functioning.

Depression as a Mediator of CHD

Anecdotal evidence has existed for some time that depression is a common correlate of CHD, especially post-MI. Since the early 1970's, empirical support for the relationship between acute cardiac illness and depressive symptomatology has been mounting. Vazquez-Barquero et al. (1985) found in a sample of patients with ischemic

heart disease a 20% incidence of *depressive neuroses*. In an earlier study, Kavanagh et al. (1975) in a group of patients assessed at 18 months post-MI, 33% were classified as suffering *severe depression*. More recently, Schleifer et al. (1989) found 18% of 283 patients met criteria for major depression days after MI. In a follow-up, 44% of those patients initially diagnosed still met criteria for major depression. Finally, a study by Carney et al. (1988) indicated that 18% of their sample of patients with CAD met DSM-III criteria for current major depressive episode. In marked contrast, numerous studies have placed the rate of depression among the general adult population at 2-4% (DIS: Robins, Helzer, Croughan, & Ratcliff, 1981; *DSM-IV*: APA, 1994). This discrepency suggests that heart patients suffer depression at a rate 2 to 5 times greater than the general population.

In light of the growing evidence that depression is an all too common factor in CHD, research has sought to answer two burning questions: (1) What is responsible for the high incidence of depression in CHD patients? and (2) How does depression impact the progression of CHD or cardiac rehabilitation? While the former is intriguing, the latter is of greatest interest for the scope of this paper because it addresses depression as a mediating factor in CHD and recovery. The research concerning the link between depression and CHD follows.

Depression has been shown to negatively impact the CHD patient in several ways. For example, depressive symptomatology has been linked to poor treatment compliance with medical therapy (Blumenthal et al., 1982). In a second, but related finding, Finnegan and Suler (1985) found that depression seemed to interfere with adherence to healthy behavioral changes essential to an effective recovery (i.e., exercise, diet, not smoking). In the recovery period, depression has been identified as a predictor of recurrent cardiac events and cardiac-related mortality (Carney et al., 1988; Carney et al., 1993; Frasure-Smith et al., 1993, 1995; Kennedy et al., 1987). What

this data means is largely speculation at this stage. One possible explanation is that the depressed patient is simply less willing and able to manage daily needs due, in part, to increased pessimism concerning their prognosis, lethargy, and reduced sense that they can cope with the demands of their illness. Whatever the underlying reasons, it would appear that depression may well impact the CHD process on a biobehavioral level through poor treatment compliance and the hindrance of pro-recovery lifestyle changes.

Depression and Ventricular Fibrillation

Depression may be capable of influencing CHD through a more direct pathway. Data and theory have converged to suggest that activity within the central nervous system (CNS) may interfere with the electrophysiologic mechanisms of the heart. More specifically, affective states (like depression) which influence CNS activity, may lower the threshold for ventricular fibrillation to occur (Lown & Verrier, 1976). Animal studies support a relationship between psychological stress and ventricular fibrillation (Lown et al., 1980). Empirical research is just beginning to isolate which affective states may contribute to cardiac arrhythmia and ventricular fibrillation. Early work has focused on the link between parasympathetic nervous system activity and heart rate variability, a central variable in ventricular fibrillation.

The first phase of the theoretical and empirical foundation for the exploration into the proposed association between depression and cardiac functioning is underway. While much work is left to be done to identify the specific agents in the causal link, it seems clear from the literature that depression mediates CHD and post MI rehabilitation on more than one level.

The Marital Relationship as a Mediator of CHD

The late 1960's and early 1970's saw the first legitimate interest in the marital relationship as a factor linked to CHD and MI. From this early work, the marriage was deemed a central factor due to its multifaceted impact on CHD. Specifically, a marriage

can engender an array of known CHD risk factors at any given time including that of environmental stressor, destroyer of emotional support, and source of hostility (Medalie & Goldbourt, 1976). In contrast, a stable marriage is thought to offer *emotional support* through which many advantages have been linked such as bolstered immunity, positive cardiac reactivity, and facilitated rehabilitation (Brown & Smith, 1992; Chandra et al., 1983; Medalie & Goldbourt, 1976; Ross et al., 1990; Smith & Leon, 1992).

Therefore, the marital relationship, while certainly a psychosocial mediator of CHD, might best be described as a complex psychosocial *context* comprised of multiple CHD mediators within the causal pathway (Appendix D, Fig. 1). As the most noteworthy of these links, both *emotional support* and *hostility*, within the context of marriage, deserve special attention.

Cardiac Benefits of Emotional Support within the Marriage

Preliminary data indicates that the marital relationship is a critical component in not only the development of CHD, but a successful recovery post MI as well (Beach et al, 1992; Case et al., 1992; Chandra et al., 1983). A handful of studies suggest that the mere presence of a spouse can be of some benefit to the recovering CHD patient. Investigations into this question have steadily increased in recent years (Case et al., 1992; Chandra et al., 1983; Coombs, 1991). The most recent and noteworthy work suggests that single men experience "higher levels of depression, anxiety, and other forms of psychological distress" as well as increased incidence of "acute conditions, chronic conditions, days of disability, and lower perceived health status" (Jones, 1992, pg. 152). Similarly, a single patient is far more likely to experience subsequent cardiac complications than his married counterpart (Case et al., 1992; Chandra et al., 1983; King et al., 1993; Waltz, 1986). Once again, the central ingredient thought to account for an enhanced resilience to illness is the *emotional support* provided by a sound

marital relationship (Coombs. 1991: Ross et al.. 1990).

One popular explanation for this marriage-disease connection is the *protection/support hypothesis* (Ross et al.. 1990). This position argues that a marriage full of conflict can contribute to the onset of illness. while an *emotionally satisfying* marriage seems to *buffer* the individual from illness (Jones. 1992). Specifically. if the marital relationship offers quality emotional benefits such as intimacy. positive affective exchange. esteem. communication and equity. the person is believed to be less vulnerable to disease (Ross et al.. 1990). Such emotional advantages are thought to lower physiological stress and have direct. positive impact on cardiac reactivity including lowered blood pressure. reduced blood levels of norepinephrine (and related biochemicals) and associated physiological changes that elevate risk of ventricular constriction. arrhythmias and coronary artery thrombus (Kohn. et al.. 1983: Smith & Leon. 1992).

Along with the beneficial physiological effects. an emotionally supportive marital relationship is thought to boost a host of psychological variables such as self-esteem. sense of well-being and positive affect. These psychological factors have been linked to reduced risk of physiopathology. psychopathology. as well as enhanced treatment compliance (King et al.. 1993: Syme & Seeman. 1983; Waltz. 1986: Wills. 1991). It is argued that these positive benefits foster improved self-care and better personal health habits as well.

To summarize. the emotional support provided by a sound marital relationship is believed to have significant implications on both physiological and psychological factors associated with CHD development and progression...giving further testimony to the role of the marital relationship as a mediator of CHD (Kulik & Mahler. 1989).

Hostility in the Marriage as a CHD Mediator

Some evidence suggests. that being married may not be enough to buffer

against disease. Jones and Jones (1988) found that, while a positive marriage may have health benefits, remaining in a conflictual marriage is potentially more damaging than living alone (Ross et al., 1990). As has been discussed above, hostility is believed to be extraordinarily toxic with respect to cardiac functioning. Within a marital context, it may be doubly so, for it may foster both negative cardiac reactivity and the removal of vital emotional support (Sanders et al., 1991)

Following a critical cardiac event, the CHD patient and spouse are likely to experience increased stress, especially when new roles and care giving/receiving tasks are required (Rankin, 1992). Coupled with an already distressed marital relationship, levels of overt and covert hostility might be expected to rise. The physiological mechanisms triggered by this upsurge in hostility may be especially damaging to an already weakened cardiac system...potentially increasing the chance for critical cardiac events such as arrhythmia (and VF) and myocardial infarction. As a viable correlate of the marital relationship, marital hostility is thought to be yet another component in the mediation of CHD and post MI recovery.

The Spouse as a Mediator of CHD & Post MI Recovery

Spousal participation is, in most cases, critical to a successful rehabilitation effort on two levels: (1) in the physiological realm via promotion of positive cardiac reactivity; and (2) in the psychosocial/biobehavioral realm via practical assistance with the daily tasks of rehabilitation (Beach et al., 1992; Davidson, 1987; Smith & Leon, 1992; Thompson & Cordle, 1988). However, it appears that during acute phases of CHD or following a critical cardiac event, the spouse is rendered less capable of assisting the patient on both of these levels as a result of the extreme (and abrupt) emotional and physical demands placed on him/her (Coyne & Smith, 1991; Stern & Pascale, 1979; Waltz et al., 1988). The spouse often must take on the multiple roles of both parents, head-of-household, dietitian, nurse, physical therapist, coach, confidant

and liaison between physician and patient (Coyne & Fiske, 1992: Hilgenberg & Crowley, 1985). The literature indicates that depression, anxiety and stress-related symptoms are common among wives of male MI patients, and, if severe, may impair her ability to assist in recovery (Mayou, et al., 1978: Nyamathi et. al., 1992: Shanfield, 1990).

If acute CHD can exact a toll on even the most resilient and dedicated of spouses, how might the spouse who is already emotionally and physically compromised due to premorbid marital distress fare? Logic would suggest that he/she is at far greater risk of being unable and/or unwilling to provide the emotional and practical support essential to a successful recovery. In addition, the threat of psychopathology and/or physical illness may be enhanced as well. The end result might be a lack of emotional intimacy and supportive interaction. A heightened concern that critical tasks such as the preparation of special foods, careful administration of medications, assistance with daily necessities (i.e. personal hygiene) are less likely to garner spousal participation when the marriage is distressed.

In short, premorbid marital stress in concert with the post MI situation is likely to contribute to depletion of emotional and physical reserves of the spouse, thus compromising the rehabilitation effort. In this way, the spouse may truly serve as a mediator of CHD, especially regarding recurrent cardiac events. Further research is necessary to clarify the mechanisms underlying this proposed linkage.

Type A Behavior Pattern as a Psychosocial Mediator of CHD

When one purveys the CHD literature, it is not long before the topic of Type A personality and/or behaviors is broached. Indeed, Type A is one of the most researched aspects of heart disease to date, especially by those in the mental health professions.

Type A Behavior Pattern (TABP) was first put forth by Friedman and

Rosenman in 1959, and has been defined as "an action-emotion complex that can be observed in any person who is aggressively involved in a chronic, incessant, struggle to achieve more and more in less and less time" (Friedman & Rosenman, 1974, p. 68). Additional characteristics of the pattern include competitiveness, aggressiveness, drive for recognition, cynicism, hostility, exaggerated response to stressors, animated speech and non-verbal communication, restless, impatient, perfectionistic and demanding of self and others (Price, 1988). While numerous studies address the relationship between TABP and CHD, the results have been mixed. However, some findings have been less ambiguous. For example, a panel for the American Heart Association concluded that *healthy* persons exhibiting TABP were at twice the risk for developing CHD than Type B persons, or those not manifesting such traits (Cooper, Detre, & Weiss, 1981). In all, TABP seems a likely contributor to CHD (Booth-Kewley & Friedman, 1987; Dembroski & Costa, 1987; Matthews, 1988).

The primary vehicle linking TABP and CHD is an exaggerated and persistent *stress response* (see previous section). Many of the elements inherent to TABP are thought to contribute to consistently stressful interactions and internal appraisals. Specifically, the byproduct of always pushing, striving, competing, demanding etc. is psychological and physical arousal stemming from omnipotent appraisals of threat (i.e., approaching deadlines, imperfect work, distrust of others etc.) and strained interpersonal relations. Therefore, the Type A individual's *inter* and *intra*personal worlds are both typically stress-filled. Hence, all facets of the stress response implicated in CAD and CHD may well apply to TABP. On a physiologic level, Type A subjects as compared to non-Type A (or Type B) subjects when exposed to potentially stressful stimuli, have been found to possess higher 24 hour testosterone levels, elevated norepinephrine levels and higher blood pressure and heart rate (Harbin, 1989). A discussion of the psychological underpinnings of TABP will be presented

when psychosocial interventions for Type A are outlined. Suffice it to say, TABP serves as a mediator of CHD, it would seem, primarily by fostering an exaggerated and persistent stress response.

Hostility as a Mediator of CHD

More recently, efforts have been made to delineate the sub-components of TABP so as to isolate the more *toxic* aspects of the construct (Dembroski et al., 1989; Hardy & Smith, 1988; Matthews 1988; Smith, 1989). Results from this work suggest that hostility and anger may be the most potent of the TABP antagonists in terms of cardiac functioning. A host of authors have explored the impact of hostility on physiological measures related to CHD. Cook and Medley Hostility Scale scores were found to positively correlate with high blood pressure in the context of stressful interactions (Smith & Allred, 1989). Additionally, hostility has been found to be associated with increased lipid levels and overall physiological reactivity (Lundberg et al., 1989 as cited in Smith & Leon, 1992), as well as cortisol secretion (Pope & Smith, 1991).

Therefore, hostility seems to access many of the same stress-induced biological mechanisms as TABP in general, but evidence indicates that it may do so far more effectively. In addition, a world-view shaped by hostility may tend to further the stress response cycle by: (a) fostering strained interpersonal relations and therefore, elicit future conflict; and (b) promoting the perception that others will be antagonistic requiring a constant state of defensiveness and mistrust (Smith & Leon, 1992).

While further research is necessary to refine the elements of TABP responsible for negative cardiac reactivity, existing literature supports the link between hostility and the physiological mediators of CHD.

From Theory to Practice:

Psychosocial Interventions Targeting Mediators of CHD

As the above causal pathway illustrates, there are a number of primary, secondary and tertiary (psychosocial) mediators of CHD. Psychosocial treatment strategies have emerged over the years targeting many of these factors in the name of CHD prevention and rehabilitation. The psychologically-based treatments more commonly cited in the literature will be outlined. The reader will notice that, as stress is common to many of the mediators of CHD, stress management strategies are employed to address a host of issues including hypertension, Type A, chronic stress, hostility (anger) and a multitude of other stress-related aspects of CHD.

Interventions Targeting Marital Distress

A review of marital therapy (models, assessment and outcome research) is well beyond the scope of this paper. However, since the marital relationship has been implicated as a potential mediator of CHD and rehabilitation, the inclusion of marital therapy in a discussion of the psychosocial treatment of the cardiac patient is necessary. A brief review of key principles of couples counseling will be offered, followed by a synopsis of outcome research in the area. It is hoped that this review will shed light on the link between the psychosocial treatment of martial distress and the reduction of mediating factors of CHD and recurrent cardiac events (i.e., hostility, depression, and lack of emotional support). Moreover, it should serve to justify the selection of marital therapy assessment and intervention strategies for the proposed model that follows.

Assessment of Marital Distress

Assessment of marital dysfunction can take many forms, from the behavioral interview to sophisticated objective measures. For our purposes, a quick glance at the mostly widely used objective measures will suffice, for they have both clinical and research value.

The *Dyadic Adjustment Scale* (DAS: Spanier. 1976) is a very popular measure for both clinical and research purposes as will be explained in more detail in Chapter III (Methodology). This 32 item self-report instrument is geared to measure four dimensions of a couple's relationship (married or cohabiting) *dyadic satisfaction. dyadic cohesion. dyadic consensus. and affectional expression* (Jacobson & Margolin. 1979. p. 74). Spanier designed the DAS such that each item discriminates between happily married and divorced samples. Similarly. the DAS has been shown to possess strong reliability and validity. The Global Distress Scale (GDS) of the Marital Satisfaction Inventory (MSI: Snyder. 1981) has been utilized in research. as well as serving as a useful clinical indices of global marital distress.

The GDS comprised of 43 true/false items and has internal consistency and test-retest coefficients of .97 and .92 respectively. Per its creator. Snyder et al. (1989). *the GDS has been shown to discriminate successfully between distressed and non distressed couples and to correlate significantly with clinician's ratings of couple's overall dissatisfaction with their marriage. chronicity of marital difficulties. deficits in problem resolution. perceived emotional distance from spouse. pessimism regarding the future of the marriage and likelihood of separation or divorce* (Snyder & Wills. 1989. pg. 40: Snyder. 1983. 1981). One major criticisms of the MSI is its length.

Finally. the *Marital Adjustment Scale* (MAS: Locke & Wallace. 1959) is a widely used measure of global marital satisfaction. Several features of the MAS may account for its popularity. First. it is brief. only 15 items. Second. reliability and validity analyses have demonstrated the MAS to be internally consistent and able to discriminate between distressed and non distressed couples. One criticism of the MAS is its apparent correlation with social desirability measures suggesting that high satisfaction scorers may be influenced by wishing to look good (Murstein & Beck. 1972).

In terms of assessing marital distress for purposes of determining need for services in a cardiac intervention program, the DAS, MSI and MAS appear to be adequate choices.

Models of Marital Therapy for a Cardiac Care Program

Countless models and versions of marital and couples therapy exist, and a great many have been found effective in producing positive change. However, the purpose here is evaluate only those that seem appropriate for a short-term cardiac care program designed to address key mediators of CHD and recurrent cardiac events quickly, effectively. Four excellent candidates emerge from the literature: (1) Behavioral Marital Therapy (BMT: Jacobson & Margolin, 1979); (2) Cognitive Marital Therapy (CMT: Waring, 1988); (3) Emotion Focused Marital Therapy (EFT: Greenberg & Johnson, 1988); and (4) Insight-Oriented Marital Therapy (IOMT: Snyder & Wills, 1989).

While each of the above models has proven effective in reducing marital discord (see Alexander et al., 1994), it seems that BMT is a sound choice for use in a short-term cardiac intervention. A primary reason for this selection is BMT's emphasis on immediate positive change. BMT is designed to elicit positive expectancies, pleasant affective interaction and the beginnings of a *collaborative set* within the very first session (through reminiscence). In cases of CHD, especially post MI, these changes might foster positive cardiac reactivity and spark improved emotional/social support, not six months down the line, but today. For couples facing the burdens of chronic and acute illness, time is of the essence, for stress is typically quite high. This is not to say that marital therapies that require more than 8 to 10 sessions are inappropriate for CHD patients. Rather, that a CHD patient within an acute phase of his/her illness (especially following a critical cardiac event) does not have the time or energy to invest in longer-term therapy that may involve an initial increase in distress as part of the

healing process (as many models do). If assessment determines the presence of marital distress as well, both patient *and* spouse need noticeable relief quickly.

In this vein, BMT requires positive *behavioral exchange* via homework assignments beginning after the first session. This may have three beneficial affects. First, the research indicates that the exchanging of positive behaviors (i.e., giving a back rub, cleaning the kitchen etc.) promotes a collaborative set and increases positive interactions between the couple. Once again, this might translate into positive reactivity and increased emotional support.

Second, an acute CHD patient is often temporarily or permanently stripped of many of his/her responsibilities and life roles (i.e., absence from work, inability to perform husband/wife-mom/dad duties)..even to the extent of suffering the indignity of being cast into role of invalid. By giving the patient tangible responsibility for his/her part of therapy, the helpless *sick role* may be thwarted in a small way.

Third, behavioral exchange forces each spouse out of their often embittered egocentric frame of reference. This process might help the patient individuate from the disease (which has become the center of his/her life), allowing him/her to begin to see the world, not as a heart attack victim, but as a spouse or lover again. In the same way, the spouse, who has been thrust into the role of cardiac nurse, may individuate from the illness as well by getting a taste of what it is like to be an equal partner again.

Third, BMT is concrete. A couple experiencing both marital distress and an acute illness are not in a good position to deal with the ambiguity and/or delayed gratification of insight exploration. Having identifiable tasks within a very simplistic theoretical framework, accompanied by strong, directive, yet supportive guidance from their *coach* (ala Jacobson & Margolin's model), the stressed couple may be more willing and able to comply.

A final point deserves mention. As has been discussed previously, depression

is a common correlate of MI. and can contribute to CHD. and impede cardiac recovery. Marital therapy (and BMT in particular) has been shown to be an effective treatment for depression in distressed couples (Jacobson. et al.. 1991). Therefore. BMT may simultaneously address more than one known CHD risk factor making it a powerful and cost effective tool for prevention of initial or recurrent cardiac events.

To summarize. any of the four models for marital therapy may be effective as part of a short-term cardiac care program. However due to its simplicity. emphasis on immediate change and action orientation. BMT seems an appropriate intervention for the CHD patient and spouse experiencing marital distress.

Interventions Targeting Depression

As we have seen. depression has been implicated in the literature as a potential mediator of CHD and cardiac rehabilitation. Any discussion of a comprehensive program designed to moderate such factors must. then. address depression and its treatment.. There are hundreds. if not thousands. of psychological and pharmacological treatments for depression. and it is beyond the scope of this project to offer even a limited review of the topic. The focus of this section. rather. is the delineation of assessment and treatment strategies for depression that are appropriate for inclusion in a short-term cardiac intervention program.

Assessment of Depression

While numerous measures exist for the assessment of depression. the *Inventory for Diagnosing Depression* (IDD: Zimmerman et al.. 1986) and the *Beck Depression Inventory* (BDI: Beck et al.. 1961) seem well suited for a short-term treatment situation.

The IDD is a 23 item self-report scale designed to assess depression per DSM-IV criteria (American Psychiatric Association. APA. 1995). While a relatively new scale. the IDD has garnered much acclaim as a reliable and valid measure of depression

that is quick. easy to administer and cost-effective. features that make an excellent candidate for inclusion in an intervention model for CHD. See Chapter III for a more detailed description of the IDD.

The BDI is a 21 item. self-administered inventory that has proven to be a widely accepted measure of depressive symptomatology and that has demonstrated solid internal reliability. test-retest reliability. as well as concurrent and construct validity. The measure's items tap several aspects of the syndrome of depression (i.e.. mood. somatic complaints and symptoms over the past week) utilizing a Likert-type scale of 0 to 3 corresponding to statements reflecting varying degrees of a given depressive symptom. Scores can range from 0 to 63. While the cutoff scores for the various degrees of depression differ with use of the instrument. widely accepted ranges are 0-9 normal: 10-15 mild: 16-19 mild-moderate: 20-29 moderate-severe: and 30-63 severe depression.

The BDI's sound test-retest reliability allows for its repeated (even weekly) administration. and makes it an appropriate measure for tracking short-term relief of depressive symptomatology. Additionally. the Beck is quick. simple and cost effective to administer and score. further adding to its appeal.

Treatment of Depression within a Short-Term Cardiac Care Program

In choosing a treatment model for depression for use within a short-term cardiac care program. four major criteria were used. One. the model must have empirical support for its efficacy as a treatment modality for depression. Two. it must lend itself to application within a short-term treatment context. Three. the model must be theoretically and clinically consistent with other techniques being utilized for other aspects of cardiac care (i.e.. stress management) so as not to confuse a patient with opposing strategies and to allow integration of treatment goals (i.e.. blending work on TABP cognitions with those underlying depression). And. four. the model must be

adaptable to either a group or individual counseling format.

Cognitive therapy for depression (CT: Beck et al., 1979) seems to meet all of the above criteria, and, therefore, will be considered a strong candidate for inclusion in a short-term cardiac care program. The therapy is based upon Beck's theory of depression which posits a negatively distorted view of self, the world and the future as core features of a depressed individual (Beck et al., 1979). It is thought that these views stem from depressive *schemata* which are firmly rooted belief patterns that skew thoughts in a negative, self-slighting and depressive manner. The development of skills for systematic monitoring (tracking) and correction (replacement) of these thoughts is believed to be the curative process in CT. A key technique employed in CT is for the client to become a *scientist* who is charged with *testing* his/her depressive thoughts (as if they were *hypotheses* about self) by seeking evidence for their validity within the environment. When little supporting *data* is found for their distorted world and self view, depressive symptomatology is said to diminish.

CT has been found effective in many empirical outcome studies (Bowers, 1990; Dobson, 1989; Thase et al., 1991). Specifically, CT was found to be more effective than no treatment or nonspecific treatment groups, and as effective as pharmacological interventions (Dobson, 1989). Length of treatment ranged form 10 to 20 sessions, supporting CT's efficacy as a short-term intervention. Interestingly, research indicates that CT may guard against relapse more effectively than medication (Evans et al., 1992).

As has been discussed, treatments of Type A, hypertension, stress, hostility and marital distress all incorporate cognitive techniques to achieve symptom relief. As a result, CT may allow for the testing/correction of Type A or other stress-inducing cognitions in conjunction with those that underlie depression. Hence, CT may prove to be a sound framework within which to attack a host of CHD mediating factors.

In sum. it appears that CT is an effective treatment for depression that lends itself to short-term intervention. Moreover, it seems quite consistent with those strategies recommended for use in treating other known CHD mediating factors. thus its inclusion would foster an integrated cardiac care program.

Interventions Targeting Hypertension

An estimated *60 million Americans are at increased health risk due to elevated blood pressure* (Eisenberg. 1993. p. 964: Stokes et al.. 1989). As has been previously established. hypertension is widely held as a central mediator in heart disease and a host of vascular disorders including stroke. It is not surprising. then. that a great many medical (Collins et al.. 1990) and psychosocial (Weiss. 1984) treatment strategies have targeted hypertension in the war on CHD (Caroll. 1984. 1992). While pharmacological treatments have proven somewhat effective in lowering blood pressure (Collins et al.. 1990) and reducing cardiac mortality (Leon. 1991: Smith & Leon. 1992). antihypertensive agents are known to carry significant side effects that hinder compliance and applicability (Curb et al.. 1985). As a potential result. increasing attention has been allotted to psychosocial treatments of mildly and severely elevated blood pressure in the past two decades. Moreover. the implication of psychological factors (such as chronic stress) in the etiology of hypertension may have furthered the interest in psychosocial interventions.

Types of Hypertension Treatment

The literature suggests that three broad categories of nondrug treatments of hypertension are in use. often concurrently: stress management (Zurawaski et al.. 1987). aerobic exercise. and dietary alterations (Blanchard. 1994). A fourth category. frequently termed *cardiac* or *cardiologic counseling*. has been suggested by others in the literature as focusing on treatment regimen (i.e.. diet. exercise. medication) compliance (Friedman et al.. 1986: Joint National Committee on Detection. Evaluation.

and Treatment of High Blood Pressure. 1988). Our focus. however. will be on the stress management interventions for hypertension reduction.

Stress management strategies are variable. but typically incorporate elements of relaxation training (progressive muscle. imagery and breathing). meditation. biofeedback and cognitive restructuring (Blanchard. 1994: Caroll. 1992: Johnston. 1985: Smith & Leon. 1992). Such programs are utilized in both individual and group treatment contexts in both the prevention and rehabilitation of critical cardiac events. A typical non-pharmacologic treatment strategy for hypertension will spend all or part of the initial sessions (1 or 2) on rapport building. supportive therapy and the dispensing of information concerning the role of stress in the etiology and course of hypertension (Bennett et al.. 1991: Patel. 1975). Information is regarded as an important element in the level of commitment and compliance to stress management training. and should not be overlooked or glossed over by simply offering handouts or booklets. In fact. the clinician can use the information phase of treatment to foster discussion and sharing to build rapport with the client and cohesion among peers if a group format is utilized. Health psychology professionals have long endorsed the stance that an individual's knowledge of his/her illness can foster better understanding and a sense of control which may translate into enhanced treatment compliance and attitude.

A second common phase of stress management treatment is training in breathing exercises. followed by either imagery or progressive muscle relaxation instruction. Beliefs and thoughts underlying and predisposing chronic stress can be dealt with in many cognitive-behavioral traditions. however Meichenbaum's Stress Inoculation Training (SIT) targeting stress-inducing self-talk is considered prototype of the C/B models (1985).

Stress management strategies for treatment of hypertension have met with mixed reviews. In a review of the literature. Blanchard et al. (1988) found stress

management techniques to offer significant reductions in blood pressure even at six month follow-up. Jacob (et al., 1991) via a meta-analytic study, argued that stress management/relaxation studies have not demonstrated a degree of efficacy warranting the wide acclaim the treatment model has received. Nevertheless, stress management techniques are widely used in the treatment of hypertension, a powerful secondary mediator of CHD.

Interventions Targeting Type A Behavior Pattern

Findings in the mid 1960's and 70's that a specific pattern of behaviors independently predicted CHD stirred a furious exploration into the phenomenon that remains to this day (Western Collaborative Group Study, Rosenman et al., 1975). Due to the immense popularity of Type A (TABP) as a proposed link to CAD and CHD, interventions geared to tame the personality/behavior pattern are numerous. While these strategies vary in their specifics, most recognize the role of cognition, physiology, behavior and the social context (i.e., a biopsychosocial basis) in the development and expression of TABP (Price, 1988). Psychosocial assessment and treatment, then, should reflect these dimensions. The following section outlines the assessment and treatment of TABP accordingly.

Assessment of TABP

TABP is not universal in its expression or influence on cardiac reactivity, for it differs as a function of the individual and the context. Therefore, the clinician cannot assume that the presence of a few of the known correlates of TABP constitutes a true pattern. Similarly, a standard treatment plan cannot and should not be applied to every client who seems uptight or competitive. Hence, accurate and valid assessment of the various dimensions of TABP is regarded as critical to effective Type A counseling (Nunes, et al., 1987; Price, 1988).

Three measures have been acknowledged as the more validated of the TABP

assessment tools (Matthews. 1988: Price. 1988): the Friedman and Rosenman Structured Interview (SI: Friedman and Rosenman. 1964: Rosenman. 1978): the Jenkins Activity Survey (JAS: Jenkins et al.. 1974): and the Framingham Type A Scale (FTAS: Haynes. Feinleib. & Kannel. 1980). The SI is a series of interview questions about a client's typical pattern of responding when faced with stressful situations. Both the JAS and FTAS are self-report measures designed to tap tendencies toward competitiveness. impatience and need for achievement. Of the three. only the SI has been shown a consistently strong correlate of CHD. However. since the SI relies greatly upon the interpretation of in-interview behaviors (i.e.. speech & motor activity). it has been criticized for being difficult to score and for allowing a high degree of scorer subjectivity (Price. 1988).

Treatment of TABP

Many types of psychosocial interventions geared toward the reduction of Type A tendencies have been formulated in the last 30 years. Most of these have focused on coping with the deadly byproduct of TABP...stress (Price. 1988). For a review of Type A counseling models. the reader is directed to Price (1988) or Thoreson and Powell (1992).

Psychoeducation focusing on risk factors. prevention and the links between CHD and TABP is a popular tactic employed by physicians. nurses and mental health professionals. Much of education's appeal may stem from its cost effectiveness and that it lends itself to most any setting including the physician's office or hospital room.

Stress management and relaxation training (i.e.. progressive muscle relaxation. imagery. breathing. biofeedback and yoga) are frequently incorporated to address the psychophysiology of TABP. It is often a goal to have the client practice the techniques thoroughly in session such that they can be applied in the client's real world environment. In this way. breathing. imagery and/or muscle relaxation can be done at

home or at the office when TABP is likely to stir a more intense and enduring stress response (Price. 1988).

Cognitive therapies to address underlying Type A cognitions (i.e.. "I must get this done more quickly") have gained momentum within the mental health field. They range from Ellis's RET (Ellis. 1962) to Meichenbaum's SIT (1985). The central objective of cognitive treatments of TABP is to challenge existing beliefs and recurrent thoughts that contribute to appraisals of threat. feelings of time urgency and a competitively hostile view of the world and those in it. As Price (1988) and others have pointed out. the majority of psychological interventions in research and practice have focused on the aftermath of TABP (i.e.. stress). rather on the underlying mechanisms which trigger the potentially lethal physiological reactivity (Meichenbaum. 1985: Meichenbaum & Fitzpatrick. 1993). It is important to note that cognitive therapy may involve behavioral techniques (i.e.. activity scheduling. activity/emotion tracking etc.) to reinforce counseling objectives (Blanchard. 1994: Haaga. 1987).

Behavior Therapy targeting Type B skill building (i.e.. assertiveness. slowing down. conflict resolution and coping with environmental demands) is a practical. yet powerful TABP treatment strategy. Assertiveness training is an intriguing component of behavioral strategies. The stereotypical Type A person (i.e.. the hard working. driven executive dashing around the office exuding an air of confidence and purpose). is not usually thought of as lacking in assertiveness. Ironically. core features of Type A are a need for acceptance and a distaste for criticism which often translate into the inability to say "no" (especially to peers and superiors) and/or to reveal their true opinions for fear of conflict or rejection (Price. 1988). These tendencies are thought to fuel resentment and hostility for others and. in turn. promote negative cardiac reactivity. Hence. teaching the skills of polite and respectful assertiveness may allow the Type A to appropriately limit his/her commitments and reduce real or perceived interpersonal

conflicts. Additionally, behavioral strategies such as time management, prioritization, activity scheduling, diet and exercise management, social skills training and conflict resolution training may assist the Type A client with managing their environment more effectively and reducing anger, hostility and overall stress (Hazaleus & Deffenbacher, 1986; Price, 1988).

Psychodynamic therapies aimed at resolving unconscious motives, strivings and conflicts, which are thought by this camp as epicenter of TABP, are not uncommon. While it is thought that a great deal of psychodynamic therapy for Type A related issues is being done in this country, its efficacy is less documented due to the lack of empirical examination of the model (Blanchard, 1994; Price, 1988).

It should be noted that little is said in the literature in reference to group versus individual counseling in the treatment of TABP. Certainly all of the above models could be adapted to either situation depending on setting, resources and clientele. Limited resources and demand for economy from third-party payers may enhance the use of the group. Anecdotal evidence and theory suggests that many of the dynamics of a good group experience (i.e., cohesion, social/emotional support, common experience, feedback etc.) may fit the needs of the CHD or post MI patient. As we will see in the discussion of treatment efficacy, the group format utilizing nearly all of the above models of treatment appears frequently in research methodology. Nevertheless, little discussion of the role of the group in recovery was found.

Regardless of modality, the central goal of Type A counseling is to counter the destructive force of the chronic and exaggerated stress response elicited by TABP. To most effectively accomplish this objective, all four dimensions of TABP (cognitions, physiology, behavior and the environment) should be addressed (Price, 1988).

Type A Outcome Research

Can the Type A pattern be changed? Many authors have asked this question

since the inception of the concept in the 50's. The data from several sources suggests that, indeed, TABP can be effectively modified, but a multidimensional strategy may be the best overall strategy (Haaga, 1987; Nune et al., 1987; Price, 1988; Thoreson & Powell, 1992).

The study regarded by many as the most comprehensive and important in the area of TABP treatment efficacy is the Recurrent Coronary Prevention Project (RCPP; Friedman, et al., 1986). In this study, nearly 900 post MI patients were randomly placed into (1) group cardiologic counseling targeting treatment compliance to dietary, exercise, and drug regimens (involving education & physician consultation); and (2) cardiologic counseling plus TABP counseling (comprised of relaxation training, cognitive restructuring, self-management and self-monitoring training). While the study is ongoing, at three years the TABP counseling group had: (a) reduced self and observer-rated Type A behaviors by one standard deviation; and (b) experienced significantly fewer nonfatal MI's and cardiac deaths.

Additional studies have been noted by Thoreson and Powell (1992) having similar results (Gill et al., 1985; Levenkron et al., 1983; Roskies et al., 1986). These three designs employed multicomponent TABP treatments with a strong cognitive-behavioral slant, and each resulted in significant reduction in Type A related behaviors.

Nunes et al. (1987) performed a meta-analysis of the leading studies examining efficacy of various TABP models and found a standardized effect size of .61, or just over one half of a standard deviation. The authors concluded that TABP was open to change, but only if multiple procedures (i.e., stress management, relaxation, education, cognitive restructuring and behavioral training) were utilized in concert. While it was noted that all treatments were performed within the group format, the authors felt additional research was necessary to differentiate efficacy of the group versus individual format.

In sum, it would seem that TABP is amenable to change, but a multidimensional attack is advisable. Hence, the literature seems to support an interactive biopsychosocial model of Type A, both in its etiology and its treatment.

Interventions Targeting Hostility

As mentioned above, hostility is thought to be the most *toxic* ingredient in Type A tendencies due to its impact on cardiac reactivity. The literature often includes anger as a correlate (and synonym in some cases) of hostility. Together, they may account for the progression of CAD and CHD, as well as contributing to recurrent cardiac events (Dembroski & Costa, 1987; Matthews, 1988). Accordingly, the reduction of anger and hostility among CHD patients has garnered much empirical and clinical interest in recent decades (the reader is directed to Biaggio, 1987).

The assessment of hostility has been a topic of some debate, for as more becomes known about the phenomenon, the more complex an issue it becomes. For example, is hostility one element, or a construct comprised of multiple varieties and components? Future research is needed to clarify this question. At present, the most notable tool for assessing hostility is the Cook and Medley Hostility (Ho) Scale (Cook & Medley,1954). Derived from the Minnesota Multiphasic Personality Inventory (MMPI), the Ho scale is believed to tap *anger proneness, resentment, cynicism, and mistrust of others* (Smith & Frohm, 1985 as cited in Smith & Leon, 1992, p. 64; Smith et al., 1990). Research has shown the Ho Scale to predict CHD development (Barefoot et al., 1989), but subsequent studies have countered this claim (Hearn, Murray, & Luepker, 1989).

Since hostility has been closely linked to TABP, it is not surprising that counseling models moderating hostility overlap those used for addressing more general Type A concerns (Biaggio, 1987; Smith, 1992). Specifically, stress management, relaxation, cognitive restructuring and skills training have demonstrated efficacy in

reducing anger and hostility (Hazeleus & Deffenbacher, 1986; Novaco, 1975). Treatment factors that may reduce anger and hostility include: (a) alteration of the world view that predisposes a client to mistrust and hostile interaction: (b) development of self monitoring and self-instruction skills so as to replace anger/hostility provoking self-talk: (c) assertiveness skills: (d) social skills development: and (e) stress management skills (Biaggio, 1987: Novaco, 1975).

As with TABP treatment, the primary objective in anger/hostility counseling should be to address the cognitive, behavioral, physiological and environmental dimensions. For, as we have seen, hostility is a product of the bidirectional interaction of all four dimensions of human functioning, and, in terms of cardiac functioning, is capable of reeking havoc in each as well. Hence, for the CHD patient (especially post MI), reduction of hostility may be of paramount importance. As research further differentiates hostility from TABP, and its assessment becomes more sophisticated, interventions aimed at its reduction will surely proliferate.

A Model for Psychosocial Interventions with the Cardiac Patient:

Putting the Causal Pathway Model into Action

One benefit of constructing a biopsychosocial causal pathway model of CHD is to ease the identification of the mediating variables, their relationship to one another and the logical progression toward or away from a disease state. Not only does this feature assist in conceptualizing etiology as we have seen, but it allows one to better fashion interventions as well. Now that the primary, secondary, and tertiary mediating factors of CHD, as well as a review of their relation to critical cardiac events have been outlined, a comprehensive model of intervention can be formulated. An assumption guiding the development of the following treatment model is that a mapping of the causal interactions of CHD is a prerequisite for effective assessment and treatment. By understanding the links in the causal chain of CHD, one is more able to accurately

assess, from a biopsychosocial perspective, which links come into play in a given case of CHD, and, in turn which treatment modalities best address the patient's needs.

Per the referral of a cardiac patient from his/her cardiologist, how should a psychologist go about working with that individual? Or, more specifically, how should the clinician uncover: (a) why the referral was necessary; (b) what psychological issues may be impacting the patient's cardiac functioning; and (c) how psychological treatment might aid in the patient's recovery? It is my contention that a biopsychosocial causal pathway model can and should guide both the assessment and intervention methodologies pertaining to any disease process, especially CHD. By using the model as a framework to guide clinical interview questions and/or the selection of measures geared toward a specific component (i.e., Cook-Medley Hostility Scale or the Beck Depression Inventory), the clinician can perform the equivalent of an informal *pathway analysis*. In much the same way that variables are selected for inclusion in a regression model, the mediating factors most relevant to the *individual patient's* disease pathway can be identified...resulting in a *model* of a particular patient's CHD. Once identified, an intervention strategy reflecting the biopsychosocial make-up of *that* individual's illness can be formulated. Hence, Mr. Jones is not subjected to cognitive restructuring of beliefs underlying hostility unless hostility is implicated as a mediator in his case. Or, as the model implies, psychosocial factors may not always mediate heart disease. Therefore, causal pathway assessment may suggest that intervention is best targeted at risk *behaviors* such as diet, exercise and smoking. In sum, the use of a causal pathway modeling has applications beyond empirical analysis. The clinician can use the same logic and rationale, both formally and informally, in the psychosocial assessment and treatment of any medical illness to better refine assessment procedures and treatment plans. Since CHD has been especially well researched and many of the mediators (both psychosocial and

biological) have been identified. a causal pathway can be mapped and used as template for clinical investigation and intervention. Precisely how this process might work is contained in the following discussions of assessment and treatment procedures.

A Biopsychosocial Causal Pathway Model for psychological assessment and treatment of CHD and its contributing biopsychosocial factors is outlined below. The model utilizes a structured assessment process (based upon the causal pathway model of CHD specified in the previous pages) to determine which treatment elements are most appropriate for a given patient. The assessment process will be briefly reviewed. followed by a description of the guidelines for a psychosocial intervention model that have emerged from the literature as efficacious in treating the various CHD mediators within in a short-term format. A brief case example concludes the section so as to demonstrate how an individualized assessment/treatment strategy within a biopsychosocial casual pathway framework might unfold.

The reader will notice that a standard cookbook-like treatment format is not presented. Since the objective of using pathway modeling is to custom build a treatment plan utilizing only those treatment models and techniques that have been found efficacious (as outlined in the previous section) and *relevant* for a given patient. only parameters for execution of a treatment model is practical.

Assessment Utilizing Biopsychosocial Causal Pathway Modeling

While most well-trained counseling psychologists gravitate toward a given client's most salient issues in an interview/assessment. the use of a more structured. solution-focussed strategy may assist in the efficiency of this process. A causal pathway model that has been specifically fashioned for a particular disease process (i.e., CHD) from theoretical and empirical literature might serve as an excellent template for structured assessment and treatment plan formulation. It is important to note that. within a causal pathway treatment model. the assessment process is the most essential

component. For. it is the assessment process that utilizes the casual pathway of CHD to flag key issues for treatment. The assessment model proposed here involves two dimensions: (1) CHD screening via informal and structured interview: and (2) objective assessment of mediating factors.

Interview Phase

Any assessment should begin with a general intake. rapport building clinical interview. In the proposed model. this interview might occur within the first meeting after the referral from the physician has been made. During this step of assessment. the clinician should attempt to determine if the client is in any acute psychological distress and/or if pervasive psychopathology is indicated. This is a critical determination. for CHD causal pathway modeling should not supersede the clinical needs of the client. To assist the clinician in determining global psychological functioning. the MMPI should be administered prior to more CHD-oriented objective assessment.

The second feature of the interview phase. which would likely occur during a second meeting with the client. is structured exploration. Specifically. it is proposed that a structured interview form be used to screen for the presence and relevance of each mediator of CHD for a particular patient. This interview guideline would be comprised of questions designed to assess the presence/severity of each of the known mediators of CHD (i.e.. TABP. hostility. depression. marital distress. stress. diet. smoking. exercise level. and emotional/social support. major life events. employment status) so as to tap the cognitive. behavioral. physiological and environmental dimensions of the disease. The interview form (tentatively entitled the Myocardial Infarction Risk Factor Inventory - Interview Form. or MIRFI-I) is currently under development by the principal investigator to compliment the MIRFI screening measure (to be introduced in Chapter III). A screening of cardiac history (timetable of critical events. current diagnoses etc.) would also be included in this interview. While not designed as a

diagnostic device, the MIRFI-I would be a tool to guide the clinical interview to insure that key areas of the CHD process are assessed.

Objective CHD Assessment Phase

The second phase of assessment would involve the administration of the Myocardial Infarction Recovery Index (MIRFI) screening instrument, a 30-item self-report measure tapping six key areas of cardiac risk (depression, marital distress, Type A, hostility, social support and treatment compliance). The next step would be the selection of specific valid and reliable measures to objectively measure those factors suggested to be relevant by the MIRFI and MIRFI-I process. The section outlining potential measures for each CHD mediator may serve as a menu from which to select appropriate assessment tools. For example, if results from the MIRFI and initial interview indicate that the CHD patient has a recent history of behaviors and symptoms consistent with TABP, depression and marital discord, the JAS, IDD and DAS might be employed in a type of *personalized assessment battery*. In so doing, the implications put forth by the MIRFI and MIRFI-I can be substantiated.

Results from the interview and objective phases of assessment will, then, guide treatment plan formulation by highlighting themes requiring attention. Moreover, the same results can serve as baseline measures in treatment outcome determination. Given at a midpoint and again toward the end of treatment, treatment progress, the need for adjustment of treatment goals or the readiness for termination can be assessed.

It is hoped that from a streamlined assessment methodology geared specifically toward CHD, central psychosocial features of the patient's illness will be more readily illuminated. In the face of managed care and health care reform, economics may demand increased efficiency in all facets of service provision. The above model based upon biopsychosocial causal pathway modeling may provide increased clinical efficiency as well as empirical accountability.

Guidelines for a Short-Term Cardiac Care Program

As the above model of assessment and its underlying assumptions suggest, not all CHD patients are the same. As we have seen, biology, personality, experiences, cognitive abilities and the environment interact to determine the type and expression of psychosocial mediators, and, in turn , the course of CHD. Treatments, then, should be fashioned to fit the *CHD profile*, if you will, of the individual patient. As a result, a cookbook-like psychosocial treatment regimen for CHD is not realistic, or, in my opinion, ethical. Rather, a cardiac program that systematically treats the personalized needs each patient is recommended.

This is not to say that a cardiac program should have no consistency from patient to patient. The literature offers some clues as to effective and practical guidelines for cardiac counseling.

Guidelines & Considerations

Treatment Length. Much of the outcome data for psychosocial interventions of CHD mediating factors were generated using relatively short-term treatment periods (i.e., 10 to 20 sessions). This suggests that a cardiac care program can be limited to a 10 to 20 week time frame. Moreover, increasing pressure from third-party payers will likely dictate short-term intervention as well. Hence, an effective and cost-efficient cardiac care program can and should approximate a two to six month period. As an added benefit, this time frame coincides with the period post MI that is regarded as critical to prognosis (Kannel, et al., 1979; Moss & Benhorin, 1990; Norris et al., 1984).

Multicomponent Approach. Second, the literature suggests that effective treatment of most CHD psychosocial correlates involve a multidimensional approach (Blumenthal & Emery, 1988). The combined use of techniques that address the cognitive, behavioral, physiological and environmental aspects of a given psychosocial

issue (i.e., TABP, stress, marital distress) are thought to characterize an effective methodology (Nunes et al., 1987; Price, 1988; Thoreson & Powell, 1992). For the clinician, this translates into knowledge of an array of efficacious psychotherapy models and techniques. The above section on CHD mediator-specific interventions was included to serve as a menu from which such techniques might be selected. However, this listing is far from comprehensive, and, therefore, the clinician should add to his/her own menu of psychosocial interventions from which to draw.

In addition, the multi-dimensional nature of CHD requires the ability to weave the various components of treatment together into a plan that is coherent and meaningful to the client. For example, stress management and therapy for depression might be woven together within a cognitive-behavioral framework and presented as such to thwart client confusion and/or perceived fragmented care.

Psychoeducation. Thirdly, psychoeducation has been established as an essential component of cardiac interventions. It is my belief that this educational process should also stem from a biopsychosocial causal pathway model of CHD. In so doing, the patient is not merely bombarded with miscellaneous facts and a list of *do's* and *don't's*, but rather is educated about how aspects of his/her life (personality, genetics, family/vocational context etc.) influence the disease process in an interactive, causal manner. It is hoped that this pathway modeling education will highlight the *role of the patient* in CHD, and, thus, invite them to take a more proactive posture in treatment. Ideally, increased appreciation for the interactive nature of the illness and recovery will translate into increased medical, behavioral and psychosocial regimen compliance.

Therapeutic Relationship. Lastly, but absolutely not least, the long-standing belief that an accepting, empathic, genuine and caring therapeutic relationship should serve as the basis for *any* psychosocial intervention certainly applies to all facets of a

cardiac care program. Whether marital therapy, Type A counseling or CT of depression is employed, the mental health practitioner should, above all else, establish a supportive environment for the CHD patient. Post MI patients and their families, especially, are likely to benefit from a calming, supportive alliance.

Personalized Cardiac Care Treatment: A Case Example

Since a biopsychosocial causal pathway model of assessment and treatment is conceptualized as a dynamic and personalized process, the application of a specific cardiac care treatment plan is best explained via a case example.

The Client

Bob, a 55 year old distribution manager for the postal service is referred by his physician for unstable angina which has been linked to ischemic heart disease. The physician notes that in discussions with Bob and his spouse during the two years of treatment since his first and only heart attack, he has become aware of several life issues that are concerning. First, the wife, Betty, describes Bob as having a volatile temper in which he *blows his top* with little provocation. Second, Bob's typical work habits include 14 hour days 6 to 7 days per week. Third, the couple had disclosed that they had been separated just prior to the heart attack, and have continued to have trouble during that past year or so.

The Intake

A 90 minute intake interview is scheduled with both Bob and Betty present. The primary objective of the first half hour of the intake is to build a trusting and supportive relationship with the couple. This process might be facilitated by gathering some general family and personal history (i.e., "How did you two meet, anyway?"), leading gradually to the period just before and since the heart attack. Additionally, a review of the cardiac care program, including the assessment process, expected duration, types of treatment, and costs would also be discussed. At this point, a

commitment for assessment would be formalized through a signed informed consent document.

In the remaining hour or so, the clinician focuses on gaining a good sense of overall psychological functioning, and a solid medical and psychosocial history. The session would conclude by scheduling an MMPI-2 administration and a session for a more structured CHD interview (including the MIRFI-I).

Structured Interview

During a 50 minute session, the MIRFI and MIRFI-I forms would be administered (the MIRFI-I could be performed in the intake session if time allows). Since Bob is married, it might be useful to have each spouse undergo the interview separately, and then together so as to gain varying perspectives on the issues. This would be a delicate matter since the couple has a known history of conflict. Above all, Bob must consent to Betty's inclusion in treatment.

The Assessment

Based on a review of the MIRFI system administrations, clinical interview and the MMPI-2 administration, three themes resembling known CHD mediators are identified: TABP, marital distress and depression. In a third session, the results are discussed with Bob (and Betty if consent is granted) to debrief and confirm/expand upon the findings.

It is determined from this debriefing session that the themes seem quite accurate, and as a result, Bob will undergo testing via the JAS, DAS and IDD. Results from the objective assessments confirm strong patterns of Type A behavior, perceived marital discord, and moderate depression. The results are discussed with Bob and his wife in another debriefing session during which both partners acknowledge severe conflict within their marriage, and a discussion of TABP and clinical depression reveal Bob's concurrence with the data. It is during this session that the clinician outlines a

proposed treatment plan. and secures a commitment (verbal contract) for treatment from the couple.

Treatment

The clinician formulates a combined treatment plan to specifically address TABP. depression and marital distress. From the menu of treatment modalities regarded as effective and applicable to a short-term format (as described in a previous section). the clinician finalizes a 20 week program consisting of stress management. relaxation training. supportive psychotherapy. cognitive therapy for depression. referral to a couples CHD support group and BMT. Depending on availability of resources and the clinical context. all of the above could be performed by the same clinician. or elements (i.e.. BMT. CHD support group) could be farmed out via referral. Given Bob's situation. a 20 week treatment plan might resemble the following:

Week 1 - 2: Begin weekly supportive psychotherapy and stress management training sessions. Refer couple to a CHD support group.

Week 3 - 10: Begin weekly BMT sessions. Begin weekly individual CT for depression and TABP concurrent with brief in-session stress management/relaxation exercises (to relax and hone skills).

Session 10: Continue support group. Administer JAS. IDD & DAS as midpoint assessment.

Week 11-19 (Session 11): Make determination if continued treatment is necessary. or if treatment goals require modification. If needed. continue CT & BMT. Continue support group.

(Session 19): Administer JAS. IDD & DAS as endpoint assessment.

Week 20 (Session 20): Reevaluate treatment. Terminate if appropriate or refer for long-term individual counseling and/or marital therapy.

As one can see. the above plan has been personalized to meet the needs of Bob

and Betty as they were defined by clinical interview and objective assessment. Certainly, many treatment plans will incorporate similar treatment elements, for, as we have seen, most cases of CHD involve similar and overlapping psychosocial correlates (i.e., stress, TABP, depression etc.). Nevertheless, there are components of treatment that could be postponed or eliminated altogether given an accurate picture of an individual's CHD biopsychosocial profile. Therefore, through causal pathway modeling assessment, treatment issues are prioritized, thus maximizing time, resources and, hopefully, CHD treatment.

Summary of the Implications for Psychological Intervention

Epidemiologic data suggest that CHD is one of this nation's most significant health care problems. Hence, effective treatment and prevention strategies are needed to combat CHD from three perspectives: (1) *primary prevention* before occurrence of CAD; (2) *secondary prevention* so as to head off initial cardiac events; and (3) *rehabilitation* to reduce the number of recurrent cardiac events.

To this end, a biopsychosocial causal pathway model of CHD has been proposed as a means by which to better conceptualize etiology, assessment and treatment. Through the delineation of the specific mediators of CHD and their interrelationships, it is thought that assessment and treatment strategies can be streamlined to address the specific factors relevant to a given patient's CHD. In this vein, the parameters for a short-term cardiac care program have been outlined. Further research is needed to better clarify the links between known mediators and CHD, as well as to uncover those yet to be identified.

Coronary heart disease is truly a biopsychosocial phenomenon that requires understanding and appreciation of the many dimensions of the disease process for effective treatment to occur. Biopsychosocial causal pathway modeling may be one way to more effectively put theory into practice.

CHAPTER III: METHODS

Participants

Sample Demographics for the Entire Baseline Sample (N=53)

The sample at baseline was comprised of a total of 54 patients. However. this number fluctuates from 52 to 54 depending upon the variables in question due to missing data. The valid sample on key demographic variables was comprised of 53 males. ages 35 to 70. hospitalized in a Veteran's Affairs Medical Center for either unstable angina or myocardial infarction. The mean age for the sample was 57.92 (SD=8.09). Of these patients. 22 (42.33%) were admitted for myocardial infarction. and 31 (59.6%) for unstable angina. The breakdown by marital status was as follows: 26 married (48.1%): 17 divorced (31.5%): 2 widowed 3.8%): 7 cohabitating (13.2%): and 1 separated (1.9%). Fifty-one of the sample (98.1%) were Caucasian. and one identified himself as Black (1.9%). The number of years in school ranged from 7 to 20. with an average of 11.83 (SD=2.35). Eighteen of the 52 participants (34.6%) were employed. 21 (40.4%%) were retired. 14 (26.9%) were unemployed. while 1 case (1.9%) was missing employment data. Annual income levels were grouped into 0-$15.000. 16-$30.000. and >$30.000 brackets. Of 53 respondents with income data available. 30 (56.6%) fell within the 0-$15.000 group. 17 (32.1%) in the 16-$30.000 group. and 6 (11.3%) in the above $30.000 group. Of the 53 participants providing information as to where they lived (city/suburb or country). 38 (71.7%) lived in the city. and 15 (28.3) lived in the country. Non-smokers out numbered current smokers. with 32 (60.4%) and 20 (39.6%) respectively. Current smokers averaged .57 (SD=.84) packs per day. Patients in the sample reported an average of 1.34 heart attacks ranging from 0 to a high of 5 (SD=1.25).

Sample Demographics for the Follow-Up Group (N=30)

The following demographic information is provided to describe the sample of

patients that participated in Time 2 follow-up assessment. All data included here is derived from the participant's status at *baseline*. Since the focus of the current study is to examine variables at hospitalization and their respective impact on recovery 5 to 8 months later. no *new* demographic data was collected at Time 2.

The sample at Time 2 Follow-Up (5 to 8 months post-hospitalization) was comprised of 30 participants (56.6% of baseline N) with an average age of 58.14 (SD=8.20. ranging from age 35 to 70). Of these. 10 (33.3%) were diagnosed with myocardial infarction at baseline. and 20 (66.7%) were classified as unstable angina at baseline. Seventeen (56.7%) of the Time 2 sample were married at baseline. 7 (23.3%) were divorced. 2 6.7%) were widowers. and 4 (13.3%) identified themselves as cohabitating. Eleven (36.7%) identified themselves as employed at baseline. 12 (40.0%) as retired. and 7 (23.3%) as unemployed. In terms of baseline income levels. 15 (50%) of the Time 2 sample were in the 0-$15.000 per year bracket. while 10 (33.3%) were in the 16-$30.000 group. and 5 (16.7%) fell within the Above $30.000 bracket. Twenty-one (70%) live in the city. and 9 (30%) live in the country. As in the baseline sample. the Caucasian group was in the majority with 29 (96.7%). while 1 member of the Time 2 sample identified himself as American Indian. Finally. 11 participants (36.7%) were smokers at baseline. and 19 (63.3%) were not active smokers at the time of hospitalization.

Instruments

See Appendix A for baseline measures and Appendix B for those at follow-up.

Marital Satisfaction at Baseline

Dyadic Adjustment Scale (DAS; Spanier. 1976)

The DAS is a 32-item self-report measure designed to assess the quality of a dyadic relationship. Unlike many other measures. the four subscales of the DAS (Dyadic Consensus; Dyadic Satisfaction; Dyadic Cohesion; and Affectional

Expression) are geared to tap dimensions of both married *and* unmarried committed *relationships* (Spanier. 1989. p. vii). Interestingly. little difference has been noted in the response patterns of gay. lesbian and heterosexual couples on the DAS (Kurdek & Schmitt. 1986). The novel flexibility has made the instrument a favorite of the researcher and clinician alike. for the DAS has been used in more than 1000 empirical investigations since its development. and is considered a widely utilized clinical aid.

Dyadic Consensus measures the degree to which the couple agrees on important relationship matters such as religion. money. household tasks. recreation and time spent together *as perceived by the respondent* (Spanier. 1989). *Dyadic Satisfaction* is based upon the level of tension in the relationship and the extent to which the respondent has considered leaving the partner such that high scores represent present satisfaction and commitment. *Affectional Expression* taps the respondent's satisfaction with the overt expression of affection and sexual activity in the relationship. Finally. *Dyadic Cohesion* measures the number of common interests and activities shared by the couple. The breakdown of the above subscales makes the DAS an especially appealing measure for the current project. for it will allow analysis of the overall relationship as well as sub-components as they relate to the other variables in question. In this way. specific interactions and correlations between psychosocial mediators of recovery and relationship dynamics can be identified. further clarifying which aspects of marital distress have the greatest impact on post hospitalization recovery.

Overall Dyadic Adjustment

For determining distressed and non distressed couples (as perceived by the respondent) for group membership and factorial designs. the *Overall Dyadic Adjustment* (ODA) score will be utilized. The ODA is a composite of all four subscales such that overall relationship satisfaction is gauged. The DAS scoring system regards *high scores as good.* or evidence of a strong presence of a positive relationship variable

(e.g., Dyadic Satisfaction). Utilizing a T score mean of 50 and a standard deviation of 10, Spanier (1989) has proposed the following interpretive cutoffs to be used with T scores for the individual subscales and the ODA:

"Positive"	Above 70	Very Much Above Average
	66 to 70	Much Above Average
	61 to 65	Above Average
	56 to 60	Slightly Above Average
	45 to 55	Average
	40 to 44	Slightly Below Average
	35 to 39	Below Average
	30 to 34	Much Below Average
"Negative"	Below 30	Very Much Below Average

From a psychometric perspective, Cohen (1985) and others (Follette & Jacobson, 1985; Johnson & Greenberg, 1985) have touted the DAS as *one of the best pencil and paper indicators of dyadic adjustment* (Spanier, 1989, p. 1). Numerous studies using Cronbach's alpha have reported relatively high internal consistency. Spanier (1976) found a total scale internal consistency reliability of .96. Subsequent research has substantiated this finding for both male and female respondents (Antill & Cotton, 1982, .90; Filsinger & Wilson, 1983, .93; Johnson & Greenberg, 1985, .84). Moreover, three of the subscales have elicited internal consistency reliabilities in various studies ranging from .73 to .92, with the Affectional Expression scale coming in slightly lower with a range from .58 to .73 (Spanier, 1989). Test-retest reliability also appears strong, with reported reliability coefficients in the .96 range (Stein et al., 1982). More recently, Whisman and Jacobson (1991) concurred that the DAS possesses high internal consistency as well as sound descriminant validity. A host of researchers have reported the DAS to possess solid concurrent and predictive validity

(Dobson. 1987: Smolen et al.. 1986: Spanier. 1989).

Depression at Baseline

Inventory for Diagnosing Depression (IDD: Zimmerman. Corvell. Corenthal. & Wilson. 1986).

The IDD is a 23-item self-report scale that was originally designed to diagnose depression per DSM-III criteria (APA. 1980) while. in its current form. the measure covers all DSM-IV (APA. 1995) criteria for depression. This differs from the widely used Beck Depression Inventory (BDI: Beck. Steer. & Garbin. 1988) in that the BDI seeks to identify the *level* of severity of only a portion of the DSM-IV specified symptoms of major depression. It is believed that utilizing a measure that aligns with the profession's established diagnostic standards helps to validate the assessment of a variable or construct. In terms of research. this may translate into a more confident interpretation regarding the variable in question. as well as a heightened ability to generalize findings to an actual clinical population who. in most cases. will be diagnosed by the very same criteria. Since a central objective of the current study is to apply findings to clinical work with the cardiac patient. the IDD's emphasis on diagnostic criteria made it an especially attractive tool.

The IDD is a relatively short. easily self-administered pencil-and-paper format that allows for rapid (5-10 minutes) completion. Specifically. each item focuses on one symptom of depression by offering five statements that reflect varying degrees of severity of that symptom. The respondent must chose one statement from each item that best describes how they have been feeling within the past two weeks. Each statement is weighted per the level of severity from 0 (no loading on the symptom) to 4 (highest loading on the symptom). The IDD yields a continuous measure of depression severity. thus allowing for a range of statistical analyses. This overall score is calculated by simply totaling the value (0 to 4) for each of the 23 endorsed statements.

Unlike the BDI, the IDD also includes with each item a question asking if the symptom has been present *less* or *more than* two weeks. This feature is an added benefit because even though the instructions plainly state that responses should be based on *the past two weeks*, it is not difficult for a subject to interpret items as describing long-standing feelings.

While the psychometric data is not as plentiful as with an older instrument, the IDD has been found in initial studies to be both a reliable and valid measure of depression (Pace & Trapp, 1995; Zimmerman & Coryell, 1987; Zimmerman et al., 1986). In one study of eight nondepressed and eight depressed psychiatric inpatients, Zimmerman et al. (1986) found consecutive day test-retest reliability to be .98, a split-half reliability of of .93 and Cronbach's alpha of .92. Subsequent split-half reliability investigations resulted in very comparable findings (Zimmerman & Coryell, 1987). In direct comparisons with the BDI, the IDD has demonstrated notable concurrent validity. Zimmerman et al. (1986) found a positive correlation between the two measures of .87 and a high incidence of comparable diagnoses (78.1% to 81.7%). In overall diagnostic concurrence, the BDI and IDD have been reported to elicit similar diagnoses of depression approximately 97.2 % of the time (Zimmerman & Coryell, 1987). Pace and Trapp (1995) report concurrent validity between the BDI and IDD per the Pearson r at .90, $p<.001$. In addition, Cronbach's alpha for the IDD was .91 suggesting sound internal consistency. While Pace and Trapp found diagnostic agreement to vary as a function of severity (score) cutoff points, the BDI and IDD diagnosed depressed and nondepressed participants almost equally at the more severe cutoff ranges. Hence, the IDD, while a young instrument relative to the BDI, seems to offer the clinician and researcher a psychometrically sound measure of depression.

For all of the above reasons, its simplicity, cost effectiveness, psychometric stability and most notably its emphasis on covering all DSM-IV diagnostic criteria, the

IDD was chosen as the primary measure of depression for the current study.

Measures of Marital Satisfaction and Depression at Time 2

Gauge of Marital Satisfaction & Depression

While the use of the DAS and IDD at Time 2 was initially intended and preferable, a change in follow-up assessment (discussed below) was necessary to accommodate assessment over the phone. The authors felt that the inclusion of the DAS and IDD in their entirety might be overly taxing for participants in a follow-up phone call, and, in turn, might jeopardize valid reporting on the recovery measure which is regarded as the most essential aspect of follow-up. As a result, marital satisfaction and depression at Time 2 were represented by one global Likert-Type question each (see Appendix B for these measures). Lower scores represented more negative degrees of the variable (e.g., 1= "Extremely depressed", 5="Less depressed than before my heart trouble").

Collateral/Risk Factor Variables at Baseline & Time 2

Myocardial Infarction Risk Factor Inventory (MIRFI)

As the above *Causal Pathway Model of CHD* (and supporting review of the literature) suggests, many potential factors may play a role in CHD and cardiac recovery. In an attempt to tap several of these variables without placing an undue burden on the participant by including a full-length measure for each, a 30- item scale was created by the principal investigator. It is beyond the scope of this study to attempt to validate a new measure. However, it would seem that a brief screening device for psychosocial risk factors of CHD and recurrent cardiac events might be useful to both medical and mental health practitioners. By incorporating the MIRFI into the current study's assessment battery, initial data as to its ability to detect the presence of four key psychosocial factors (in relation to other measures in the project) can be generated. Moreover, limited statistical analysis can be performed to assess any relationships

between MIRFI factors and recovery without a handful of additional measures being added to the already crowded packet.

Complimenting the *Causal Pathway Model of CHD*, the MIRFI will target depression, marital distress, Type A behavior pattern, hostility, as well as social support and treatment compliance (two other factors suggested by the literature to impact recovery). Each of the six factors will be represented by five statements that have been extracted from both the literature and existing instruments thought to be reliable and valid measures of each respective construct. The origins of the items are as follows: (1) depression: IDD (Zimmerman et al., 1986); (2) marital distress: DAS (Spanier, 1976); (3) Type A: Jenkins Activity Survey (JAS; Jenkins, Zyzanski & Rosenman, 1974); (4) hostility: Cook & Medley Hostility Scale (Cook & Medley, 1954); (5) social support (literature); and (6) treatment compliance (literature). Much validation analysis must occur subsequent to the current study to refine the MIRFI such that *only* those items that *best* predict the presence of a given psychosocial construct are included in the screening device.

Each item within the six factors of the MIRFI is a statement eliciting a response via a five point Likert-type scale with labels ranging from *strongly disagree* to *strongly agree*. A composite score for each factor will be determined by totaling the five Likert weighted responses. A *MIRFI Total Index Score* will be calculated by totaling all six composite factor scores.

Since Factor 1 of the MIRFI is a gauge of marital satisfaction, only married and cohabitating participants will be administered the *entire* MIRFI. All others will complete the MIRFI minus the Factor 1 questions. To allow comparisons of MIRFI Total Index Scores across marital status groups, a *MIRFI Adjusted Total Index Score* will be computed for all valid participants. This adjusted score is simply the sum of Factors 2 thru 6, and it will be the primary MIRFI variable so as not to limit MIRFI analysis to

only married and cohabitating participants.

The composite score for each of the six factors (the sum of the five questions comprising the factor) will also be examined for their relationship to key variables.

MIRI Chest Pain Index

The literature suggests that the frequency, duration, and intensity of chest pain is often employed by physicians to gauge disease severity. The first four items of the *Myocardial Infarction Recovery Index- Short Form* (MIRI-SF; see below for description) specifically inquire as to existing chest pain frequency, severity, duration, and movement required to bring about pain. To gauge disease severity, these four questions (presented in a Likert-type format with weights from 1 to 5) are to be summed to comprise an overall chest pain index entitled the *MIRI Chest Pain Index*. This measure will be collected both at Baseline and Time 2.

It is important to note that a handful of physiological measures are gaining favor in the determination of disease severity (e.g., ejection fraction, percent of arterial blockage via MRI & CT Scan). Unfortunately, within the current sample, no uniform method of assessing CHD severity was employed. As a result, it was impossible to find a single variable with which to track disease severity consistently across the entire sample. For this reason, the *MIRI Chest Pain Index* seemed a viable, albeit crude, alternative.

Dependent Variable Measures at Time 2

Recovery Post Hospitalization

Myocardial Infarction Recovery Index-Short Form (MIRI-SF)

The primary dependent variable is post MI recovery as measured by the Myocardial Infarction Recovery Index-Short Form (MIRI-SF) which was adapted from the Myocardial Infarction Recovery Index (MIRI; Utz & Beach, 1988). In its original form, the MIRI was composed of a weighted combination of seven factors determined

from past research to be good indicators of recovery: sexual activity (10 points): self-perception of recovery (10 points): angina (10 points): return to subsequent activities (10 points): absence of subsequent MI (20 points): absence of subsequent emergency department visits (10 points): and absence of hospitalizations related to coronary difficulties (15 points). The maximum score a patient can receive is 85 points. A component of the dependent measure is cardiac death within the five to eight month time frame. Death is scored as a 0 MIRI score for the assessment period in which the fatality occurred. The MIRI has shown relatively sound concurrent validity in the past (Beach et al., 1992). Since the MIRI is an unpublished manuscript, comprehensive reliability and validity data are not readily available. The measure has, however, been utilized in previous research with success. Moreover, each variable of recovery in the index was chosen for inclusion based upon theoretical and empirical research suggesting its link to overall cardiac recovery post MI (Beach et al. 1992). Hence, the measure has a great deal of face validity. To compensate for the lack of psychometric data, additional measures will be utilized for cross validation purposes.

The original MIRI was designed as a nursing interview questionnaire. In an attempt to make it more palatable as a self-report measure, the MIRI was altered by the current authors and the MIRI-SF was formed. Essentially, the content of the seven factors and the 34 questions comprising them were not changed for the MIRI-SF. However, the wording of the questions and the likert-type responses were adjusted to make them read more as self-report statements.

The MIRI was further altered to accommodate the follow-up assessment phases. Specifically, the measure was shortened to only 20 questions, and the graphic layout was designed to make it much easier to read as a self-report measure. The authors felt that brevity of the instrument was essential for successful participant compliance. The seven overall content areas addressed by the MIRI remained intact

within the MIRI-SF, but several questions that either seemed redundant or extraneous were deleted. Unlike the original MIRI which weighted many of the items differently, the MIRF-SF has for each of the 20 questions the same likert-type scaling (0-5).

In sum, the primary measure of cardiac recovery is the MIRI-SF Total Index Score which is simply the sum of the five factors. See Appendix B for a copy of the MIRI-SF.

CHD-Related Mortality

Cardiac death has historically been employed as a primary dependent measure of post MI health status and recovery. While incorporated into the MIRI-SF scoring, cardiac death was used as a distinct dependent measure of recovery. Simply put, mortality frequency was tallied.

Index of Nonfatal Recurrent Cardiac Events

Previous research has often utilized recurrent (post-hospitalization) nonfatal cardiac events as a gauge of recovery. At Time 2 Follow-Up (5 to 8 months post-hospitalization) the authors tracked (1) recurrent heart attacks, (2) emergency room visits linked to cardiac symptoms, and (3) hospitalizations for cardiac-related symptoms for each patient. This was accomplished using the three items of the MIRI-SF which specifically inquire about the status of the three variables. When summed, they comprise Factor V ("Recent Trouble With My Heart") of the MIRI-SF.

Procedures

Source of the Sample & Inclusion Criteria

The sample of males age 70 and under was recruited from a large regional veterans affairs medical center in the south-central United States. Each participant was contacted while hospitalized in the cardiac intensive care or the cardiac care units. The protocol for patient selection was developed by the authors in conjunction with staff cardiologists. Initial inclusion criteria were as follows. The patient was deemed: (1)

as an unstable angina or post MI patient; (2) mentally competent; (3) medically stable; (4) age 70 or younger; and (5) had not recently undergone heart transplantation. These initial criteria were evaluated by the staff cardiology fellow who was in charge of all cardiac patients admitted to the VA hospital. A research assistant then contacted the cardiology fellow weekly for a list of patients meeting the inclusion requirements. Prior to contacting the patient, the research assistant spoke with the attending nurse to ensure that the patient was up to seeing non-family that day, and to avoid making contact on a day that a medical procedure had been done. Once approved by the attending nurse, the patient was contacted in their hospital room. All assessment procedures performed during the inpatient stay were regarded as *Baseline Assessment.*

Target Sample Size

Since both correlation and factorial designs will be employed, the sample must be large enough to provide adequate power at the .01 and .05 levels for a variety of statistical procedures. Previous research is lacking for many of the variables under study, however, based upon the few variables that have been linked to MI recovery via correlational research (i.e., spouse's sexual satisfaction, overall marital distress and depression; reference to come), a sample of at least 50 participants is needed (per power analysis of H.E. Garrett, 1966 as cited in Borg & Gall, 1989).

Baseline Data Collection Procedures

Once approved by the cardiology fellow and attending nurse, the patient was contacted in his hospital room by a research assistant. At this time, the Informed Consent Form was reviewed and signed, and administration of the baseline assessment battery was begun. The baseline assessment packet contained: one cover letter outlining the study; one Informed Consent Form; a Personal History Questionnaire (targeting familial, educational, occupational, marital and medical history); one *Dyadic Adjustment Scale* (DAS; Spanier, 1976) to be filled out by the patient; one *Myocardial*

Infarction Recovery Index (MIRI: Utz and Beach, 1988); one *Myocardial Infarction Risk Factor Inventory* (MIRFI; Keller & Pace, unpublished); and one *Inventory for Diagnosing Depression* (IDD; Zimmerman, Coryell, Corenthal, & Wilson, 1986).

In most cases, the instruments were orally administered to reduce the discomfort of sitting up in bed. To make it easier for the patient to respond to the questions while laying down, visual aids were constructed for each of the assessment instruments. These aids offered the likert-type responses to each question in very large, bold type, on a laminated flip chart. In this way, the patient was able to read along with the research assistant, or simply point to the most correct response. If the patient was not married or cohabitating with a significant other, the DAS was excluded from the assessment. See Appendix A for the baseline assessment battery.

Follow-Up Assessment Procedures

One follow-up assessment phase was planned at approximately five to eight months post-hospitalization. Initially, follow-up was to be done via mailings of assessment packets. Fifteen packets were, in fact, mailed at the appropriate interval. Only five were returned, and, of these, two were incomplete. It was determined that, for successful follow-up assessment, administration of the instruments would have to be done by phone. A phone assessment protocol was created that was abbreviated to minimize the time needed for each phone call. Specifically, the IDD and DAS were excluded, and replaced by a single likert-type question tapping overall feelings of depression and marital satisfaction. While the exclusion of the IDD and DAS were not preferable, the authors felt that the most essential component of follow-up assessment was a gauge of recovery. Hence, phone follow-up consisted of: (1) a brief reminder of the nature of the study; (2) a brief review of confidentiality issues; (3) administration of the MIRFI; (4) administration of the MIRI-SF; and (5) administration of the two likert-type questions for depression and marital satisfaction.

See Appendix B for a copy of the phone follow-up assessment battery.

The timing of the follow-up assessments was problematic. Unknown to the researchers prior to data collection, the VA computer system (which is regarded as the main database for each veteran) had many outdated phone numbers and addresses. Phone numbers and addresses were not gathered as part of the data collection because the author was assured that the computer was a very reliable source in this regard. As a result, many participants could not be reached in a timely manner, and many others were never located. Therefore, a segment of the sample were contacted in a timely manner (approximately month six), while a portion were assessed at varying intervals from month 5 to month 8. Follow-up assessment, then, was categorized as Baseline (in the hospital), and (5-8 months post-hospitalization). See *Results Section* for specifics pertaining to the final sample.

Data Entry and Processing

The data was encoded from the questionnaires, and entered into SPSS Base 7.0 for Windows. It should be noted that SPSS for Windows, as with many other PC statistical packages, uses some approximations and abbreviations when computing regression equations. In the case of stepwise regression, this shortcut may translate into a reduction in the number of models considered for selection. Hence, models resulting from stepwise regression may not represent *the only* valid and significant combination of variables.

In many of the following tables summarizing results, data has been rounded to two decimal places.

Statistical Analysis

The two primary statistical modalities employed in the current study are product moment correlation and multiple regression. The continuous nature of the majority of the primary variables, the research questions posed, as well as the limited sample size,

made correlation and regression appropriate methodologies. Where the variable type and sample size allowed. group comparison analysis was performed.

CHAPTER IV: RESULTS

For organizational purposes, results are presented in four sections. The first section reviews the baseline sample (N=53) and the follow-up group (N=30) in terms of frequencies and descriptive statistics related to key variables. The remaining three sections represent three distinct phases of analysis. Phase 1 includes only those results directly related to the most essential research questions of the study, or the relationships among *baseline* depression and *baseline* marital satisfaction and recovery. Phase 2 includes analysis involving variables assessed at *time 2 follow-up* and their respective association with recovery. Phase 3 focuses on exploratory analyses including the investigation into predictive models of recovery and preliminary validation of the MIRFI.

Description of the Baseline Sample (N=53) on Key Variables

Frequency of Depressed and Maritally Distressed Patients

Frequency of Depressed vs. Not-Depressed Participants

Based upon the IDD's protocol for determining if a subject meets DSM-IV criteria for major depression, 14 (26.4% of N=53) met full criteria for major depression, while 39 (73.6%) did not.

Frequency of Participants in Distressed vs. Not-Distressed Marriages

Using the T-Score guidelines for determining the presence of notable marital distress outlined above (T-Scores $\leq$ 40 on the Overall Dyadic Adjustment Scale), only 2 patients (6.3% of the 32 married or cohabitating participants) were identified as having a markedly distressed relationship. In contrast, 30 (93.8%) fell within the average or above average range of marital adjustment.

Descriptive Statistics for the Psychosocial Measures for the Baseline Sample

See Table 5.1 for a summary of mean scores for the primary measures across demographic groups for the entire baseline sample (N=53).

Table 5.1
Means for Primary Measures for the Baseline Sample (N=53) Across Demographic Groups

	Baseline Measures			
	IDD Total N, Mean (SD)	DAS ODA N, Mean (SD)	MIRFI Adjusted N, Mean (SD)	MIRI Chest Pain N, Mean (SD)
Total	53, 21.06 (11.55)	32, 117.3 (13.42)	52, 60.69 (15.59)	50, 8.82 (2.94)
MI	22, 21.68 (12.75)	10, 116.8 (17.27)	21, 58.33 (20.25)	20, 8.35 (2.03)
UA	31, 20.61 (10.81)	22, 117.5 (11.74)	31, 62.29 (11.54)	30, 9.13 (3.41)
Married	26, 20.38 (11.28)	25, 119.1 (12.72)	25, 63.44 (10.20)	24, 8.67 (2.62)
Divorced	17, 20.59 (12.95)	NA	17, 57.94 (22.26)	17, 9.59 (3.66)
Widowed	2, 9.50 (3.54)	NA	2, 42.50 (24.75)	2, 7.00 (1.41)
Cohabit.	7, 27.29 (8.94)	7, 110.7 (14.81)	7, 63.14 (7.34)	6, 7.33 (1.37)
Separated	1, 26.00 (00.0)	NA	1, 58.00 (00.0)	1, 12.00 (00.0)
Employed	18, 21.17 (11.25)	13, 122.1 (9.78)	18, 60.61 (17.36)	18, 8.39 (2.30)
Unempl.	14, 25.29 (13.54)	8, 111.3 (17.72)	14, 63.36 (10.46)	12, 8.50 (2.50)
Retired	21, 18.14 (9.93)	11, 116.0 (12.76)	20, 58.90 (17.29)	20, 9.40 (3.65)
0-$15,000	30, 23.47 (12.42)	16, 114.9 (12.26)	29, 61.76 (15.87)	28, 8.86 (3.39)
16-$30	17, 18.59 (8.73)	12, 118.4 (16.21)	17, 58.06 (16.33)	16, 8.94 (2.46)
>$30	6, 16.00 (12.73)	4, 123.3 (8.02)	6, 63.00 (13.36)	6, 8.33 (2.07)

Note: IDD=Baseline Depression; DAS ODA=Baseline Marital Satisfaction
MIRFI Adjusted=Baseline Risk Factor Index (excludes Marital Satisfaction subscale)
MIRI Chest Pain=Baseline Disease Severity

Statistical Analysis Performed on the Baseline Sample (N=53)

The Relationship Between Depression & Age

The literature also suggests that depressive symptomatology tends to increase with age, especially in older populations (above age 60). To explore this issue, a Pearson Correlation Coefficient was obtained for the IDD Total Scores and Age. No significant correlation emerged (r=-.050, p=.796).

The Relationship Between Marital Status and Depression

The literature has suggested that divorced and separated individuals may be at greater risk for depression. Participants within the baseline sample (N=53) were divided into two groups. Group 1 was comprised of married and cohabitating

participants (N=33). while Group 2 represented those who were divorced. separated. and widowed (N=20). The groups were compared on mean IDD Total Scores via an independent samples t-test. Results indicated that the groups did not differ significantly on the primary measure of depression at baseline when equality of variances was not assumed (t=.619, df=36.51. p=.540: Ramsey. 1981).

The Relationship Between the MIRI Chest Pain Index & Depression

The MIRI Chest Pain Index is a rough gauge of disease severity. It has been proposed that as the severity of heart disease (and related symptoms) increase. the likelihood of depression increases. Results from a Pearson Correlation (2-tailed) between the MIRI Chest Pain Index and the Total IDD Score did not support this hypothesis (r=-.229. p=.242).

The Relationship Between the MIRI Chest Pain Index & Marital Satisfaction

Consistent with previously stated hypotheses. it is proposed that marital functioning is often associated with poor cardiac reactivity. Therefore. exploratory analysis was performed on the MIRI Chest Pain Index and the DAS Overall Dyadic Adjustment Scale. The resulting Pearson Correlation Coefficient was not significant (r=-.091. p=.712).

The Relationship Between the MIRI Chest Pain Index & the MIRFI Adjusted Score

An exploration was undertaken into the possible link between degree of CHD severity as gauged by the MIRI Chest Pain Index and the CHD risk factors as measured by the MIRFI Adjusted Score. No significant correlation was indicated (r=-.108. p=.585).

Description of the Follow-Up Group (N=30) on Key Variables

Depression and Marital Satisfaction Among the Follow-Up Group (N=30)

Depression: Six (20%) of the Time 2 sample met DSM-IV criteria for Major Depression per their IDD scores at baseline. and 24 (80%) did not meet criteria. By

cardiac diagnosis, 3 (10% of the follow-up sample) myocardial infarction patients met criteria for major depression, while 3 of those diagnosed with unstable angina also met criteria for depression.

Marital Satisfaction. Among the 21 married and cohabitating patients of the Time 2 sample, only 1 (4.8%) had a DAS Overall Dyadic Adjustment T-Score ($T \leq 40$) suggestive of notable marital distress. The other 20 (95.2%) did not reach the T-Score cutoff for significant distress on this determinant of marital distress.

Means for Key Variables for the Follow-Up Group (N=30)

See Table 5.2 for a summary of mean scores for primary measures administered at Time 2 Follow-Up.

Table 5.2
Means of Baseline Measures for the Follow-Up Group (N=30) Across Demographic Groups

	Baseline Measures			
	IDD Total N, Mean (SD)	DAS ODA N, Mean (SD)	MIRFI Adjusted N, Mean (SD)	MIRI Chest Pain N, Mean (SD)
Total	30, 19.60 (11.91)	21, 116.2 (12.03)	30, 60.73 (16.11)	28, 8.82 (3.01)
MI	10, 21.40 (14.92)	6, 122.0 (10.62)	10, 60.30 (22.75)	9, 7.89 (2.26)
UA	20, 18.70 (10.42)	15, 113.9 (12.10)	20, 60.95 (12.29)	19, 9.26 (3.36)
Married	17, 19.35 (11.12)	17, 118.8 (10.79)	17, 63.00 (10.99)	16, 8.31 (2.68)
Divorced	7, 16.57 (13.01)	NA	7, 56.14 (25.20)	7, 10.68 (3.89)
Widowed	2, 9.50 (3.54)	NA	2, 42.50 (24.75)	2, 7.00 (1.41)
Cohabit.	4, 31.00 (10.30)	4, 105.2 (12.04)	4, 68.25 (3.95)	3, 8.00 (1.00)
Employed	11, 20.91 (12.45)	9, 120.1 (11.02)	11, 66.18 (13.81)	11, 8.55 (2.54)
Unempl.	7, 23.86 (14.87)	5, 110.6 (12.36)	7, 60.71 (9.20)	5, 9.40 (2.51)
Retired	12, 15.92 (9.19)	7, 115.3 (12.98)	12, 55.75 (20.20)	12, 8.83 (3.71)
0-$15,000	15, 24.27 (13.06)	9, 110.1 (11.10)	15, 65.27 (14.83)	14, 8.86 (3.68)
16-$30	10, 15.00 (5.81)	8, 119.6 (12.48)	10, 54.10 (18.30)	9, 9.11 (2.37)
>$30	5, 14.80 (13.85)	4, 123.2 (8.02)	5, 60.40 (13.13)	5, 8.20 (2.28)

Note: IDD=Baseline Depression for Follow-Up Group
DAS ODA=Baseline Marital Satisfaction for Follow-Up Group
MIRFI Adjusted=Baseline Risk Factor Index (excludes Marital Satisfaction subscale)
MIRI Chest Pain=Baseline Disease Severity for Follow-Up Group

Means for the Recovery Measure Across Demographic Groups

See Table 5.3 for a summary of means for the primary measure of recovery (MIRI-SF) across demographic groups.

Table 5.3
Mean Recovery Scores (MIRI-SF) Across Demographic Groups at 5 to 8 Months Post-Discharge

	Recovery Measure (MIRI-SF) N, Mean (SD)
Total	30, 67.43 (13.47)
MI	10, 68.20 (19.83)
UA	20, 67.05 (9.50)
Depressed	6, 62.33 (18.95)
Not Dep.	24, 68.71 (11.93)
Married	17, 66.06 (12.94)
Divorced	7, 70.14 (11.04)
Widowed	2, 78.50 (13.44)
Cohabit.	4, 63.00 (20.51)
Employed	11, 66.55 (15.12)
Unempl.	7, 65.43 (13.60)
Retired	12, 69.42 (12.73)
0-$15,000	15, 64.07 (12.56)
16-$30	10, 70.20 (10.59)
>$30	5, 72.00 (20.58)
Smoker	11, 64.27 (12.08)
Non-Sm.	19, 69.26 (14.20)

A Comparison of Myocardial Infarction and Unstable Angina Patients on Key Variables

To support the theoretical claim that myocardial infarction (MI) and unstable angina (UA) patients are comparable, statistical analyses were performed pitting MI versus UA on the major variables.

MI vs. UA on the Frequency of DSM-IV Diagnosis of Major Depression

Chi Square was performed comparing the rate of major depression among myocardial infarction and unstable angina patients comprising the baseline sample (N=53). Among those diagnosed with myocardial infarction (N=22), six (11.3% of total N) met criteria for major depression, while sixteen (30.2%) did not. In comparison, eight patients diagnosed with unstable angina (15.1% of total N) met criteria for depression, and 23 (43.4%) did not. Per Chi Square analysis, the rate of depression between MI and UA patients was not significant.

Comparative Analysis (MI vs. UA)

for the Baseline Sample (N=53) on Key Variables

A series of t-tests was performed to analyze the comparability of myocardial infarction and unstable angina patients on the primary variables used in the study. This comparative analysis was done for the entire baseline sample (N=53), and again for the group that participated in follow-up assessment (N=30). The results are summarized in two tables (Tables 5.4 and 5.5). Table 5.4 reports the t-tests comparing MI and UA patients for the entire baseline sample, while Table 5.5 displays results of the comparison for the follow-up sample.

Table 5.4
T-Test Analysis Comparing MI to UA Patients on Key Variables for the Baseline Sample (N=53)

Variable of Comparison	MI Group	UA Group	T-Test Equal Variances Assumed	T-Test Equal Var. Not Assumed
	N, Mean (SD)	N, Mean (SD)	t p value (df)	t p value (df)
IDD Total Score	22, 21.68 (12.75)	31, 20.61 (10.81)	.329 .743 (51)	.320 .751 (41)
DAS	10, 116.8 (17.27)	22, 117.5 (11.74)	-.135 .894 (30)	-.117 .909 (13)
T1 MIRFI Adj.	21, 58.33 (20.25)	31, 62.29 (11.54)	-.896 .374 (50)	-.811 .424 (29)
T1 MIRFI Tot.	10, 74.90 (13.09)	22, 72.05 (09.90)	.683 .500 (30)	.614 .549 (14)
T1 MIRI Chest	20, 08.35 (02.03)	30, 09.13 (03.41)	-.922 .361 (48)	-1.02 .315 (47)

Note: IDD=Baseline Depression; DAS=Baseline Marital Satisfaction
T1 MIRFI Adj.= Baseline MIRFI Adjusted Total (excludes Marital Satisfaction Subscale)
T1 MIRFI Tot.=Baseline MIRFI Total (includes Marital Satisfaction Subscale)
T1 MIRI Chest=Baseline MIRI Chest Pain Index (disease severity at baseline)

Table 5.5
T-Test Analysis Comparing MI to UA Patients on Key Baseline Variables for the Follow-Up Group

Variable of Comparison	MI Group N, Mean (SD)	UA Group N, Mean (SD)	T-Test Equal Variances Assumed t p value (df)	T-Test Equal Var. Not Assumed t p value (df)
IDD Total Score	10, 21.40 (14.92)	20, 18.70 (02.33)	.579 .567 (28)	.513 .616 (14)
DAS	6, 122.00 (10.62)	15, 113.9 (12.10)	1.42 .171 (19)	1.51 .161 (10)
T1 MIRFI Adj.	10, 60.30 (22.75)	20, 60.95 (12.29)	-.102 .919 (28)	-.084 .934 (12)
T1 MIRFI Tot.	6, 74.33 (15.72)	15, 73.47 (10.09)	.152 .881 (19)	.125 .904 (07)
T1 MIRI Chest	9, 07.89 (02.26)	19, 09.26 (03.26)	-1.14 .267 (26)	1.29 .209 (22)
T2 MIRI-SF	10, 68.20 (19.83)	20, 67.05 (09.50)	.217 .830 (28)	.174 .865 (11)

Note: IDD=Baseline Depression; DAS=Baseline Marital Satisfaction
T1 MIRFI Adj.=Baseline MIRFI Adjusted Total (excludes Marital Satisfaction Subscale)
T1 MIRFI Tot.=Baseline MIRFI Total (includes Marital Satisfaction Subscale)
T1 MIRI Chest=Baseline MIRI Chest Pain Index (disease severity at baseline)
T2 MIRI-SF=MIRI-Short Form (recovery at follow-up)

Phase 1: Key Findings

The Relationships Among Baseline Depression, Baseline Marital Satisfaction and the Recovery Measure

The Relationship Between Baseline Depression and Recovery

Correlation

A Pearson Correlation (2-tailed) between the IDD Total Score at baseline and the recovery measure (MIRI-SF) resulted in a significant negative correlation (N=30, r=-.442, p<.05). Higher scores on the IDD reflect greater degrees of depressive symptomatology, while lower scores on the MIRI-SF reflect poorer recovery, thus accounting for the negative correlation. See Table 5.6 for a summary correlation matrix of the baseline variables, or Appendix C for a matrix of all possible correlations for the study.

A Pearson Correlation was also performed on the MIRFI Factor 2 (Depression) at baseline and the MIRI-SF Total at Time 2. No significant correlation was indicated.

Table 5.6
Correlations Among the Baseline Measures and Recovery for the Follow-Up Group (N=30)

		DAS ODA	IDD Tot.	MIRFI 1	MIRFI 2	MIRFI 3	MIRFI 4	MIRFI 5	MIRFI 6	MIRFI Adj.	MIRI Tot.
r's	DAS ODA	1.000	-.315	-.777**	-.363	.015	-.100	-.577**	-.357	-.443*	.155
	IDD Total	-.315	1.000	.222	.721**	.485**	.436*	.543**	.353	.608**	-.442*
	MIRFI 1	-.777**	.222	1.000	.313	.179	-.078	.543*	-.016	.332	.070
	MIRFI 2	-.363	.721**	.313	1.000	.563**	.425*	.657**	.273	.682**	-.221
	MIRFI 3	.015	.485**	.179	.563**	1.000	.559**	.414*	.284	.528**	.122
	MIRFI 4	-.100	.436*	-.078	.425*	.559**	1.000	.498**	.348	.586**	-.216
	MIRFI 5	-.577**	.543**	.543*	.657**	.414*	.498**	1.000	.061	.654**	-.420*
	MIRFI 6	-.357	.353	-.016	.273	.284	.348	.061	1.000	.374*	-.147
	MIRFI Adj.	-.443*	.608**	.332	.682**	.528**	.586**	.654**	.374*	1.000	-.350
	MIRI-SF	.155	-.442*	.070	-.221	.122	-.216	-.420*	-.147	-.350	1.000
p's	DAS ODA	.	.164	.000	.106	.950	.668	.006	.112	.044	.501
	IDD Total	.164	.	.334	.000	.007	.016	.002	.056	.000	.014
	MIRFI 1	.000	.334	.	.167	.438	.737	.011	.944	.142	.763
	MIRFI 2	.106	.000	.167	.	.001	.019	.000	.145	.000	.240
	MIRFI 3	.950	.007	.438	.001	.	.001	.023	.128	.003	.522
	MIRFI 4	.668	.016	.737	.019	.001	.	.005	.059	.001	.252
	MIRFI 5	.006	.002	.011	.000	.023	.005	.	.747	.000	.021
	MIRFI 6	.112	.056	.944	.145	.128	.059	.747	.	.042	.437
	MIRFI Adj.	.044	.000	.142	.000	.003	.001	.000	.042	.	.058
	MIRI-SF	.501	.014	.763	.240	.522	.252	.021	.437	.058	.
n's	DAS ODA	21	21	21	21	21	21	21	21	21	21
	IDD Total	21	30	21	30	30	30	30	30	30	30
	MIRFI 1	21	21	21	21	21	21	21	21	21	21
	MIRFI 2	21	30	21	30	30	30	30	30	30	30
	MIRFI 3	21	30	21	30	30	30	30	30	30	30
	MIRFI 4	21	30	21	30	30	30	30	30	30	30
	MIRFI 5	21	30	21	30	30	30	30	30	30	30
	MIRFI 6	21	30	21	30	30	30	30	30	30	30
	MIRFI Adj.	21	30	21	30	30	30	30	30	30	30
	MIRI-SF	21	30	21	30	30	30	30	30	30	30

**. Correlation is significant at the 0.01 level (2-tailed). *.Correlation is sig. at the 0.05 level.

Note: DAS ODA=Baseline Marital Satisfaction; IDD=Baseline Depression
MIRFI 1=MIRFI Marital Satisfaction Subscale at Baseline
MIRFI 2=MIRFI Depression Subscale at Baseline
MIRFI 3=MIRFI Type A Subscale at Baseline
MIRFI 4=MIRFI Hostility Subscale at Baseline
MIRFI 5=MIRFI Social Support Subscale at Baseline
MIRFI 6=MIRFI Treatment Compliance Subscale at Baseline
MIRI-SF=MIRI Short-Form (Primary Recovery Measure Assessed at Follow-Up)

Group Differences Analysis of Depressed vs. Not Depressed Patients

An Independent Samples T-Test with Levene's Test for Equality of Variances (F=2.004. p=.168) was performed on those participants meeting DSM-IV criteria for major depression (N=6) versus those who did not (N=24) at baseline in terms of recovery (MIRI-SF Total Score) at Time 2. Results indicated the two groups did not differ on MIRI-SF Total Scores at Time 2 when equal variance were not assumed (t=-

.786. df=6.026. p=.462). These results should be regarded as tentative due the notable discrepancy in sample sizes.

The Relationship Between Baseline Marital Satisfaction and Recovery

Correlation

Pearson Correlation (2-tailed) analysis of the DAS Factor 5 (Overall Adjustment Scale) and cardiac recovery. as measured by the MIRI-SF Total Score. revealed no significant correlation between the two variables. No significant correlation was found between any of the four DAS subscales and the MIRI-SF Total Score at Time 2. See Table 5.6 for a summary of the above correlations.

Pearson Correlations were also calculated for the Baseline MIRFI Factor 1 (Marital Satisfaction) and the MIRI-SF Total Score at Time 2. Results also indicated no significant correlation. See Table 5.6 for a summary correlational matrix of these variables.

Regression

The four subscales of the DAS at baseline were entered as a four-predictor regression with the recovery measure (MIRI-SF) as the dependent variable to clarify the measure's ability to predict recovery from baseline across 5 to 8 months. The results indicated that. as a model. the four DAS subscales did not predict recovery at follow-up. The results are summarized in Table 5.7.

Table 5.7
Simultaneous Regression for DAS Subscales 1, 2, 3, 4 on Recovery

Model	Association		ANOVA				Coefficients				
	R^2	Adj. R^2	SS	F	df	p	B	SE B	β	t	p
DAS Subscales 4, 1, 3, 2	.359	.199	1428.34	2.25	(4,16)	.110					
DAS Subscale 1							2.69	.960	1.10	2.80	.013
DAS Subscale 2							-2.95	1.41	-.854	-2.10	.052
DAS Subscale 3							-2.88	1.81	-.394	-1.60	.130
DAS Subscale 4							1.85	1.04	.435	1.78	.093

Note: DAS 1=Dyadic Consensus DAS 3=Affectional Expression
DAS 2=Dyadic Satisfaction DAS 4=Dyadic Cohesion

The Interaction Between Baseline Depression and Marital Satisfaction

Correlation

Pearson Correlations (2-tailed) were run to examine the relationship between depression and marital satisfaction at baseline among the entire baseline sample (N=53). Please note, these correlations are *not* represented in Table 5.6, for this table is comprised of baseline correlations for the 30 follow-up participants only. Within the overall baseline sample (N=53), the following correlations emerged linking the IDD (measure of depression) with the DAS (measure marital satisfaction):

- IDD Total Score & DAS 5 (Overall Dyadic Adjustment): r=-.315, p<.05
- IDD Total Score & DAS 1 (Consensus): r=-.430, p<.05
- IDD Total Score & DAS 2 (Satisfaction): r=-.367, p<.05
- MIRFI Factor 2 (Dep.) & DAS 5 (Overall Adjustment): r=-.359, p<.05
- MIRFI Factor 2 (Dep.) & DAS 1 (Dyadic Consensus): r=-.368, p<.05

Regression

Baseline IDD Total Score and baseline DAS Overall Adjustment Score were entered simultaneously into a 2-predictor regression model with the recovery measure (MIRI-SF) as the dependent variable. The results indicated that, as a model, the IDD Total Score and DAS Overall Adjustment Score did not significantly explain the variance in the primary measure of recovery. Results of the analysis are summarized in Table 5.8.

Table 5.8
Simultaneous Regression for DAS ODA Score and IDD Total Score on Recovery

Model	Association		ANOVA				Coefficients				
	R^2	Adj. R^2	SS	F	df	p	B	SE B	β	t	p
DAS ODA & IDD Tot.	.130	.033	516.84	1.35	(2,18)	.285					
DAS ODA Score							5.5E-02	34.06	.047	.204	.840
IDD Total Score							-.413	.279	-.343	-1.48	.156

Note: DAS ODA=DAS Overall Dyadic Adjustment at Baseline (primary measure of marital satisfaction)
IDD Total=Primary Measure of Depression at Baseline

Also at baseline, the MIRFI Factor 1 (Marital Satisfaction subscale) and MIRFI Factor 2 (Depression subscale) were entered simultaneously into a 2-predictor regression model in relation to the MIRI-SF Total Score at Time 2. As a model, the two variables did not adequately account for the variance in the recovery measure at Time 2. Results are summarized in Table 5.9.

Table 5.9
Simultaneous Regression for Baseline MIRFI Factors 1 and 2 on Recovery

Model	Association		ANOVA				Coefficients				
	R^2	Adj. R^2	SS	F	df	p	B	SE B	β	t	p
T1 MIRFI Factors 1, 2	.019	-.090	76.54	.177	(2,18)	.839					
T1 MIRFI Factor 1							.508	1.14	.109	.445	.661
T1 MIRFI Factor 2							-.549	1.07	-.126	-.514	.614

Note: T1 MIRFI Factor 1=MIRFI Marital Satisfaction Subscale at Baseline
T1 MIRFI Factor 2=MIRFI Depression Subscale at Baseline

Phase 2: The Relationships Among Depression and Marital Satisfaction Measured at Follow-Up

The Relationship Between Depression and Recovery Among the Follow-Up Sample

Correlation

A Pearson Correlation (2-tailed) was performed on the MIRFI Factor 2 (Depression) at Time 2 and the recovery measure (MIRI--SF). Results indicated a significant negative correlation (N=30, r=-.699, p<.001) between the two variables.

A Pearson Correlation (2-tailed) was also performed on the Depression Gauge at Time 2 and the MIRI-SF Total Score at Time 2. The procedure resulted in a significant positive relationship (N=30, r=.581, p<.001). Lower scores on the Depression Gauge reflect greater degrees of depression, while lower scores on the MIRI-SF reflect poorer recovery, hence the positive relationship. See Table 5.10 for a matrix of the correlations between major Time 2 measures and recovery.

Table 5.10
Correlations Among Time 2 Measures and Recovery for the Follow-Up Group (N=30)

		Dep. Gauge	Marit. Gauge	T2 MIRFI 1	T2 MIRFI 2	T2 MIRFI 3	T2 MIRFI 4	T2 MIRFI 5	T2 MIRFI 6	MIRI-SF
r's	Dep. Gauge	1.000	.255	-.257	-.731**	-.356	-.211	-.403*	-.230	.581**
	Marital Gauge	.255	1.000	-.811**	-.472*	.112	-.081	-.382	-.028	.380
	T2 MIRFI 1	-.257	-.811**	1.000	.312	-.103	-.282	.266	.294	-.325
	T2 MIRFI 2	-.731**	-.472*	.312	1.000	.292	.351	.609**	.330	-.699**
	T2 MIRFI 3	-.356	.112	-.103	.292	1.000	.472**	.449*	.185	-.173
	T2 MIRFI 4	-.211	-.081	-.282	.351	.472**	1.000	.597**	.085	-.364*
	T2 MIRFI 5	-.403*	-.382	.266	.609**	.449*	.597**	1.000	.048	-.567**
	T2 MIRFI 6	-.230	-.028	.294	.330	.185	.085	.048	1.000	-.215
	MIRI-SF	.581**	.380	-.325	-.699**	-.173	-.364*	-.567**	-.215	1.000
p's	Dep. Gauge	.	.218	.215	.000	.054	.263	.027	.221	.001
	Marital Gauge	.218	.	.000	.017	.595	.699	.060	.894	.061
	T2 MIRFI 1	.215	.000	.	.129	.623	.172	.199	.154	.113
	T2 MIRFI 2	.000	.017	.129	.	.117	.057	.000	.074	.000
	T2 MIRFI 3	.054	.595	.623	.117	.	.008	.013	.327	.362
	T2 MIRFI 4	.263	.699	.172	.057	.008	.	.000	.654	.048
	T2 MIRFI 5	.027	.060	.199	.000	.013	.000	.	.801	.001
	T2 MIRFI 6	.221	.894	.154	.074	.327	.654	.801	.	.253
	MIRI-SF	.001	.061	.113	.000	.362	.048	.001	.253	.
n's	Dep. Gauge	30	25	25	30	30	30	30	30	30
	Marital Gauge	25	25	24	25	25	25	25	25	25
	T2 MIRFI 1	25	24	25	25	25	25	25	25	25
	T2 MIRFI 2	30	25	25	30	30	30	30	30	30
	T2 MIRFI 3	30	25	25	30	30	30	30	30	30
	T2 MIRFI 4	30	25	25	30	30	30	30	30	30
	T2 MIRFI 5	30	25	25	30	30	30	30	30	30
	T2 MIRFI 6	30	25	25	30	30	30	30	30	30
	MIRI-SF	30	25	25	30	30	30	30	30	30

**. Correlation is significant at the 0.01 level (2-tailed). *. Correlation is sig. at the 0.05 level.

Note: Dep. Gauge=Gauge of Depression (Depression Measure at Follow-Up)
Marital Gauge=Gauge of Marital Satisfaction (Mar. Satisfaction at Follow-Up)
T2 MIRFI 1=MIRFI Marital Satisfaction Subscale at Follow-Up
T2 MIRFI 2=MIRFI Depression Subscale at Follow-Up
T2 MIRFI 3=MIRFI Type A Subscale at Follow-Up
T2 MIRFI 4=MIRFI Hostility Subscale at Follow-Up
T2 MIRFI 5=MIRFI Social Support Subscale at Follow-Up
T2 MIRFI 6=MIRFI Treatment Compliance Subscale at Follow-Up
MIRI-SF=MIRI Short-Form (Primary Recovery Measure Assessed at Follow-Up)

Regression

A Stepwise Multiple Regression was performed utilizing the MIRFI Factors 1 (Marital Satisfaction), 2 (Depression), 4 (Hostility), and 5 (Social Support) at Time-2 follow-up. These subscales were included because all but Factor 1 were shown to correlate significantly with the recovery measure. The Marital Satisfaction subscale

was included to explore its contribution to the predictive model. Results from the analysis indicated that the Time 2 MIRFI Depression subscale explained the majority of the variance in the measure of recovery. Results of the stepwise regression are summarized in Table 5.11.

Table 5.11
Stepwise Regression for MIRFI Factors 1, 2, 4, and 5 at Time 2 on Recovery

Remaining Model	Association		ANOVA				Coefficients				
	R^2	Adj. R^2	SS	F	df	p	B	SE B	β	t	p
T2 MIRFI Factor 2	.551	.532	2514.23	28.28	(1,23)	<.001	-2.64	.496	-.743	-5.32	.000
Excluded Variables: T2 MIRFI Factor 1 T2 MIRFI Factor 4 T2 MIRFI Factor 5											

Probability of F to enter: <.50. Probability of F to remove: >.10

Note: T2 MIRFI Factor 1=MIRFI Marital Satisfaction at Follow-Up
T2 MIRFI Factor 2=MIRFI Depression Subscale at Follow-Up
T2 MIRFI Factor 4=MIRFI Hostility Subscale at Follow-Up
T2 MIRFI Factor 5=MIRFI Social Support Subscale at Follow-Up

The Relationship Between Marital Satisfaction Assessed at Follow-Up and Recovery

Results from a Pearson Correlation (2-tailed) examining the MIRFI Factor 1 (Marital Satisfaction) at Time 2 and the recovery measure (MIRI-SF) indicated no significant correlation between the two variables.

The relationship between the Marital Satisfaction Gauge and the MIRI-SF Total Score, both at Time 2, was also analyzed via correlation, and no significant correlation emerged. See Table 5.10 for a summary correlational matrix of follow-up variables.

The Relationship Between Time 2 Depression & Time 2 Marital Satisfaction Correlation

Pearson Correlation Coefficients (2-tailed) were derived to explore the relationship between depression and marital satisfaction assessed at follow-up. See Tables 5.10 for a summary matrix of relevant Time 2 correlations. The Gauge of

Marital Satisfaction (a follow-up measure of marital satisfaction) and the Time 2 MIRFI Factor 2 (Depression subscale) were significantly and negatively correlated (r=-.472. p<.05).

Regression

The MIRFI Factors 1 and 2 at Time 2 were entered as a 2-predictor regression model with the MIRI-SF Total as the dependent variable. Findings indicate that. as a model. the two follow-up variables significantly associate with the recovery measure. Results are summarized in Table 5.12.

Table 5.12
Simultaneous Regression for Time 2 MIRFI Factors 1 and 2 on Recovery

Model	Association		ANOVA				Coefficients				
	R^2	Adj. R^2	SS	F	df	p	B	SE B	ß	t	p
T2 MIRFI Factors 1, 2	.561	.521	2558.04	14.06	(2,22)	<.001					
T2 MIRFI Factor 1							-.311	.448	-.103	-.694	.495
T2 MIRFI Factor 2							-2.52	.528	-.710	-4.78	<.001

Note: T2 MIRFI Factor 1=MIRFI Marital Satisfaction Subscale at Follow-Up
T2 MIRFI Factor 2=MIRFI Depression Subscale at Follow-Up

The Gauge of Depression (a Time 2 measure of depression) and Gauge of Marital Satisfaction (a Time 2 measure of marital satisfaction) were also entered as a 2-predictor regression model in an attempt to clarify their combined ability to explain the variance in the recovery variable (MIRI-SF Total Score). The results indicated that the two follow-up measures of depression and marital satisfaction. as a model. significantly predicted recovery. Results are summarized in Table 5.13.

Table 5.13
Simultaneous Regression for Time 2 Gauges of Depression and Marital Satisfaction on Recovery

Model	Association		ANOVA				Coefficients				
	R^2	Adj. R^2	SS	F	df	p	B	SE B	ß	t	p
T2 Marital, T2 Depression	.353	.295	1560.98	6.01	(2,22)	.008					
T2 Gauge of Marital Sat.							4.81	1.81	.472	2.67	.014
T2 Gauge of Depression							2.78	1.90	260	1.47	.157

Note: T2 Marital=Gauge of Marital Satisfaction at Follow-Up (exploratory measure)
T2 Depression=Gauge of Depression at Follow-Up (exploratory measure)

Phase 3: Exploratory Analyses

Models Among the Key Variables that are Predictive of Recovery

Baseline Variables as Predictive Models of Recovery

Each of the major baseline variables (MIRFI Adjusted Score, DAS Overall Adjustment, IDD Total Score) were entered simultaneously into a 3-predictor regression model to explore their predictive power as a model. Results indicated that the three variables, when placed in a model simultaneously, did not significantly associate with recovery. The findings are summarized in Table 5.14.

Table 5.14
Simultaneous Regression for Baseline MIRFI Adjusted, DAS ODA Score, and IDD Total Score on Recovery

Model	Association		ANOVA				Coefficients				
	R^2	Adj. R^2	SS	F	df	p	B	SE B	β	t	p
T1 MIRFI Adj., DAS, IDD	.132	.021	524.364	.862	(3,17)	.480					
IDD Total Score							-.462	.382	-.383	-1.21	.243
T1 MIRFI Adjusted Score							9.0E-02	.465	.065	.193	.850
DAS ODA Score							7.4E-02	.295	.063	.251	.805

Note: T1 MIRFI Adj.=MIRFI Adjusted Score (excludes Marital Satisfaction Subscale) at Baseline
DAS ODA=DAS Overall Dyadic Achievement Subscale at Baseline
IDD Total Score=Primary Measure of Depression at Baseline

The IDD Total at baseline and the MIRFI Factor 5 (Social Support) at baseline were entered into a 2-predictor regression model. These were the only two variables at baseline that correlated significantly with the recovery measure (MIRI-SF Total Score) at Time 2. The procedure was intended to examine if the two, as a model, provided greater explanation of the variance in the MIRI-SF Total Score than each individually. The results indicated that the model was significantly associated with recovery. Results are summarized in Table 5.15.

To clarify which of the main baseline variables contributed the most to predicting recovery, an initial stepwise regression analysis was performed involving Age, DAS 5, IDD Total, T1 MIRFI Adjusted Total, T1 MIRI Chest Pain Index, and the

Table 5.15
Simultaneous Regression for Baseline MIRFI Factor 5 and the IDD Total Score on Recovery

Model	Association		ANOVA				Coefficients				
	R^2	Adj. R^2	SS	F	df	p	B	SE B	β	t	p
T1 MIRFI Factor 5, IDD Tot.	.241	.185	1270.25	4.30	(2,27)	.024					
T1 MIRFI Factor 5							-.960	.751	-.255	-1.28	.212
IDD Total Score							-.343	.226	.304	-1.52	.140

Note: T1 MIRFI Factor 5=Baseline MIRFI Social Support Subscale
IDD Total Score=Baseline Primary Measure of Depression

T1 MIRFI Factor 5 (Social Support). The analysis indicated that the baseline MIRFI Factor 5 (Social Support) remained as the best predictor of recovery. At first glance, this finding seems confusing given that the IDD was shown to be the most highly correlated variable with the recovery measure (r=-.442) with the MIRFI Factor 5 coming in a close second (-.420) for the follow-up sample (N=30). However, when the sample is limited to *only married and cohabitating individuals that participated in follow-up assessment* (N=21), the MIRFI Factor 5 emerges as the most highly correlated with the recovery measure (r=-.434) versus the IDD (r=-.358). Because the DAS was included in this regression analysis, only participants responding to the DAS *and* the follow-up measure of recovery were included; that is, married and cohabitating participants within the follow-up sample. As a result, the MIRFI Factor 5 (Social Support), due to its prominent correlation with the recovery measure within this limited segment of the follow-up sample, emerged as the best predictor of recovery. Results are summarized in Table 5.16.

To investigate the best predictors of recovery among the baseline measures without limiting the analysis to only married and cohabitating individuals, the DAS was excluded from the stepwise regression. Accordingly, Age, T1MIRFI Adjusted Total, T1 MIRFI Factor 5 (Social Support), T1 MIRI Chest Pain Index, and the IDD were all

Table 5.16
Stepwise Regression for Age, DAS ODA, IDD Total Score, Baseline MIRFI Adjusted, MIRFI Soc. Support, and Baseline MIRI Chest Pain Index on Recovery

Remaining Model	Association		ANOVA				Coefficients				
	R^2	Adj. R^2	SS	F	df	p	B	SE B	β	t	p
T1 MIRFI Factor 5	.290	.249	1012.26	6.96	(1,17)	.017	-1.91	8.47	-.539	-2.64	.017
Excluded Variables: Age											
DAS ODA Score											
IDD Total Score											
T1 MIRFI Adjusted											
T1 MIRI Chest Pain Index											

Probability of F to enter: <.50. Probability of F to remove: >.10
Note: T1 MIRFI Factor 5=MIRFI Social Support Subscale at Baseline

entered into a stepwise regression with the recovery measure (MIRI-SF) as the dependent variable. The results, summarized below (Table 5.17), are consistent with correlational data placing the IDD as the baseline variable most strongly correlated with recovery.

Table 5.17
Stepwise Regression for Age, IDD Total Score, Baseline MIRFI Adjusted, MIRFI Soc. Support Subscale, and Baseline MIRI Chest Pain Index with Cardiac Recovery as Dependent Variable

Remaining Model	Association		ANOVA				Coefficients				
	R^2	Adj. R^2	SS	F	df	p	B	SE B	β	t	p
IDD (Baseline Depression)	.277	.248	1313.66	9.57	(1,26)	.005	-.584	.189	-.526	-3.09	.005
Excluded Variables: Age											
T1 MIRFI Adjusted											
T1 MIRFI Factor 5											
T1 MIRI Chest Pain Index											

Probability of F to enter: <.50. Probability of F to remove: >.10
Note: IDD Total Score=Baseline Depression
T1 MIRFI Adjusted=Baseline gauge of overall psychosocial risk (excludes Marital Satis. subscale)
T1 MIRFI Factor 5=Baseline Social Support subscale of the MIRFI
T1 MIRI Chest Pain Index=Baseline gauge of disease severity

Time 2 Variables as Predictive Models of Recovery

In an effort to glean the model that best predicts recovery among the Time 2 measures, the variables demonstrating the highest correlation with the recovery measure

(MIRI-SF Total Score) were entered into a stepwise regression procedure. The analysis resulted in two models that demonstrated significant predictive ability. In the first, the MIRI Chest Pain Index administered at follow-up was shown to be significantly linked with the recovery measure. The second model demonstrated that the MIRI Chest Pain Index, in concert with the Gauge of Depression, were the most predictive model among the variables entered into the stepwise regression analysis. Results are summarized in Table 5.18.

Table 5.18
Stepwise Regression for Key Follow-Up Measures:
MIRFI Factor 2, MIRFI Adjusted Score, Gauge of Depression, and MIRI Chest Pain Index

Remaining Models	Association		ANOVA				Coefficients				
	R^2	Adj. R^2	SS	F	df	p	B	SE B	β	t	p
Model 1: T2 MIRI Chest	.620	.607	3263.03	45.72	(1,28)	<.001	2.07	.306	.788	6.76	<.001
Model 2: T2 MIRI Ch. and	.795	.607	4181.64	52.28	(2,27)	<.001	1.82	.235	.693	7.76	<.001
T2 Gauge of Dep.							4.51	.941	.428	4.79	<.001

Excluded Variables for Model #1:	Excluded Variables for Model #2:
T2 MIRFI Factor 2 (Depression)	T2 MIRFI Factor 2 (Depression)
T2 Gauge of Depression	T2 MIRFI Adjusted Total Score
T2 MIRFI Adjusted Total Score	

Probability of F to enter: <.50. Probability of F to remove: >.10
Note: T2 MIRI Chest=MIRI Chest Pain Index at Follow-Up
T2 Gauge of Depression=Measure of Depression at Follow-Up (exploratory)
T2 MIRFI Factor 2=MIRFI Depression Subscale at Follow-Up (exploratory)
T2 MIRFI Adjusted=Risk Factor Index Total (excludes Marital Satisfaction Subscale) at Follow-Up

The MIRFI as a Screening Instrument for Depression and Marital Distress: Preliminary Validation

The MIRFI Factor 1 (Marital Satisfaction Subscale) as a Screen of Marital Distress

The Association Between the MIRFI Factor 1 & the DAS

See Appendix C for a summary matrix of the following correlations. Please note, these correlations were generated using the baseline scores for the subjects participating in follow-up assessment (N=30), not the entire baseline sample of N=53.

A Pearson Correlation (2-tailed) was performed between the MIRFI Factor 1 (Marital Satisfaction) at baseline and the DAS Overall Dyadic Adjustment Scale for the married and cohabitating participants in the baseline sample. A significant negative correlation was indicated ($r=-.777$, $p<.001$) suggesting that the five questions comprising the MIRFI Factor 1 (Marital Satisfaction subscale), as an index, correlate significantly with its parent instrument, the Dyadic Adjustment Scale. It should be noted that, unlike the DAS, the MIRFI's questions are stated such that higher scores equate with lower satisfaction. The DAS is designed in the opposite manner in that higher scores reflect higher degrees of satisfaction. Hence, the negative correlation between the two instruments.

The baseline MIRFI Factor 1 also correlated significantly with the DAS Dyadic Consensus Subscale ($r=-.712$, $p<.001$), the DAS Dyadic Satisfaction Subscale ($r=-.806$, $p<.001$), and the DAS Affectional Expression Subscale ($r=-.570$, $p<.001$).

Time 2 MIRFI Factor 1 & Baseline DAS

The MIRFI Factor 1 administered at Time 2 Follow-Up also correlated negatively with the DAS Overall Dyadic Adjustment scale administered at baseline ($r=-.437$, $p<.05$). Additionally, the MIRFI Factor 1 administered at Time 2 Follow-Up correlated with the DAS Dyadic Consensus Subscale ($r=-.468$, $p<.05$), and the DAS Dyadic Satisfaction Subscale ($r=-.463$, $p<.05$).

The MIRFI Factor 2 (Depression Subscale) as a Screen of Depression

The Association Between the Baseline MIRFI Factor 2 & the IDD

See Appendix C for a summary of the following correlations. A Pearson Correlation Coefficient (2-tailed) was obtained for the MIRFI Factor 2 (Depression) at baseline and its parent measure, the Inventory for Diagnosing Depression (IDD) at baseline. Results indicated a significant positive correlation between the two variables ($r=.721$, $p<.001$). It should be noted that for both measures, higher scores equate with

a greater degree of depression. hence the positive relationship. These preliminary results suggest that the MIRFI 2 may be an approximate gauge of depression.

The MIRFI as a Predictor of Cardiac Recovery

To address the role of the MIRFI administered at baseline as a predictor of cardiac recovery 5 to 8 months post-hospitalization. exploratory regression analysis was conducted utilizing the three subscales of greatest empirical and theoretical interest for the current study: MIRFI Factor 1 (Marital Satisfaction): MIRFI Factor 2 (Depression subscale): and MIRFI Factor 5 (Social Support subscale). Results are summarized in Table 5.19 below.

Table 5.19
Simultaneous Regression for MIRFI Factors 1, 2, and 5 at Baseline on Recovery

Model	Association		ANOVA				Coefficients				
	R^2	Adj. R^2	SS	F	df	p	B	SE B	β	t	p
T1 MIRFI Factors 1, 2, 5	.387	.279	1537.86	3.58	(3,17)	.036					
T1 MIRFI Factor 1							2.09	1.05	.449	1.98	.064
T1 MIRFI Factor 2							1.45	1.07	.332	1.35	.195
T1 MIRFI Factor 5							-3.31	1.04	-.889	-3.19	.005

Probability of F to enter: <.50 Probability of F to remove: >.10
Note: T1 MIRFI Factor 1=Baseline MIRFI Marital Satisfaction subscale
T1 MIRFI Factor 2=Baseline MIRFI Depression subscale
T1 MIRFI Factor 5=Baseline MIRFI Social Support subscale

CHAPTER V: DISCUSSION

Summary of the Research Problem & Methods

Review of the Research Problem & Objectives

The purpose of the current study was to further investigate the role of psychosocial factors in recovery post-hospitalization for myocardial infarction and unstable angina. The primary mission of the project was to build upon previous research which has suggested that depression and marital conflict may be toxic to the cardiac patient in recovery post-hospitalization. Specifically, the current study sought to examine the relationship between depression, marital satisfaction, and cardiac recovery in the 5 to 8 months post-hospitalization for myocardial infarction and unstable angina.

A secondary objective was the exploratory investigation of other psychosocial variables also reported in the literature as potentially harmful to cardiac functioning and recovery after a cardiac event.

To better clarify the linkage between psychosocial functioning and cardiac pathophysiology, the author developed an overarching causal pathway model of coronary heart disease in which key psychosocial variables such as depression and marital distress are delineated as mediators of the disease process and potential obstacles to successful recovery. Growing from the CHD causal pathway model was the concept for a self-report measure to be used as a screening instrument for several psychosocial factors put forth in the literature as potential mediators of CHD and critical cardiac events (Marital Distress, Depression, Type A Personality, Hostility, and Social Support). The *Myocardial Infarction Risk Factor Inventory* (*MIRFI*) was constructed and used for this purpose in the current study. Preliminary validation of the Marital Satisfaction and Depression subscales of the MIRFI was an exploratory component of the project.

Finally, exploratory analysis was performed on both the primary (depression and marital distress) and secondary (Type A, Hostility, Social Support) variables to determine which factors seemed most predictive of cardiac recovery in the 5 to 8 months post-hospitalization.

Review of Methods

The original sample was comprised of 53 male patients hospitalized in a Veteran's Affairs Medical Center for myocardial infarction and unstable angina. Participants were assessed for depression, marital satisfaction and the secondary psychosocial variables while in the hospital and again 5 to 8 months post-discharge via phone interview. Due to the unexpected inaccuracy of the VA computer system (wrong addresses and phone numbers), the researcher had difficulty locating many of the baseline participants at follow-up. As a result, a total of 30 patients participated at Time 2 follow-up.

Due to the small sample size (thus limiting the ability to group by demographic or psychosocial variables), as well as the overarching research problem, correlation and multiple regression were the primary statistical analyses used to address the nine research questions posed *a priori*.

Review of Demographic Data

The Sample at Baseline

The baseline sample of 53 male patients was comparable to those utilized in previous research in terms of central demographic variables (i.e., age, race, and income level). Had the sample been much larger, it would have been preferable to include both male and female participants from divergent racial groups. However, given the relatively small sample of the current study, the use of only male patients from one racial group may have allowed for better control of these factors. The majority of the baseline group (approx. 61%) were either unemployed or retired. Given the mean age

of 57.9 years. this statistic seems realistic. Of the 53 participants at baseline. 60% identified themselves as non-smokers. while 40% indicated that they were currently smokers at the time of their hospitalization. The mean number of years of school (11.83) suggests that the majority of participants attended at least a few years of high school. and over 71% currently live in the city/suburbs versus 28% residing in rural areas.

Of particular import is the relatively high rate of participants falling within the 0-$15.000 (N= 30. 56.6% of total N=53) and 16 to $30.000 (N=17. 32.1%) annual income groups. Collectively. almost 90% of the baseline sample earned below $30.000 annually as a family suggesting that the sample was comprised of low to moderate SES participants. Lower income has been implicated in higher incidence of depression. as well as poorer general health. Therefore. SES may have contributed to both the relatively high rate of depression in the sample (as will be discussed below). as well as potentially influencing recovery. The relative homogeneity of the sample in terms of SES. however. may have allowed for better control of economic differences within the sample. In all. the current sample seems analogous to those employed in previous research. especially those utilizing VA Medical Center populations.

The Follow-Up Group

Since the main recovery measure (MIRI-SF) was only administered at Time 2 (5 to 8 months post-hospitalization). the examination of the research problems focused only on the 30 participants who were assessed at baseline and Time 2. Therefore. in terms of statistical analysis of the research problems. the Time 2 sample is the focus.

Key Findings

The Relationship Between Depression at Baseline and Recovery

As stated in Chapter I and II. depression has been implicated in poorer cardiac recovery in the months following hospitalization. and is a major focus of this study.

Results indicated that depression at baseline, as measured by the IDD, significantly correlated with recovery 5 to 8 months post-hospitalization such that higher degrees of depression were associated with poorer recovery as measured by the MIRI-SF. These findings support notable studies which found depression at baseline to be associated with recurrent cardiac events and mortality (Carney et al., 1988; Carney et al., 1993; Frasure-Smith et al., 1993 & 1995). As was discussed previously, it has been postulated that depression may jeopardize cardiac recovery by negatively impacting behavior (e.g., poor treatment compliance and substandard health behaviors), psychosocial functioning (promoting isolation, poor social/emotional support), and physiology (promoting arrhythmia and ventricular fibrillation). While the above findings do not speak to causal linkages between depression and cardiac recovery, they seem to add additional support to the claim that a relationship exists.

In contrast to the above results, those participants meeting DSM-IV criteria for Major Depression on the IDD did not differ from non-depressed participants on the Time 2 measure of recovery. It is possible that a small sample size at Time 2 (N=30), and an even smaller group of depressed patients at Time 2 (N=6) may have dramatically reduced the statistical power needed to detect an effect.

The MIRFI Factor 2 (Depression) was used as an exploratory measure of depression at baseline. However, it was not shown to correlate significantly with the primary measure of recovery at 5 to 8 months post-hospitalization. Although the MIRFI Factor 2 at baseline was shown to strongly correlate with the IDD, it failed to demonstrate the same relationship with recovery as did its parent measure. It seems plausible that the MIRFI Factor 2 (comprised of only five items) does not effectively capture the same range of depressive symptomatology as does the IDD. For example, the MIRFI Factor 2 includes only one item related to physical symptoms of depression, while the IDD contains at least eight items that address physical symptoms. Therefore,

it is possible that a dimension exists within the IDD that relates the measure to recovery that the brief MIRFI Factor 2 fails to reproduce.

Previous research has relied upon self-report measures such as the IDD and Beck depression Inventory (BDI) to assess depression. This author wonders if the *physical symptoms of depression* (e.g., lack of energy, slower gross and fine motor activity, decreased libido, sleep changes, weight fluctuation, poor appetite, concerns about physical health) contained within these are greatly responsible for the statistical relationship between self-reported depression and CHD, MI, and cardiac-related mortality. Many, if not all, of the symptoms listed above are likely to emerge in patients hospitalized for advanced heart disease or MI. Others have argued this point as well. Specifically, it has been postulated that severe medical illness, and the associated physical symptoms, may mimic the somatic symptoms of depression (Jefferson, 1985; Kuhn & Schwab, 1987; Wells et al., 1993). Inversely, it has also been proposed that the physical aspects of illness may may mask an underlying depression. Is it not possible, then, that the MIRFI 2 (at baseline) did not relate significantly to recovery, while the IDD did, because the MIRFI contained *only one item* addressing physical symptoms, while the IDD contained at least eight? The question remains, therefore, Is there truly a relationship between depression and recovery that transcends the apparent statistical correlation between the commonly used self-report measures of depression and physical symptoms of heart disease? Or, put another way, are heart patients that experience poorer recovery reporting a mood disturbance, or simply numerous physical complaints that may resemble depressive symptoms? Needless to say, much additional research is necessary to clarify which aspects of depression are most strongly linked to cardiac recovery. Such insight might illuminate just how the MIRFI Factor 2 is lacking.

Prevalence of Depression Within the Baseline Sample

Of the 53 participants at baseline, 26.4% were identified as meeting DSM-IV

criteria for Major Depression based upon their responses to the *Inventory for Diagnosing Depression* (IDD). This percentage is consistent with previous reports placing the rate of depression among medical inpatients at 22-33% (Levinson et al., 1987). The current sample's rate of depression also seems comparable to previous research citing the incidence of depression among patients diagnosed with coronary heart disease as approximately 20% (Carney et al., 1987; Vasquez-Barquero et al., 1985). Depression among post-myocardial infarction patients has reportedly ranged from 18-33% (Kavanagh et al., 1975; Schleifer et al., 1989), which is also comparable to the rate found within the current sample.

Most striking, however, is the rate of depression among the current sample relative to the general adult population, which reportedly has a 2-4% rate of depression (DIS; Robins, Helzer, Croughan, & Ratcliff, 1981; *DSM-IV*; APA, 1994). Simply put, these results (consistent with previous research) indicate a rate of major depression among hospitalized heart patients as nearly six times greater than the general adult population. It has been argued that patients suffering from acute cardiac events experience greater psychological stress and lifestyle disruption than healthy adults, and as a result, are at much greater risk for developing depressive symptoms.

Past research has indicated that the course of depression is more destructive among patients hospitalized for myocardial infarction than many other populations of depressives (Wells et al., 1993). Moreover, both the current study and previous research have linked depression to poorer recovery. Given both the high rate of depression, and its negative impact on health behaviors and rehabilitation efforts, it would seem that effective detection and treatment of depressive symptoms deserve high priority for those working with heart attack and unstable angina patients. The strong psychosocial component contributing to depression among heart patients (i.e., change in lifestyle, stress, mortality issues, marital and familial disruption etc.), as well as the

empirical validation of psychological treatment of depression, support the inclusion of psychological services in cardiac rehabilitation programs. Health Psychologists may prove useful in efforts to screen, detect, and treat depression and other psychosocial correlates of acute cardiac illness that threaten the emotional and physical well-being of the heart patient.

The Relationship Between Follow-Up Measures of Depression and Recovery

Results of the current study indicate that the two measures of depression at Time 2 (MIRFI Factor 2 and the Gauge of Depression) each correlated significantly with the recovery measure at Time 2. Exploratory stepwise regression analysis of the four MIRFI subscales at Time 2 (MIRFI Factors 2: Depression; 4: Hostility; 5: Social Support; and the Adjusted Total Score) that correlated significantly with the recovery measure (MIRI-SF) resulted in the depression subscale emerging as the sole member of the model for predicting recovery.

While both the MIRFI Depression Factor and the follow-up Gauge of Depression were constructed by the author as exploratory instruments used to replace the more cumbersome IDD in an already taxing Time 2 assessment battery, both the MIRFI Factor 2 (administered at Time 2) and the Gauge of Depression significantly correlated with the IDD at baseline. As stated in a previous section, the MIRFI Factor 2 lacks psychometric validation, and may not fully tap the breadth of depressive symptomatology that its parent measure, the IDD, does. Moreover, the Gauge of Depression, being a single Likert question, is certainly psychometrically questionable. Nevertheless, it is worthy of note that a relationship between the Time 2 depression measures and cardiac recovery was established.

It is unclear why the MIRFI Factor 2 *at baseline* did not correlate with the recovery measure at Time 2, while the MIRFI Factor 2 at *Time 2* did. It is possible that

the elements of depression addressed by the MIRFI Factor 2 are more relevant to recovery 5 to 8 months out than during the hospitalization phase.

First, it is important to note that the IDD, the primary measure of depression at baseline, was found to correlate significantly with the recovery measure. This result seems to support that depressive symptoms of one kind or another were being reported by hospitalized patients. Hence, the argument that the MIRFI Factor 2 at baseline did not associate with the recovery measure due simply to a lack of depressive symptomatology among hospitalized patients seems unfounded. However, one potential explanation for the discrepancy between the baseline and follow-up MIRFI depression subscale is that physical symptoms are more salient for the heart patient at the time of hospitalization than psychosocial issues. This seems a logical premise since the unstable heart patient, by definition, is admitted due to extreme and frequent chest pain and associated symptoms (nausea, weakness, fatigue). Upon arrival to the CICU, the patient is then quizzed by medical personnel as to the frequency, duration, location, and severity of his chest pain. Needles and IV's are inserted, the chest is shaved and heart monitor electrodes are attached, vital signs are taken at the half hour. In short, the heart patient typically undergoes considerable poking and prodding from the minute they enter the unit. Therefore, it makes intuitive sense that hospitalized patients are more focused on physical symptoms and the flurry of activity following the cardiac event, while the emotional impact of their situation may not be fully realized until the crisis subsides weeks or months later. Given the MIRFI Factor 2's emphasis on affective components of depression and the near exclusion of physical features, the measure may simply elicit lower responses from hospitalized (baseline) participants because the measure is geared toward subjective feelings of depression, while the patient is absorbed by his physical well-being and the adjustment to an inpatient hospital stay. At 5 to 8 months post-hospitalization, a more affective response to poor recovery may be more likely as the dust settles from the event and the reality of the

situation sets in (pain, lifestyle disruption, familial and marital stress etc.).

A final and related thought is that depression at Time 2 (as measured by the MIRFI Factor 2) may relate to the recovery measure more as a *marker* of poorer physical recovery than as a *causal agent* and/or predictor of poor recovery. Specifically, patients who have not progressed (or have even declined) across the 5 to 8 months post-discharge may be more prone to a depressive reaction to their situation. Put simply, poor physical recovery over several months may take an emotional toll on the patient and his family. Moreover, the shock and denial of the precipitating cardiac event and hospitalization that once numbed affect has likely given way to the reality of the situation with the passing of several months. Enter the risk for a depressive reaction. While offered only as a tentative and explanation of the data, it seems a plausible accounting of the association between the follow-up depression measure and recovery. A great deal of research is necessary to better understand the causal pathway linking depression and recovery. An item analysis of depression measures such as the MIRFI Factor 2 might assist in the identification of which aspects of depression (items on a measure) are tapped during the hospitalization phase, and which are more likely to be endorsed at follow-up. Specifically, it might prove beneficial to clarify if the more physiologically-based items within the self-report measures of depression (like the IDD and BDI) are consistently rated higher during hospitalization (allegedly reflecting a patient's emotional shock and focus on bodily functions and survival), while the more affectively-laden items of depression are rated higher in the months that follow. Additionally, it would prove interesting to explore how the self-report measures of depression would correlate with recovery if the physiological component was removed from the instrument. Each of these proposed investigations fall well beyond the scope of the current project and, as such, must for now remain recommendations for future research.

Myocardial Infarction and Unstable Angina:

A Comparison on Key Variables

To the knowledge of the author, no other study examining psychosocial correlates of cardiac recovery has combined post-myocardial infarction and unstable angina patients in the same sample. In fact, doing so was not the original intent for the project. As in previous research, the author initially sought to examine only post-myocardial infarction patients, but due to a variety of issues, it was felt that their inclusion would not hinder the goals of the study, and might actually enhance its contribution to the literature by providing a comparison of two very similar populations of heart patients on key psychosocial variables.

With the inclusion of both post-MI and unstable angina patients, it was necessary to explore their comparability on the primary variables in question so that, when combined, statistical analysis and the interpretation of findings could be generalized across the two groups. Results indicated that mean scores for post-MI (N=22) and unstable angina patients (N=31) comprising the baseline sample (N=53) did not differ significantly on any of the central independent variables (i.e., the IDD, DAS, MIRFI Adjusted Total, MIRFI Total Index, and the MIRI Chest Pain Index), or the primary measure of recovery (MIRI-SF). In sum, from a statistical perspective, those hospitalized for heart attack and unstable angina seemed comparable in terms of psychosocial functioning at baseline and recovery 5 to 8 months post-discharge.

The above findings seem to support the notion that the MI and UA patient share many of the same experiences related to their illness. From a psychosocial perspective, they both suffer almost daily pain, are often forced to restrict social, vocational, and leisure activities, and to modify health behaviors (i.e., diet, smoking, exercise, etc.). Many MI and UA patients struggle with the adjustment from self-reliant provider to one who is dependent upon others for many daily functions. Additionally, the hospitalized

MI and UA patient are treated almost identically on the Cardiac Intensive Care Unit (CICU). There is little distinction in protocol in terms of the admission process, diagnostic procedures, cardiac monitoring, medications, and options for treatment. In short, the results may suggest that the UA patient, simply because he has not infarcted, is not buffered from the psychosocial fallout that many have previously reserved for the heart attack victim. Certainly, MI and UA are, in many respects, different physiological events. However, it would appear from this preliminary investigation that the two are relatively similar with respect to several key psychosocial variables highlighted in the literature as correlates of heart disease.

It should be reiterated that the psychosocial comparison of MI and UA patients was not a central objective of the current study, and was undertaken solely as an exploratory venture. Future research is necessary to clarify the psychological dynamics of the two diagnoses. Nevertheless, the current results may begin to portray the unstable angina patient in a different, potentially less optimistic light in terms of the psychosocial impact of his illness. If subsequent research supports the preliminary findings of the current project, it is plausible that the patient hospitalized for unstable angina could benefit from psychosocial interventions typically reserved for those having suffered a heart attack.

Additional Findings

The Role of Marital Satisfaction in Recovery

Baseline Measures of Marital Satisfaction as They Related to Recovery

Only 2 participants at baseline (6.3%) reported significant marital distress as determined by T-score cutoffs for the *Dyadic Adjustment Scale* (DAS). Additionally, the mean DAS Overall Adjustment Scale (the primary measure of marital satisfaction/distress) of 116.80 places the sample, as a group, at a T-score of 51, or within the average range of marital adjustment. Even in light of the small sample size, the proportion of distressed

marriages appears rather low. As will be discussed below, the limited scatter within the DAS rendered experimental analysis of the variable impossible, and likely thwarted its correlation to key variables...including recovery.

Neither the primary (DAS and its four subscales) nor secondary (MIRFI Factor 1) measures of marital satisfaction at baseline were significantly correlated with the recovery measure at Time 2. The four subscales of the DAS were entered as a four-predictor regression model with the recovery measure (MIRI-SF) as the DV to investigate the instrument's association with the recovery measure. The model did not significantly predict recovery. These findings do not support the mostly theoretical linkage between marital satisfaction and cardiac recovery.

There are several issues that may have contributed to the above findings. One explanation that must be considered is that marital satisfaction, as measured by the DAS, simply does not relate to cardiac recovery as assessed by the MIRI-SF. If this were the case, innumerable reasons could be conceived as to why the responses to the two instruments led to insignificant findings. One could postulate that aspects of one or both of the measures, or some aspect of the assessment process itself, hampered accurate measuring of marital functioning at baseline.

An alternative explanation might be that, within this sample of 53 male heart patients, the incidence of marital distress was truly low, thus reducing variability in the DAS Overall Adjustment Scores.

A third issue that deserves consideration is that the sample of married and cohabitating patients at baseline was relatively small (N=32). With such a small sampling, it seems likely that variability among DAS scores could well have been adversely impacted, thus reducing the statistical power for detecting a trend between marital satisfaction and cardiac recovery.

Finally, one might hypothesize that heart patients hospitalized for a life

threatening cardiac event, such as MI or unstable angina, are less willing or able to report marital difficulties than the population at large. Similar to the above discussion concerning the reporting of depression at baseline, it is possible that while in the midst of a crisis situation, individuals are more focused on survival and physical functioning than emotional and interpersonal issues. Additionally, the onset of denial is not uncommon as a means for coping in the early stages of a traumatic event. Often individuals will resist the acceptance of the severity of the situation, as well as collateral problems, to assist in their adjustment to an event that is out of the norm of their daily experience. The literature supports the claim that emotional shock and denial of this sort are relatively common among heart attack victims in the hours and days following the event (Blumenthal, 1982; Smith & Leon, 1992). Some have postulated that denial may actually aid in the medical stabilization of the patient in the days after hospitalization (Blumenthal, 1982; Levenson et al., 1989; Levine, et al., 1987). If present in the hospitalized sample of the current study, denial would likely lead to the underreporting of emotional unrest, and contribute to insignificant findings.

With the above considerations in mind, future research might benefit from the use of both larger samples, and more than one *psychometrically sound* instrument for assessing marital satisfaction. The current study was too reliant upon a single measure, and the use of exploratory instruments. In addition, future efforts should consider the impact of assessing emotional and marital functioning during the hospitalization phase. The potential for underreporting of negative symptoms may be fostered by the affective numbing and/or denial components of coping in the hours and days following a traumatic event. It may be advisable, as some have done in previous studies, to wait until discharge to assess the heart patient's emotional and relationship functioning.

Follow-Up Measures of Marital Satisfaction as They Related to Recovery

Neither of the measures of marital satisfaction at Time 2 (the MIRFI Factor 1

and the Gauge of Marital Satisfaction) were significantly correlated with the primary recovery measure (MIRI-SF). Both measures were exploratory instruments designed to replace their parent instrument, the DAS, in an effort to reduce the complexity of the Time 2 follow-up procedure. It was felt that maximizing participation at follow-up and minimizing participant fatigue/resistance took priority over the inclusion of the more cumbersome DAS. This decision was bolstered by the notion that, at Time 2, a valid assessment of recovery was the primary objective, and efforts to streamline the phone interview follow-up procedure were necessary. As a result, marital satisfaction at Time 2 was solely reliant upon instruments lacking psychometric validation and known reliability. While many of the same arguments concerning the assessment of marital functioning at baseline hold true at follow-up (e.g., small sample size: N=21 at follow-up), the lack of sound and reliable measures at Time 2 may have further weakened the investigation into marital satisfaction and cardiac recovery. Unfortunately, all of the above severely limit the interpretability of the negative findings.

Future research utilizing psychometrically sound instruments and adequate sampling is needed to clarify the role of marital functioning in recovery at 5 to 8 months post-hospitalization.

The Relationship Between Depression and Marital Satisfaction

Baseline Measures of Depression and Marital Satisfaction

The primary baseline measures of marital satisfaction (DAS Overall Dyadic Adjustment score and the IDD) correlated significantly with one another supporting findings of previous research that the two entities are often associated. Specifically, as marital satisfaction decreases, the reporting of depressive symptomatology tends to increase. In addition, the exploratory measure of depression at baseline, the MIRFI Factor 2, also correlated significantly with the DAS and one of its subscales (Subscale I: Dyadic Consensus) offering additional evidence of a relationship.

Follow-Up Measures of Depression and Marital Satisfaction

The instruments used to assess depression (MIRFI Factor 2) and marital satisfaction (Gauge of Marital Satisfaction) at follow-up were found to correlate significantly with one another suggesting that, 5 to 8 months post-hospitalization, the relationship is maintained.

In sum, it would appear that results from the current project support claims made in previous research that depression and marital satisfaction are often associated. This finding is especially intriguing in that, as has been discussed, there appears to be relatively little reporting of marital distress among the baseline and Time 2 samples. It seems, therefore, that the association between depressive symptoms and marital functioning may exist even within less extreme ranges of the variables. Put another way, it appears that the link between depression and martial satisfaction may persist in the absence of more severe symptomatology.

The Interaction Between Baseline Measures of Depression and Marital Satisfaction: Implications for Recovery

Regression analysis of the primary baseline measures of depressions (IDD) and marital distress (DAS Overall Dyadic Adjustment) in relation to predicting recovery indicated that, as a two-predictor model, the tandem did not significantly explain the variance in the recovery measure (MIRI-SF) at Time 2. While depression at baseline was associated independently with recovery, it would appear that the poor correlation between marital distress and the recovery measure rendered the model ineffective as a predictor. The inter-correlation between the IDD and DAS may have also reduced the predictive power of the model.

The secondary baseline measures of marital satisfaction (MIRFI Factor 1) and depression (MIRFI Factor 2) were also shown to be non-significant predictors of recovery. This finding is not particularly surprising given the poor respective

correlations between the two baseline variables and the recovery measure.

The Interaction Between Follow-Up Measures of Depression and Marital Satisfaction: Implications for Recovery

The exploratory measures of martial satisfaction and depression at Time 2 (MIRFI Factors 1 and 2) were entered as a two-predictor regression model to determine if the two variables combine to adequately explain the variance in the recovery measure. Results indicated that, indeed, the follow-up MIRFI factors significantly predicted recovery. Similar significant findings emerged when the Gauges of Depression and Marital Satisfaction were analyzed as a two-predictor model in relation to the recovery measure.

Worthy of note is the fact that neither Time 2 measure of marital satisfaction was independently correlated with the recovery measure, while each of the Time 2 depression measures was strongly associated with recovery. Moreover, the regression analysis data plainly demonstrate that the depression variables in each of the two models carried the bulk of the association with the recovery measure. Therefore, it would seem for the current sample that depression was the most powerful predictor of recovery, and that the interaction between depression and marital satisfaction did not enhance this predictive association.

Models Among the Key Variables that are Predictive of Recovery

Predictive Models Among Baseline Variables

Correlational analysis among baseline measures indicated that only the IDD and MIRFI Factor 5 (Social Support) were significantly associated with the recovery measure. To address the predictive power of these variables in concert with the primary baseline measure of marital satisfaction (DAS Overall Adjustment), the three were investigated via regression analysis as a three-predictor model. As a baseline threesome, the model was not significantly associated with recovery. In an effort to

counteract the potentially damaging effects of the inclusion of the DAS (a variable found to be poorly associated with the recovery measure in the current sample) in a regression model, only the IDD and MIRFI Factor 5 were analyzed as a two-predictor regression model. Results indicated that this model was significantly associated with recovery suggesting that among the baseline variables, primary measures of depression and social support were, independently and cooperatively, the best predictors of recovery.

An overarching investigation of several primary baseline variables was undertaken to clarify which was the most predictive of recovery. Among age, the DAS Overall Adjustment scale, the MIRFI Adjusted Total Score (gauge of psychosocial risk factors), the MIRI Chest Pain Index (gauge of disease severity), and the MIRFI Social Support scale, the MIRFI Social Support scale emerged as the most strongly associated with recovery via a stepwise regression procedure. However, as noted in the "Results" section, this regression involved *only* those participants within the follow-up group who had taken the DAS: that is married and cohabitating individuals. Within this limited segment of the follow-up group (N=21 vs. N=30 for the entire follow-up group), the MIRFI Social Support subscale was the baseline variable most highly correlated with the recovery measure. Hence, it emerged as the best baseline predictor of recovery *under these circumstances.* In contrast, when the DAS was not employed in stepwise regression analysis of these same baseline variables, and the entire N=30 follow-up group was involved, the IDD emerged as the best predictor of recovery among these key baseline variables. The reader will note that the correlations cited in Table 5.6 illustrate the IDD to be the strongest correlate with the recovery measure among the baseline measures. This correlation involves all members of the follow-up group (N=30). Therefore, this second regression analysis which, (a) excludes the DAS, and (b) identifies the IDD as the best single predictor of recovery, is consistent

with correlation data.

In sum, the current results suggest that among the major baseline variables, depression and social support were the two most strongly associated with the cardiac recovery. Moreover, among these two variables, depression assessed during hospitalization was the best single predictor of recovery. It should be noted that the MIRFI Social Support subscale (Factor 5) was created by the author as an exploratory measure of social support. In the absence of proper psychometric validation of the MIRFI subscale, interpretations of the results should be considered preliminary. Nevertheless, the finding is worthy of note since social support has gained much favor in the literature as one of the theoretical mechanisms linking psychosocial and physiological functioning (Coombs et al., 1992; Ross et al., 1990). The data from the current study may, in some small way, support this linkage.

Predictive Models Among Measures Administered at Follow-Up

Among the follow-up measures, the MIRFI Factor 2 (Depression), the MIRFI Adjusted Total, the Gauge of Depression, and the MIRI Chest pain Index were the most highly correlated with the recovery measure. To better understand which of these best predicted cardiac recovery, they were entered into a stepwise regression analysis. Results indicated that the MIRI Chest Pain Index (the primary gauge of disease severity), was the most strongly associated with the recovery measure among the key Time 2 variables.

It is not entirely clear why the chest pain index was most predictive of recovery in the Time 2 sample. One plausible explanation is its close association with its parent measure, the MIRI-SF. Being a subscale of the MIRI-SF, the Chest Pain Index certainly has statistical overlap with its parent measure that would likely enhance the correlation of the two. From a more theoretical standpoint, chest pain, while not an ideal estimation of disease severity, is regarded as a useful clinical tool for this purpose.

In some ways, chest pain is actually a gauge of recovery. Therefore, it seems logical that the MIRI Chest Pain Index is strongly associated with the MIRI-SF recovery measure.

These findings may reinforce the notion that gauging chest pain frequency, duration, and severity has real clinical validity in assessing disease severity and level of recovery. However, the fact that the *baseline* MIRI Chest Pain index was not correlated with the recovery measure may suggest that chest pain provides useful information as to the *current* state of functioning, but may be of little value for *predicting* future recovery.

The MIRFI as a Screening Instrument for Marital Satisfaction and Depression

It is important to reiterate that the examination of the MIRFI as a viable measure of marital satisfaction and depression was strictly exploratory. The central objectives for doing so were (a) to clarify if the two subscales (Factor 1 and Factor 2) related well enough to their parent instruments (the DAS and IDD respectively) to be used in a meaningful way in the analysis of marital satisfaction, depression, and cardiac recovery; and (b) to provide preliminary insight into the validity of the subscales, and to set the stage for later, more comprehensive validation of the MIRFI.

The MIRFI Factor 1 as a Measure of Marital Satisfaction

The MIRFI Factor 1, created as a marital satisfaction subscale, was shown to correlate significantly with its parent instrument, the DAS. More specifically, the MIRFI Factor 1 administered at baseline correlated strongly with the DAS Overall Dyadic Adjustment Scale, as well as the Dyadic Consensus, Dyadic Satisfaction, and Affectional Expression subscales.

These results suggest that within the current sample, the MIRFI Factor 1 was an adequate approximation of marital satisfaction as measured by its parent instrument, the

DAS. Interestingly, the MIRFI Factor 1 did not relate well to the Dyadic Cohesion subscale of the DAS suggesting that the items of the MIRFI subscale did not adequately address the common interests and activities aspects of marital functioning.

The MIRFI Factor 2 as a Measure of Depression

The MIRFI Factor 2 was designed as a measure of depressive symptomatology utilizing items from its parent instrument, the IDD. Correlational analysis indicated that the MIRFI Factor 2 administered at baseline was associated significantly with the IDD at baseline. The subscale at administered at Time 2 maintained a significant correlation with its baseline parent instrument. These findings suggest that the MIRFI Factor 2 may be an adequate gauge of depression as measured by the IDD. However, given that Factor 2 did not correlate with the recovery measure while its parent measure did, the subscale might be improved by increasing its coverage of depression by including additional physical symptoms.

Summary of Findings

While the current results offer limited support for the effectiveness of the MIRFI Factors 1 and 2 as measures of marital satisfaction and depression, much additional research is needed to more fully establish the psychometric validity of the MIRFI subscales. Future efforts should pit the MIRFI subscales against other accepted and valid measures of marital functioning and depression to determine if they generalize beyond their parent measures. In addition, item analysis of the MIRFI subscales in relation to empirically validated measures should illuminate the instrument's shortcomings, and promote the inclusion of items that most efficiently tap the dimension in question. At present, the MIRFI subscales are comprised of items that were selected on the basis of theory, past research, and face validity. While it appears that the marital satisfaction and depression subscales performed adequately in the current project, empirical validation is essential prior to the application of the MIRFI as

a measure of these or any other psychosocial phenomena.

The MIRFI at Baseline and Time 2 as a Predictor of Recovery: A Preliminary Investigation

The investigation into the value of the MIRFI as a predictive instrument relative to cardiac recovery was purely exploratory. Therefore, statistical analysis of the MIRFI was simplistic, and in no way should be considered as psychometric validation of the instrument. However, the data gleaned from this exploration represents a first step toward the validation of the MIRFI as a screen for psychosocial risk factors, as well as a predictor of cardiac recovery post-hospitalization. The reader will note that the majority of effort was expended on the baseline MIRFI and its association with recovery, for this relationship was most central to the overarching objective of this phase of the project...the use of a baseline MIRFI as a predictor of recovery.

The MIRFI at Baseline as a Predictor of Recovery

Correlational analysis between each of the MIRFI factors at baseline and the recovery measure at Time 2 resulted in only the MIRFI Factor 5 (Social Support) being significantly correlated with the recovery measure. Although the MIRFI Factor 2 (Depression) was strongly and significantly associated with its parent instrument, the IDD, and the IDD was found to correlate with the recovery measure, the MIRFI subscale did not produce a similar relationship. As discussed in a previous section, it is possible that the more comprehensive IDD taps a greater array of depressive symptomatology than does the MIRFI Factor 2. As a result, the MIRFI subscale at baseline may not be picking up on the dimension of depression relating to the recovery measure as did the IDD.

It is not entirely clear why the Social Support factor was the sole predictor of recovery among the baseline MIRFI subscales. Social and emotional support have emerged in the theoretical literature as potential mediators of illness (Jones et al., 1992;

Kohn et al., 1983; Ross et al., 1990). Many have postulated that having the practical and emotional support of those around us not only assists in maintaining health behaviors (diet, exercise, treatment regimens), but may have direct physiological benefit as well such as reduced stress and related cardiac processes (King et al., 1993; Syme & Seeman, 1983; Waltz, 1986; and Wills, 1991). This *protection/support hypothesis* (Ross et al., 1990) may be one plausible explanation of the current finding. Moreover, heart patients hospitalized for unstable angina and MI may require adequate emotional and social support to assist with the behavioral and psychosocial demands of their illness. In the absence of such, per the hypothesis, cardiac functioning and overall recovery may be jeopardized. In fact, it could be argued that the heart patient may be at greater risk than other medical patients due the volatile nature of their illness. Stress and poor treatment compliance can directly impact cardiac reactivity. For those with advanced CHD and substantial arterial blockage (a prerequisite for MI and unstable angina), such antagonists seem logical impediments to recovery. In sum, the current finding linking social support to recovery may offer some support for the *protection/support hypothesis.*

Particularly important to the question of the baseline MIRFI's relationship to recovery was the fact that the scales designed to represent an overall risk factor index at baseline (MIRFI Adjusted Total and MIRFI Total Index Score) were not significantly correlated with recovery. These results suggest that the primary gauges of overall psychosocial risk were not predictive of recovery 5 to 8 months post-hospitalization. In light of all but one subscale having no significant correlation with the recovery measure, it is not surprising that the overall index scores were not significantly associated with recovery.

In contrast, exploratory regression analysis indicated at least one regression model involving the baseline MIRFI subscales was significantly associated with

recovery. The baseline MIRFI Factors 1 (Marital Satisfaction), 2 (Depression), and 5 (Social Support), as a 3-predictor model, associated significantly with the recovery measure. See Table 5.19 for these results.

To summarize, the current findings suggest that the MIRFI may be of limited use in predicting cardiac recovery in its present form. However, exploratory analysis at baseline indicates that the relationship with recovery is strongest for a 3-predictor model comprised of Factors 1 (Marital Satisfaction), 2 (Depression), and 5 (Social Support). While the current results are preliminary, they may suggest that the predictive power of the MIRFI may be enhanced by: (a) modifying the items to maximize the subscales' comparability with more comprehensive and validated measures; and (b) limiting the instrument to only those psychosocial variables that showed promise as predictors of recovery. In the current sample, Factors 3 (Type A) and 6 (Treatment Compliance) showed little relationship with the recovery measure. Future research should seek to clarify which subscales seem most predictive, and Factors 3 and 6 should receive special scrutiny.

The primary objective of the exploratory analysis regarding the MIRFI's ability to predict recovery was to establish it as a potential screening instrument for psychosocial risk factors of heart disease and cardiac recovery. As a brief self-report questionnaire, the MIRFI can screen for the presence of six risk factors in approximately 5 minutes. Eventually, it is hoped that the MIRFI will be used by health care providers to aid in the detection and treatment of possible threats to cardiac functioning. The current study provided a piloting of the MIRFI, and preliminary data suggests that its Marital Satisfaction and Depression subscales relate well to their respective parent measures. Much additional validation research is needed to confirm the MIRFI's value as a screening instrument.

CHAPTER VI: SUMMARY & CONCLUSIONS

Summary

The primary mission of the current study was to explore the relationships among depression, marital satisfaction, and cardiac recovery 5 to 8 months post-hospitalization for myocardial infarction (MI) and unstable angina (UA). Secondarily, several other psychosocial variables (e.g., Type A, hostility, social support, and treatment compliance) were investigated in terms of their relationship to recovery. As an exploratory component of the project, the *Myocardial Infarction Risk Factor Inventory* (MIRFI) was examined as a screening instrument for depression and marital satisfaction, as well as a tool for predicting recovery post-discharge. Unique to this area of research, the current study combined MI and UA patients into the same sample. In so doing, their statistical comparability on key psychosocial and recovery measures was also explored.

The results of the current project suggested that while MI and UA are somewhat different physiological events, the two types of heart patients did not differ significantly on the central psychosocial and recovery measures. To the knowledge of the author, no previous study has sought to compare these diagnostic populations in this way, and the results may suggest that the UA and MI patient share many of the same experiences related to their illness, as well as a host of psychosocial characteristics. If future research substantiates the current findings, the UA patient may gain recognition as a population at the same risks as those suffering MI, and in need of the same rehabilitation services currently reserved for those who have infarcted.

Findings of the current study were consistent with previous research in demonstrating that patients suffering critical cardiac events have a much higher rate of depression than the general adult population. More specifically, 26.4% of the current sample of male heart patients met DSM-IV criteria for major depression, while rates for

the general population of adult males are estimated to range from 2-4%. These results provide additional support for previous findings, and emphasize the need for the effective detection and treatment of depression for heart patient populations. Accordingly, the inclusion of the Health Psychologist on the cardiac unit and rehabilitation teams may prove useful to maximize screening and treatment efforts.

The current results supported findings from previous research that depression at baseline was associated with poorer recovery in the months following hospitalization. This relationship also existed between depression and recovery at the time of follow-up. Marital satisfaction, however, was not shown to correlate with recovery either at baseline or at the Time 2 follow-up. The small number of married and cohabitating participants at Time 2, as well as the near absence of marital distress within the sample, may have contributed to the negative finding. In addition, the relationship between depression and marital functioning cited in previous research was also supported by the findings of the current project. Interestingly, when combined, depression and marital satisfaction were not significantly associated with cardiac recovery suggesting that, for the current sample, the two variables do not interact to influence recovery. The poor association between marital satisfaction variables and the primary recovery measure may have adversely effected the predictive value of the joint model by overshadowing the correlation between baseline depression and recovery.

Among the baseline variables, social support (as measured by the exploratory MIRFI Factor 5) and depression (as measured by the IDD) were independently, and as a two-predictor model, significantly associated with cardiac recovery. Of the two, depression emerged as the most strongly associated with recovery via stepwise regression.

While the MIRFI's Adjusted Total and Total Index Scores were not found to be sound correlates with recovery, the Social Support subscale was significantly

associated with recovery. Moreover, the Marital Satisfaction, Depression, and Social Support subscales, as a 3-predictor model, significantly predicted recovery. In sum, the MIRFI at baseline was not stellar in its prediction of recovery as a total instrument, however, the findings suggest that portions of it did associate significantly with the recovery measure. Additional research is necessary to clarify which psychosocial subscales should be incorporated into the MIRFI to maximize its predictive power.

The current findings did indicate that the MIRFI's Marital Satisfaction and Depression subscales correlated strongly with their parent instruments, the DAS and IDD respectively. Much additional validation research is needed to substantiate the psychometric value of MIRFI subscales. However, the current study suggests that in its preliminary form, the MIRFI's Factors 1 and 2 performed adequately as screens of marital satisfaction and depression.

Limitations and Suggestions for Future Research

As with most research, the current project has a number of limits affecting generalizability and interpretability of its findings.

The most critical shortcoming was the relatively small sample size at follow-up (N=30). Due to many problems securing follow-up participants, the total number of married and cohabitating subjects (N=21) made experimental analysis impossible, and seems to have contributed to little scatter among DAS scores. As a result, the exploration of marital satisfaction as it relates to recovery was severely impaired.

A second issue that limited the project was the lack of systematic control of disease severity. Ideally, this confounding variable would have been controlled for by the use of physiological gauges of CHD severity such as ejection fraction or cardiac enzymes. However, the institution from which the sample was collected does not generate ejection fractions for all patients. In fact, less than 25% of the sample had ejection fraction data at the time of discharge. Cardiac enzymes, while excellent for

diagnosing the occurrence of MI, are not helpful in dealing with the unstable angina patient. Since the unstable angina patient has not, by definition, suffered an infarct, cardiac enzymes linked to the death of myocardial cells are not typically elevated. Hence, cardiac enzymes were not a viable option for gauging disease severity. The authors were left, therefore, with chest pain characteristics as the only rough estimate of disease severity. Chest pain is regularly used by physicians and nursing personnel to approximate the patient's status, but typically in conjunction with other physiological data. Therefore, used in isolation, chest pain is not a reliable gauge of disease severity. The current study, and future research in the area, would benefit from obtaining accurate and reliable medical data on all participants to better control for disease severity, and utilize it in the analysis of predictors of recovery.

The current project did not control for medical procedures in any way. While this is not uncommon among studies in this area, factoring in whether a patient has undergone an invasive, corrective cardiac procedure would certainly allow for the control of this variable when examining correlates of recovery. Several of the more notable studies examining cardiac recovery have had the luxury of selecting post-MI patients with very similar degrees of disease severity and no invasive procedures post-hospitalization. Due to the time and financial constraints of the current project, inclusion criteria had to be much more broadly defined to ensure an adequate sample size. If there was a benefit to the use of a convenience sample, any trends and correlations found significant were detected amidst many confounding variables. Thus, for a link between a psychosocial variable and recovery to emerge, its association would likely have to be quite strong to overcome the noise of the many uncontrolled factors present in the current design.

Strengths of the Study

While the shortcomings listed above are significant, it is believed that they do

not overshadow the positive aspects of the project. Many of the limitations were the direct result of obtaining a real clinical sample with the inherent threats to research vigor therein. It is the opinion of this author that what was lost to rigor may be compensated for in relevance. The use of a clinical sample of heart patients should increase the generalizability of the findings significantly. In other words, the results from the study speak directly to the very population in question, thus facilitating the clinical application of key findings. Additionally, the use of very personalized data collection procedures (face-to-face interviews at baseline and phone call interview at follow-up) in lieu of mailings likely enhanced the reliability of the self-report data, and seemed to be a positive and reinforcing experience for the patients.

Finally, no study to date has provided a comprehensive model illustrating the theoretical causal pathways linking psychosocial constructs to cardiac functioning. The current study not only brought order to a rather fragmented literature base, but demonstrated how a causal pathway system might aid clinical work and research efforts. This model was the basis for the study, sparked the creation of a new risk factor inventory, and guided the empirical investigation. In short, the current project put into practice the very model it proposed. While the findings were limited and rigor was certainly compromised, in many ways the study demonstrated how psychological research must be theory driven and clinically applicable to be of practical utility.

The current project contributed to the literature by replicating key previous findings, as well as by breaking new ground in the arena. For example, the current project replicated findings from a small group of studies that had established a relationship between depression and cardiac recovery. Moreover, the current sample reemphasized the the high rate of depression among heart patients relative to the general adult male population by finding a proportion of major depression that was commensurate with past research. It was the first study (to this author's knowledge) to

compare post-myocardial infarction and unstable angina patients on relevant psychosocial measures, and to combine them into one sample for analysis. Finally, no other study addressing psychosocial correlates of cardiac recovery has acknowledged the documented relationship between depression and marital satisfaction, and specifically investigated this association relative to recovery.

While limitations and methodological flaws existed, the conceptual and empirical findings of the project most certainly advanced the state of research in the area of psychosocial correlates of cardiac recovery.

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Appendix A

Baseline Assessment Packet

Informed Consent
&
Intake Interview Materials

- **Informed Consent Form**
- **Personal History Questionnaire**
- **Research Record Form**

The materials contained in this packet are to be completed prior to the participant's discharge from the hospital. A researcher should assist the patient in filling out each of the enclosed forms.

IRB#: 06906

University of Oklahoma Health Sciences Center
Veterans Affairs Medical Center

Individual's Consent to Voluntary Participation in a Research Project

Title of Study

I, _____, understand that this study, entitled "The relationships among depression, marital distress and cardiac recovery six months post myocardial infarction" is sponsored by the V.A. Medical Center and is being directed by Ravi Mehta, M.D., John Tassey, Ph.D., Terry M. Pace, Ph.D. and Jim R. Keller, M.Ed.

Purpose of the Study

I realize that this study is concerned with how psychological factors (personality, mood and relationships) might affect a patient's recovery from a heart attack. I have been asked to participate because I have recently suffered my first heart attack and have just begun the recovery process. Specifically, the researchers would like to see if my personality (the way I see the world), my emotions (happy, sad, mad etc.) and my relationship with my spouse (if married) has anything to do with how quickly and how well I recover from my heart attack. To investigate these issues, I will be asked to complete a few short questionnaires that have questions about my personality, emotions and marriage. This information will be analyzed to see if any of these things may have effected my recovery. I understand that it is the goal of this study to use the information generated by my participation in assisting health care professionals to better understand the needs of heart attack patients like myself.

Description of the Study & Confidentiality

If I decide to participate in the study, I will be asked to spend a few minutes during my hospital stay to give some general information about myself on a brief Personal History Questionnaire (medical history, current living situation, job etc.). My name *will not* appear on this form.

I will be asked to complete one packet of questionnaires at three different times during the study. The first packet will be given to me at the beginning of the study, the second will be given to me three months from now, and the third will be given to me six months from now. There are four questionnaires per packet which are designed to assess several aspects of my life including the way I view my marriage (if married), my overall personality, my mood, and the way I tend to interact with others. On average, each questionnaire involves 25 questions in which I simply circle the appropriate response. To make things easier, the researchers will call me to arrange a convenient time and place to complete each packet of questionnaires.

To return the completed questionnaires, I will simply hand them to the researchers (if completed at the V.A.) or place them in the stamped and addressed envelope that will be enclosed in each packet and mail them back to the researchers. It is estimated that *each packet* will take about 20 to 30 minutes to complete. I understand that if I wish to withdraw from the study, I can simply mark the space provided on the packet cover sheet and drop it in the mail to the researchers.

Once again, I understand that **my name will not appear on any of the packets, questionnaires or envelopes**, only my research number will be displayed. At the end of the study, I will be given the opportunity to talk to the researchers at length about the project and my test results if I so chose.

Costs of the Study

My participation in the study will not cost me anything. All materials will be sent to me with an addressed and stamped return envelope that can simply be sealed and dropped in the mail box.

Risks Associated with the Study

I understand that, since my name will not appear on any information form and stringent steps are to be taken to maintain my confidentiality, the only foreseen risk of the study relates to the experience of completing the questionnaires. I realize that while the questionnaires are not designed to uncover unpleasant thoughts or memories, some questions may be uncomfortable to answer. It is clear to me that if any discomfort should arise that is extreme or very troubling to me, the researchers are prepared to discuss this with me and/or refer me for appropriate services such as counseling. I also understand that if the results of my questionnaires indicate that I am in severe emotional distress, the researchers are prepared to contact me to assist me in finding appropriate services.

Benefits of the Study

I understand that I will not receive any money or other personal benefits for my participation in this study.

Alternative to Participation in the Study

I have the option not to participate in this study.

Subject's Assurances

I understand that my participation in this study is voluntary. I have not given up any of my legal rights or released any individual or institution from liability for negligence.

I also understand that I may withdraw from this study at any time without penalty or loss of benefits to which I am otherwise entitled. My treatment by and relations with the physicians and organizations involved in this study will not be affected now or in the future if I decide not to participate, or if I start the study and decide later to withdraw.

I understand that records of this study will be kept confidential, and that I will not be identified by name or description in any reports or publications about this study. I understand that my file will be assigned a number so that the only document with my name on it will be this Informed Consent Form, which will be placed under lock and key so that no one is able to connect personal information with my name.

If I have questions about this study, or need to report any adverse effects from the research procedures, I will contact Ravi Mehta, M.D. or John Tassey, Ph.D. at (405)270-0501 during the work day or at (405)270-5495 in the evening or on weekends. If I have questions about my rights as a research subject, I may contact the Director of Research Administration, in the OUHSC Office of Research Administration, at (405)271-2090.

I have read this consent document. I understand its contents, and I freely consent to participate in this study under the conditions described. I will receive a copy of this consent form.

Research Subject:_____ Date:_____

Witness:_____ Date:_____

Investigator:_____ Date:_____

Personal History Questionnaire

INSTRUCTIONS: Please complete the following sections, but please do not put your name on this or any other form, this will insure that all of your information remains absolutely confidential.

Please check the box next to the statement that best describes why you have been hospitalized.

☐ I have been hospitalized for a heart attack. ☐ I have been hospitalized for chest pain, *not* a heart attack.

Demographic Information

Please fill in the blanks or circle the appropriate choices.

(1) Age:____ (2) Height:____ (3) Weight:____ (4) Gender: M F (5) Years of School:____

(6) Employed? Y (yes) N (no) (7) Yearly Family Income: 0- $15,000 $16,000-$30,000 Above $30,000
Please Circle One Please Circle One

(8) Race or ethnicity (optional):____ (9) Number of years married:____

Health Behaviors

Please fill in the blanks or circle the appropriate choices.

(1) Smoke: Y N If yes, # of packs per day:____ (2) Drink: Y N If yes, avg. # of drinks per week____
Please Circle One Please Circle One

(3) I follow my doctor's recommended diet: Not At All Sometimes Most of the Time Always
Please Circle One

(4) Number of times per week I exercise (walking, running, biking etc.): 1 2 3 4 or more
Please list the types of exercise you do regularly (twice or more a week): _____

Current Living Situation

Please fill in the blanks and circle the appropriate choices.

(1) Number of people living in your home:____ (2) Number of years in your current home:____

(3) Number of children living in your home:____ (4) I live in the: City (or suburb) Country
Please Circle One

(5) Do you have anyone assisting with your daily living (e.g., nurse, healthcare provider): Y N
Please Circle One

Medical History

Please fill in the blanks and circle the appropriate choices.

(1) How many heart attacks (that were confirmed by a doctor) have you had in your life: ____

(2) Please list any current illnesses (e.g., diabetes, arthritis, stomach disorders etc.): _____

(3) Please list current medications:_____

(4) Do you suffer from chronic (daily) pain (e.g., backaches, headaches, arthritis etc.)? Y N
Please Circle One

(5) Did you have any type of surgery related to your heart attack? Y N If yes, describe:_____
Please Circle One

Research Record Form

This form is to be used by the researchers to gather information from medical records and the V.A. computer file. Information on this form is to be entered into the computer database.

☐ **Myocardial Infarction** ☐ **Unstable Angina** ☐ **Other Diagnosis**

Medical Diagnosis Information:

Additional Case Information:

Notes and Comments Concerning Recruitment / Intake Interview:

The
Assessment Packet

- **Cover Letter**
- **Patient Status Form**
- **Inventory for Diagnosing Depression (IDD)**
- **Dyadic Adjustment Scale (DAS)**
- **Myocardial Infarction Risk Factor Inventory (MIRFI)**
- **Myocardial Infarction Recovery Index (MIRI)**

The materials contained in this packet are to be completed at Month 1, Month 3 and Month 6. Please do not put your name on any of the forms to insure confidentiality. Upon completion, this packet can be given to a researcher in person, or mailed in the enclosed stamped and addressed envelope.

Assessment Packet Cover Letter

Introduction

This is your Assessment Packet for the Veterans Affairs Medical Center study entitled, *The relationships among depression, marital satisfaction and cardiac recovery six months post myocardial infarction.* We thank you again for your participation in our study. We hope that this project (with your help) will go toward developing programs that will help heart patients get well more quickly and stay well.

The Researchers

The researchers conducting this study are John Tassey, Ph.D. (Dept. of Behavioral Medicine, V.A. Medical Center); Ravi Mehta, M.D. (Psychiatric Resident, V.A. Medical Center); Jim R. Keller, M.Ed. (Dept. of Educational Psychology, University of Oklahoma); Terry M. Pace, Ph.D. (Dept. of Educational Psychology) and Kala Mehta, M.D. (Dept of Cardiology, V.A. Medical Center).

If You Have Questions or Comments

If you have any questions about your participation in the study, how to complete a questionnaire or wish to speak to one of the principle researchers, please call 270-0501 and ask for any of the V.A. Medical Center staff listed above to be paged.

You may also call the Department of Educational Psychology of the University of Oklahoma at 325-5974 to speak with Jim Keller or Terry Pace.

If at any time you feel that you are need of services (such as counseling) to address emotional distress (like extreme depression), please contact your physician or one of the researchers immediately.

How to Complete the Packet

Please complete all of the forms and questionnaires contained within this packet to the best of your ability. Please remember that it is very important that you try to be as open as possible. Please read the directions at the top of each questionnaire, and select only one response for each question. To ensure that no one is able to identify your name with any of the questionnaires, **please, do not put your name on this or any of the enclosed forms.**

Thank you again for taking the time to participate in our project!

Sincerely,

Jim R. Keller, M.Ed.
Research Investigator,
Dept. of Educational Psychology, U. of Oklahoma

Patient Status Form

Please complete this form when your are filling out Assessment Packet #2 and Assessment Packet #3. It is very important to our study that we know of any major changes in your life situation. **Please, do not put your name on this form.**

(1) My marital status has changed since I completed the last assessment packet. Yes No
Please circle one

(2) I am now: Married Separated Divorced Widowed
Please circle one

(3) I have lost my job since the I completed the last assessment packet. Yes No
Please circle one

(4) I have Increased / Decreased my exercise since the last assessment packet.
Please circle one

(5) My smoking habits have changed since the last assessment packet. Yes No Avg. # of packs per week____
Please circle one

(6) My living arrangements have changed since the last assessment packet. Yes No If yes, please describe how it has changed: _____
Please circle one

_____

(7) I have had treatment from a psychologist or psychiatrist since my last assessment packet. Yes No
Please circle one

(8) Please describe any other life changes that you feel are important since your last assessment packet: _____

_____

_____

Additional Comments: _____

_____

_____

_____

_____

_____

_____

Inventory for Diagnosing Depression (IDD)

I.D.D.

Instructions:

1. On this questionnaire are groups of 5 statements.
2. Read each group of statements carefully. Then pick out the one statement in each group that best describes the way you have been feeling the PAST TWO WEEKS. Circle the number next to the statement you picked.
3. Once you have circled one of the 5 statements, make a mark in the blank which best indicates how long you have felt that way.

---

1. 0 I do not feel sad or depressed.
   1 I occasionally feel sad or down.
   2 I feel sad most of the time, but I can snap out of it.
   3 I feel sad all the time, and I can't snap out of it.
   4 I am so sad or unhappy that I can't stand it.

   How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

2. 0 My energy level is normal.
   1 My energy level is occasionally a little lower than normal.
   2 I get tired more easily or have less energy than usual.
   3 I get tired from doing almost anything.
   4 I feel tired or exhausted almost all of the time.

   How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

3. 0 I have not been feeling more restless and fidgety than usual.
   1 I feel a little more restless or fidgety than usual.
   2 I have been very fidgety, and I have some difficulty sitting still in a chair.
   3 I have been extremely fidgety, and I have been pacing a little bit almost every day.
   4 I have been pacing more than an hour per day, and I can't sit still.

   How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

4. 0 I have not been talking or moving more slowly than usual.
   1 I am talking a little slower than usual.
   2 I am speaking slower than usual, and it takes me longer to respond to questions, but I can still carry on a normal conversation.
   3 Normal conversations are difficult because it is hard to start talking.
   4 I feel extremely slowed down physically, like I am stuck in mud.

   How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

5. 0 I have not lost interest in my usual activities.
   1 I am a little less interested in 1 or 2 of my usual activities.
   2 I am less interested in several of my usual activities.
   3 I have lost most of my interest in almost all of my activities.
   4 I have lost all interest in all of my usual activities.

   How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

6. 0 I get as much pleasure out of my usual activities as usual.
   1 I get a little less pleasure from 1 or 2 of my usual activities.
   2 I get less pleasure from several of my usual activities.
   3 I get almost no pleasure from most of the activities which I usually enjoy.
   4 I get no pleasure from any of the activities which I usually enjoy.

   How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

7. 0 I have not noticed any recent change in my interest in sex.
   1 I am only slightly less interested in sex than usual.
   2 There is a noticeable decrease in my interest in sex.
   3 I am much less interested in sex now.
   4 I have lost all interest in sex.

   How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

8. 0 I have not been feeling guilty.
   1 I occasionally feel a little guilty.
   2 I often feel guilty.
   3 I feel quite guilty most of the time.
   4 I feel extremely guilty most of the time.

   How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

9. 0 I do not feel like a failure.
   1 My opinion of myself is occasionally a little low.
   2 I feel I am inferior to most people.
   3 I feel like a failure.
   4 I feel I am a totally worthless person.

   How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

10. 0 I haven't had any thoughts of death or suicide.
    1 I occasionally think life is not worth living.
    2 I frequently think of dying in passive ways (such as going to sleep and not waking up), or that I'd be better off dead.
    3 I have frequent thoughts of killing myself, but I would not carry them out.
    4 I would kill myself if I had the chance.

    How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

11. 0 I can concentrate as well as usual.
    1 My ability to concentrate is slightly worse than usual.
    2 My attention span is not as good as usual and I am having difficulty collecting my thoughts, but this hasn't caused any problems.
    3 My ability to read or hold a conversation is not as good as it usually is.
    4 I cannot read, watch TV, or have a conversation without great difficulty.

    How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

12. 0 I make decisions as well as I usually do.
    1 Decision making is slightly more difficult than usual.
    2 It is harder and takes longer to make decisions, but I do make them.
    3 I am unable to make some decisions.
    4 I can't make any decisions at all.

    How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

13. 0 My appetite is not less than normal.
    1 My appetite is slightly worse than normal.
    2 My appetite is clearly not as good as usual, but I still eat.
    3 My appetite is much worse now.
    4 I have no appetite at all, and I have to force myself to eat even a little.

    How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

14. 0 I haven't lost any weight.
    1 I've lost less than 5 pounds.
    2 I've lost between 5-10 pounds.
    3 I've lost between 11-25 pounds.
    4 I've lost more than 25 pounds.

15. 0 My appetite is not greater than normal.
1 My appetite is slightly greater than normal.
2 My appetite is clearly greater than usual.
3 My appetite is much greater than usual.
4 I feel hungry all the time.

How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

16. 0 I haven't gained any weight.
1 I've gained less than 5 pounds.
2 I've gained between 5-10 pounds.
3 I've gained between 11-25 pounds.
4 I've gained more than 25 pounds.

17. 0 I am not sleeping less than normal.
1 I occasionally have slight difficulty sleeping.
2 I clearly don't sleep as well as usual.
3 I sleep about half my normal amount of time.
4 I sleep less than 2 hours per night.

How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

18 0 I am not sleeping more than normal.
1 I occasionally sleep more than normal.
2 I frequently sleep at least 1 hour more than usual.
3 I frequently sleep at least 2 hours more than usual.
4 I frequently sleep at least 3 hours more than usual.

How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

19. 0 I do not feel anxious, nervous, or tense.
1 I occasionally feel a little anxious.
2 I often feel anxious.
3 I feel very anxious most of the time.
4 I feel terrified and near panic.

How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

20. 0 I do not feel discouraged about the future.
    1 I occasionally feel a little discouraged about the future.
    2 I often feel discouraged about the future.
    3 I feel very discouraged about the future most of the time.
    4 I feel that the future is hopeless and that things will never improve.

    How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

21. 0 I do not feel irritated or annoyed.
    1 I occasionally get a little more irritated than usual.
    2 I get irritated or annoyed by things that usually don't bother me.
    3 I feel irritated or annoyed almost all of the time.
    4 I feel so depressed that I don't get irritated at all by things that used to bother me.

    How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

22. 0 I am not worried about my physical health.
    1 I am occasionally concerned about bodily aches and pains.
    2 I am worried about my physical health.
    3 I am very worried about my physical health.
    4 I am so worried about my physical health that I cannot think about anything else.

    How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

23 To what extent, if any, do the items you endorsed interfere with your job or school performance?

    0 Does not apply–I did not endorse any items.
    1 Does not interfere with my job or school.
    2 Mild–they result in only minor impairment.
    3 Moderate–a lot of interference, but I can still function on the job or at school.
    4 Severe–a significant interference; makes it nearly impossible to go to school, go to work, or do my job.

    How long have you felt this way? ___less than 2 weeks ___more than 2 weeks

Dyadic Adjustment Scale
(DAS)

DAS by Graham B. Spanier, Ph.D.

Name:_____ Sex: M F Marital Status:_____ Age:_____

Most persons have disagreements in their relationships. Please indicate below the approximate extent of agreement or disagreement between you and your partner for each item on the following list. Circle the star under one answer for each item.

	Always Agree	Almost Always Agree	Occasionally Disagree	Frequently Disagree	Almost Always Disagree	Always Disagree
1. Handling family finances						
2. Matters of recreation						
3. Religious matters						
4. Demonstrations of affection						
5. Friends						
6. Sex relations						
7. Conventionality (correct or proper behavior)						
8. Philosophy of life						
9. Ways of dealing with parents or in-laws						
10. Aims, goals, and things believed important						
11. Amount of time spent together						
12. Making major decisions						
13. Household tasks						
14. Leisure time interests and activities						
15. Career decisions						

	All The Time	Most Of The Time	More Often Than Not	Occasionally	Rarely	Never
16. How often do you discuss or have you considered divorce, separation, or termination of your relationship?						
17. How often do you or your mate leave the house after a fight?						
18. In general, how often do you think that things between you and your partner are going well?						
19. Do you confide in your mate?						
20. Do you ever regret that you married (or lived together)?						
21. How often do you and your partner quarrel?						
22. How often do you and your mate get on each others' nerves?						

	Every Day	Almost Every Day	Occasionally	Rarely	Never
23. Do you kiss your mate?					

	All Of Them	Most Of Them	Some Of Them	Very Few Of Them	None Of Them
24. Do you and your mate engage in outside interests together?					

How often do the following occur between you and your mate?	Never	Less Than Once A Month	Once Or Twice A Month	Once Or Twice A Week	Once A Day	More Often
25. Have a stimulating exchange of ideas						
26. Laugh together						
27. Calmly discuss something						
28. Work together on a project						

These are some things about which couples sometimes agree or disagree. Indicate if either item caused differences of opinions or were problems in the past five weeks.

	Yes	No
29. Being too tired for sex		
30. Not showing love		

31. The stars on the following line represent different degrees of happiness in your relationship. The middle point, "Happy," represents the degree of happiness of most relationships. Circle the star above the phrase which best describes the degree of happiness, all things considered, of your relationship.

Extremely Unhappy	Fairly Unhappy	A Little Unhappy	Happy	Very Happy	Extremely Happy	Perfect

32. Which of the following statements best describes how you feel about the future of your relationship? Circle the letter for one statement.

A. I want desperately for my relationship to succeed, and would go to almost any length to see that it does.
B. I want very much for my relationship to succeed, and will do all I can to see that it does.
C. I want very much for my relationship to succeed, and will do my fair share to see that it does.
D. It would be nice if my relationship succeeded, but I can't do much more than I am doing now to keep the relationship going.
E. It would be nice if it succeeded, but I refuse to do any more than I am doing now to keep the relationship going.
F. My relationship can never succeed, and there is no more that I can do to keep the relationship going.

Myocardial Infarction Risk Factor Inventory (MIRFI)

Myocardial Infarction Risk Factor Inventory (MIRFI)

INSTRUCTIONS: Complete each item by circling the number that corresponds to the word or phrase (e.g., *Agree, Disagree*) that best describes your level of agreement with the statement at the left. You do not need to calculate any composite scores or the overall index score, the researchers will take care of this for you. Thank you for your cooperation.

Factor 1

	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
(1) My spouse and I have a good marriage	1	2	3	4	5
(2) My spouse and I agree on a many things	1	2	3	4	5
(3) Our sex life is satisfactory	1	2	3	4	5
(4) I often feel like my spouse and I are a team	1	2	3	4	5
(5) I do not often think of leaving my spouse	1	2	3	4	5

Factor 1 Composite Score ☐

Factor 2

	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
(1) I rarely feel sad during the day	1	2	3	4	5
(2) I have as much energy lately as I usually have	1	2	3	4	5
(3) I get as much pleasure out of things than I used to	1	2	3	4	5
(4) My life is worth living	1	2	3	4	5
(5) I do not feel discouraged about the future	1	2	3	4	5

Factor 2 Composite Score ☐

Factor 3

	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
(1) I rarely worry about the time	1	2	3	4	5
(2) I rarely get annoyed when others interrupt me	1	2	3	4	5
(3) It does not bother me to wait in line	1	2	3	4	5
(4) I'm not really very competitive	1	2	3	4	5
(5) I don't eat or speak much faster than others do	1	2	3	4	5

Factor 3 Composite Score ☐

Factor 4

	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
(1) Others would not consider me irritable	1	2	3	4	5
(2) Most people are honest and fair	1	2	3	4	5
(3) I don't often defend my opinions	1	2	3	4	5
(4) I usually forgive and forget	1	2	3	4	5
(5) I enjoy talking with people I do not know well	1	2	3	4	5

Factor 4 Composite Score ☐

Factor 5

	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
(1) I feel I have friends I can count on	1	2	3	4	5
(2) My family is very supportive of me	1	2	3	4	5
(3) I rarely feel lonely or isolated	1	2	3	4	5
(4) I have someone with whom I can share feelings	1	2	3	4	5
(5) The people around me understand my needs	1	2	3	4	5

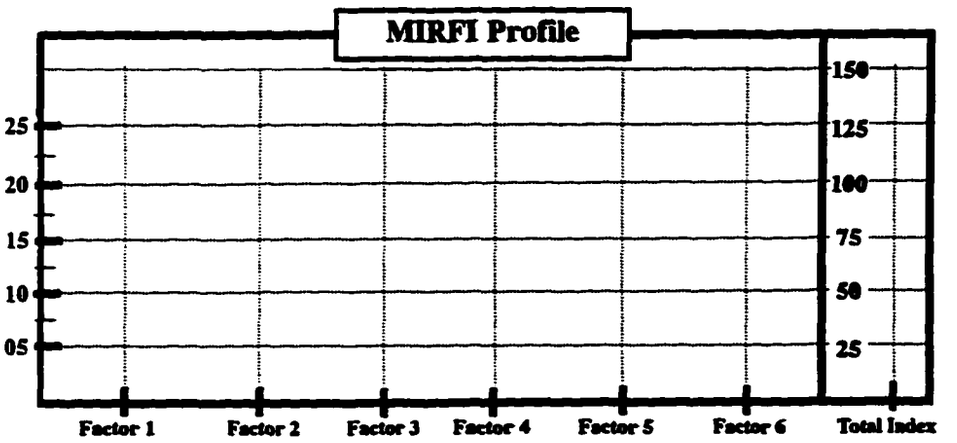
Factor 5 Composite Score ⇩ □

Factor 6

	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
(1) I rarely forget to take my medication	1	2	3	4	5
(2) I follow my doctor's advice very closely	1	2	3	4	5
(3) I do things daily to help myself get better	1	2	3	4	5
(4) I've given up things in my life that hurt my heart	1	2	3	4	5
(5) Overall, I follow my treatment plan very well	1	2	3	4	5

Factor 6 Composite Score ⇩ □

Total MIRFI Index: ➡ □



Myocardial Infarction Recovery Index (MIRI)

Nurses' Myocardial Infarction Recovery Index (MIRI-3)

Adapted for self-administration by Jim R. Keller, M.Ed.

It is:

1 to 2 Weeks...	1 Month...	3 Months...	6 Months...
after my heart attack	after my heart attack	after my heart attack	after my heart attack

I. Return to Physical Well Being

These questions look at your current physical abilities as compared to before your heart attack.

A. Physiological Measures

Please circle the number over the response that best describes your situation.

1. Heart Rate Response to Exercise

Since my heart attack, my heart returns to a resting heart rate about:

1	2	3	4	5
30 Minutes... or more after exercise	20-30 Minutes... after exercise	10-20 Minutes... after exercise	5-10 Minutes... after exercise	2-5 Minutes... after exercise

2. Blood Pressure

Since my heart attack, my blood pressure returns to normal about:

1	2	3	4	5
30 Minutes... or more after exercise	20-30 Minutes... after exercise	10-20 Minutes... after exercise	5-10 Minutes... after exercise	2-5 Minutes... after exercise

3. Change in Chest Pain

Since my heart attack, I get pain in my chest with:

1	2	3	4	5
No Movement... it wakes me up at night	Minimal Movement... like moving about the house	Moderate Movement... after meals or exercise	Vigorous Movement... like during exercise	I Never Have chest pain

4. (a) Chest Pain Severity

Since my heart attack, my chest pain is best described as:

1	2	3	4	5
Severe... chest pain	Moderate... chest pain	Mild... chest pain	Slight... discomfort	I Never Have chest pain

4. (b) Frequency of Chest Pain

Since my heart attack, my chest pain occurs:

1	2	3	4	5
Once to Several... times a day	Once to Several... times a week	Once to Several... times per month	Rarely... fewer than once/month	I Never Have chest pain

A. Physiological Measures (cont.) Page 2

Please circle the number over the response that best describes your situation.

4. (c) Duration of Chest Pain

Since my heart attack, my chest pain typically lasts:

1	2	3	4	5
15-20 Minutes... or more	10-15 Minutes	3-10 Minutes	3 Minutes... or less	I have no chest pain

4. (d) Medication Needed to Relieve Chest Pain

Since my heart attack, my chest pain:

1	2	3	4	5
Is *Not* Relieved by 3... nitroglycerine tablets	Is Relieved by 3... nitroglycerine tablets	Is Relieved by 2... nitroglycerine tablets	Is Relieved by 1... nitroglycerine tablet	Requires No... nitroglycerine

5. (a) Ability to Sleep

Since my heart attack:

1	2	3	4	5
I Am Unable... to sleep at all	I Take a Long Time to... fall asleep & I wake early	I Take Short Naps... as my main form of sleep	I Sleep the Same as... before my heart attack	I Sleep Better Now... than before the attack

5. (b) Quality of Sleep

Most mornings since my heart attack, I wake up feeling:

1	2	3	4	5
Exhausted	Slightly Tired	Neither... exhausted nor refreshed	Slightly Refreshed	Fully Refreshed

6. (a) Return to My Job

Since my heart attack, I am:

0	2	5	10	15
Retired	Doing Less Than 50%... of my previous work load	Doing 50% or More... of my previous work load	Doing the Same... work as before the attack	Doing More Now... than before

6. (b) Overall Job Functioning

With regard to my ability to work now as compared to before the heart attack, I am:

0	2	5	10	15
Unable to Function	Doing Less Than 50%... of my previous work load	Doing 50% or More... of my previous work load	Doing the Same... work as before the attack	Doing More Now... than before

7. (a) Return to Driving

With regard to my driving habits since my heart attack, I:

1	2	3	4	5
Didn't Drive Prior... to my heart attack	Have Not Tried to Drive... since my heart attack	Every time I Drive... I experience chest pain	I Drive the Same ... as before my heart attack	I Drive Better Now... than before

Please circle the number over the response that best describes your situation.

7. (b) Return to Shopping

With regard to my shopping habits since my heart attack, I:

0	1	2	3	4	5
Didn't Shop Prior... to my heart attack	Have Not Tried... to shop since my heart attack	Experience pain &... shortness of breath before I finish shopping	Shop... with frequent rest periods	Shop... just as I did before my heart attack	Shop... better than before the attack

7. (c) Return to Sexual Activities

With regard to my sexual activity since my heart attack, I have:

0	2	10	15
Had No Opportunity... to engage in sex	Been Engaging Less... in sex than before the attack	Been Doing About the Same... as before the attack	Been Doing More... than before the attack

7. (d) Changing Bed Linen

With regard to my ability to change the bed since my heart attack:

1	2	3	4	5
I Do Not Attempt... to change the bed	I'm Unable to... pull up blanket & spread	I'm Able to Pull Up... spread with rest periods	I'm Able to Change... the bed with rest periods	I'm Able to Change... the bed as before

7. (e) Yard Work

With regard to my ability to do yard work since my heart attack:

1	2	3	4	5
I Do Not Attempt... yard work	Unable to Do... any yard work	Able to Garden... while sitting	Able to Do Yard Work Requiring... both upper & lower body work	Able to Do the Same... yard work as before

7. (f) Stair Climbing

Regarding my ability to climb stairs since my heart attack, I:

1	2	3	4	5
Can't Climb 2 Flights... of stairs due to chest pain & shortness of breath	Can Climb 2 Flights... slowly with occasional pain & shortness of breath	Can Walk Stairs... slowly without chest pain & shortness of breath	Can Walk Stairs... as well as before the heart attack	Can Walk More... stairs than before the heart attack

7. (g) Heavy House Work Like Vacuuming

Regarding my ability to do heavy house work like vacuuming since my heart attack, I:

1	2	3	4	5
Have Not Attempted... to vacuum	Can Not Vacuum... the house at all	Can Vacuum... with a few rest periods	Can Vacuum... the same as before	Can Vacuum More... than before

B. Patient's Perception of Physical Recovery

Please circle the number over the response that best describes your situation.

1. How I View My Recovery

When compared to other heart attack patients of the same age and general physical condition, I think I am:

0	2	5	10	15
Not Recovering... at all	**Recovering Slower...** than expected	**Recovering...** about the same as expected	**Recovering Faster...** than expected	**Completely...** recovered

2. My Progress...Attainment of My Goal for Recovery

With regard to my own goals for recovery after my heart attack, I feel that I am:

0	2	5	10	15
Not Progressing... at all	**Progressing...** very little	**About Half Way...** to my goals	**Satisfied...** I have met my goals	**Beyond My Goals...** I have surpassed my goals

C. Spouse's Perception of Physical Recovery

For this section, ask your partner about his/her view of how you are recovering.

Please circle the number over the response that best describes your situation.

1. Spouse's (or Partner's) View of Patient's Recovery

My spouse's (partner's) opinion of my recovery is that I am:

0	2	5	10	15
Not Recovering... at all	**Recovering Slower...** than expected	**Recovering...** about the same as expected	**Recovering Better...** than expected	**Completely...** recovered

D. Subsequent Cardiac Events (since the beginning of the study)

Please circle the number over the response that best describes your situation.

1. Subsequent Heart Attacks

Since the heart attack that hospitalized me at the beginning of the study, I have had:

0	20
At Least One Other Heart Attack	**No Other Heart Attacks**

2. Subsequent Emergency Room Visits (related to my heart)

Since the heart attack that hospitalized me at the beginning of the study, I have had:

0	5	10
2 or More Emergency Room Visits	**1 Emergency Room Visit**	**No Emergency Room Visits**

2. Subsequent Hospital Stays (related to my heart)

Since the heart attack that hospitalized me at the beginning of the study, I have had:

0	5	10
2 or More Hospital Stays	**1 Hospital Stay**	**No Hospital Stays**

II. Return to Emotional Well Being

These questions look at your emotional well being since your heart attack.

A. Emotional Recovery Measures

Please circle the number over the response that best describes your situation.

1. State Anxiety (anxiety related to my heart attack)

With regard to feeling anxious since my heart attack, I feel I am:

1	2	3	4	5
Extremely More... anxious than before the attack	**Moderately More...** anxious than before	**Mildly More...** anxious than before	**Same Level...** of anxiety as before the attack	**Less Anxious...** than before

2. Depression

With regard to any depression since my heart attack, I feel I am:

1	2	3	4	5
Extremely... depressed	**Moderately...** depressed	**Mildly...** depressed	**As Depressed...** as before the attack	**Less Depressed...** than before the attack

3. My Outlook for the Future

I feel that my outlook on the future since my heart attack is that I:

1	2	3	4	5
Am Unable to Envision... a future for myself	**Envision a Negative...** future for myself	**Envision a Limited...** future for myself	**Envision a Positive...** future for myself	**Have Set Goals...** for my future

4. Expression of Emotion

With regard to the way I express emotion since my heart attack, I feel that I show:

1	2	3	4	5
No Emotions... outwardly	**Very Little...** emotion to others	**Happiness (Smiles)...** while talking about sad things	**Too Much Emotion...** cry too easily or laugh too hard	**Appropriate...** emotions for the situation

5. The Grieving Process

Of the five stages of the grieving process, I am currently experiencing:

1	2	3	4	5
Denial... I refuse to acknowledge the seriousness of my situation	**Anger...** I'm angry about my situation	**Bargaining...** I'm making deals with God to get better	**Hope...** I am hopeful that I will recover	**Acceptance...** I accept the reality of my heart attack

6. My Sense of Control

With regard to my ability to maintain control of my life and my recovery, I feel that I am:

1	2	3	4	5
Out of Control... all of the time	**Out of Control...** most of the time	**Out of Control...** some of the time	**In Control...** some of the time	**In Control...** all of the time

III. Return to Social Well Being

These questions look at your social life and functioning since your heart attack.

A. Social Recovery Measures

Please circle the number over the response that best describes your situation.

1. Family-Related Activities

With regard to doing activities with my family since my heart attack, I am doing:

1	2	3	4	5
No Family Activities... I never did them before the attack	**No Family Activities...** since the heart attack	**Fewer Activities...** than before the attack	**The Same Number...** of family activities as before	**More Activities...** than before

2. Social Activities (with friends and colleagues)

With regard to doing activities with friends and colleagues since my heart attack, I am doing:

1	2	3	4	5
No Social Activities... I never did them before the attack	**No Social Activities...** since the heart attack	**Fewer Activities...** than before the attack	**The Same Number...** of social activities as before	**More Activities...** than before

IV. Return to Spiritual Well Being

These questions look at your spiritual well being since your heart attack.

A. Spiritual Recovery Measures

Please circle the number over the response that best describes your situation.

1. Source of Power and Strength Outside of Myself

With regard to my faith in a higher power (like God) since my heart attack, I feel:

1	2	3	4	5
Abandoned... by higher power	**Less Connected to...** higher power	**Neutral Towards...** higher power	**Slightly Connected to...** higher power	**Strongly Connected to...** higher power

2. Finding Positive Meaning in My Heart Attack Experience

I feel that my heart attack experience has been:

1	2	3	4	5
Totally Negative... a form of punishment	**Slightly Negative...** not all bad	**Neither...** not good or bad	**Slightly Positive...** some good has come of it	**Very Positive Meaning...** has come from it

Appendix B

Follow-Up Assessment Packet

$00017

Final
Follow-Up Assessment

To be completed at approximately 6 to 9 months post hospitalization.

RIN #_____

Date:_____
Researcher:_____

Phone #:( )_____
Address:_____
_____

Measures Completed

MIRI-SF..... ____
Quick Gauge of Depression..... ____
Quick Gauge of Marital Satisfaction..... ____
MIRFI..... ____

Measures To Be Mailed

MIRI-SF..... ____
MIRFI..... ____
IDD..... ____
DAS..... ____

Notes / Comments

_____
_____
_____
_____
_____
_____
_____

Questionnaire

The

MIRI-Short Form

How long should it take to complete the MIRI? 5 to 10 Minutes.

INSTRUCTIONS:

(1) There are 20 short items on this questionnaire.

(2) Each item **describes an aspect of your recovery** following your recent heart trouble.

(3) You are to **circle the number over the phrase** that best describes your situation *in the past few weeks.*

EXAMPLE ITEMS

1. The Level of Activity That Makes My Chest Hurt

In the last few weeks, I get pain in my chest with:

1	2	3	4	5
No Movement... while at rest or sleeping	Minimal Movement... like moving about the house	Moderate Movement... after meals or exercise	Vigorous Movement... like during exercise	I Never Have chest pain

2. The Severity of My Chest Pain

In the last few weeks, my chest pain is best described as:

1	2	3	4	5
Severe... chest pain	Moderate... chest pain	Mild... chest pain	Slight... discomfort	I Never Have chest pain

Nurses' Myocardial Infarction Recovery Index - Short Form

Adapted for self-administration by Jim R. Keller, M.Ed.

Today's Date: _____

DIRECTIONS: Circle the number above the response that best describes how you have been doing for the past few weeks.

I. My Physical Recovery

These questions look at your current physical ability since the heart trouble that hospitalized you at the beginning of the study.

1. The Level of Activity That Makes My Chest Hurt

In the last few weeks, I get pain in my chest with:

1	2	3	4	5
No Movement... while at rest or sleeping	Minimal Movement... like moving about the house	Moderate Movement... after meals or exercise	Vigorous Movement... like during exercise	I Never Have chest pain

2. The Severity of My Chest Pain

In the last few weeks, my chest pain is best described as:

1	2	3	4	5
Severe... chest pain	Moderate... chest pain	Mild... chest pain	Slight... discomfort	I Never Have chest pain

3. The Frequency of My Chest Pain

In the last few weeks, my chest pain typically occurs:

1	2	3	4	5
Once to Several... times a day:	Once to Several... times a week	Once to Several... times per month	Rarely... fewer than once/month	I Never Have chest pain

4. How Long My Chest Pain Lasts

In the last few weeks, my chest pain typically lasts:

1	2	3	4	5
15-20 Minutes... or more	10-15 Minutes	5-10 Minutes	3 Minutes... or less	I have no chest pain

5. The Number of Nitro Tablets Needed to Relieve My Chest Pain

In the last few weeks, my chest pain:

1	2	3	4	5
Is *Not* Relieved by 3... nitroglycerine tablets	Is Relieved by 3... nitroglycerine tablets	Is Relieved by 2... nitroglycerine tablets	Is Relieved by 1... nitroglycerine tablet	Requires No... nitroglycerine

6. My Ability to Sleep

In the last few weeks:

1	2	3	4	5
I Am Unable... to sleep at all	I Take a Long Time to... fall asleep & I wake early	I Take Short Naps... as my main form of sleep	I Sleep the Same as... before my heart trouble	I Sleep Better Now... than before

7. My Ability to Do Daily Chores

With regard to my ability to get things done as compared to before my hospitalization, I am:

1	2	3	4	5
Unable to Function... can't do hardly any of my usual daily chores	Doing Less Than Half... of the daily chores that I used to be able to do	Doing More Than Half... of the daily chores that I used to do	Doing the Same... things around the house as I used to	Doing More Now... than before my heart trouble

8. My Ability to Engage in Sexual Activities

In the last few weeks:

1	2	3	4	5
've Not Had Any Sex... mainly because of the trouble with my heart	I'm Having Much Less... sex than before my recent heart trouble	I'm Having Less... sex than before my heart trouble	I'm Having the Same... sexual activity as before my recent heart trouble	I'm Having More... sex than before my recent heart trouble

9. My Ability to Climb Stairs

At the present time:

1	2	3	4	5
I Cannot Climb Stairs... due to chest pain & shortness of breath	I Can Climb Stairs... slowly with occasional pain & shortness of breath	I Can Climb Stairs... slowly without chest pain & shortness of breath	I Can Climb Stairs... as well as before my heart trouble	I Can Climb More... stairs than before my heart trouble

II. My Social Life

These questions look at your social life and functioning since your hospitalization at the beginning of the study.

1. My Return to Family-Related Activities

With regard to doing activities with my family in the last few weeks I am doing:

1	2	3	4	5
o Family Activities... nainly because of my heart trouble	Very Few Family Activities... since the onset of my recent heart trouble	Fewer Activities... than before my recent heart trouble	The Same Number... of family activities as before my recent heart trouble	More Activities... than before

2. Social Activities (with friends and colleagues)

With regard to doing activities with friends and colleagues since my recent heart trouble, I am doing:

1	2	3	4	5
o Social Activities... nainly because of my heart trouble	Very Few Social Activities... since the onset of my recent heart troubles	Fewer Activities... than before my heart trouble	The Same Number... of social activities as before my heart trouble	More Activities... than before my heart trouble

III. My Emotional Well Being

These questions look at how you've been *feeling* emotionally since the heart trouble that hospitalized you a few months ago.

1. Feeling anxious about my heart problem.

In the last few weeks I have felt:

1	2	3	4	5
Extremely... anxious	**Moderately...** anxious	**Mildly...** anxious	**No More...** anxious than usual	**Less...** anxious than usual

2. Depression

In the last few weeks I have felt:

1	2	3	4	5
Extremely... depressed	**Moderately...** depressed	**Mildly...** depressed	**As Depressed...** as before my heart trouble	**Less Depressed...** than before my heart trouble

3. My Outlook for the Future

Lately, I feel like my future is:

1	2	3	4	5
Totally Negative... I have no real future	**Mostly Negative...** the future is mostly bad	**Limited...** few things to look forward to	**Mostly Positive...** looks pretty good	**Very Positive...** much to look forward to

4. How I View My Recovery

When compared to other heart patients of the same age and general physical condition, I think I am:

1	2	3	4	5
Not Recovering... at all	**Recovering Slower...** than expected	**Recovering...** about the same as expected	**Recovering Faster...** than expected	**Completely...** recovered

5. Has Something Good Come Out of My Heart Problems?

When all is said and done, the experiences I have had related to my heart problems have been:

1	2	3	4	5
Totally Negative... like I'm being punished	**Slightly Negative...** not all bad	**Neither...** not good or bad	**Slightly Positive...** some good has come of it	**Very Positive Meaning...** has come from my illness

IV. Job Functioning

1. Overall Job Functioning

With regard to my ability to work now as compared to before the heart attack, I am:

1	2	3	4	5
Unable to Function	**Doing Less Than 50%...** of my previous work load	**Doing 50% or More...** of my previous work load	**Doing the Same...** work as before the attack	**Doing More Now...** than before

V. Recent Trouble with My Heart

1. Heart Attacks

Since I left the hospital after my heart trouble (at the beginning of the study) I have had:

1	2	3	4	5
More Than Three Heart Attacks	Three Heart Attacks	Two Heart Attacks	One Heart Attack	No Heart Attacks

2. Emergency Room Visits (related to my heart)

Since I left the hospital after my heart trouble (at the beginning of the study) I have had:

1	2	3	4	5
More Than Three ER Visits	Three ER Visits	Two ER Visits	One ER Visit	No ER Visits

3. Inpatient Hospital Stays (related to my heart)

Since I left the hospital after my heart trouble (at the beginning of the study) I have had:

1	2	3	4	5
More Than Three Hospital Stays	Three Hospital Stays	Two Hospital Stays	One Hospital Stay	No Hospital Stays

Quick Gauge of Depression

for Phone Administration

In the last few weeks I have felt:

1	2	3	4	5
Extremely... depressed	Moderately... depressed	Mildly... depressed	As Depressed... as before my heart trouble	Less Depressed... than before my heart trouble

Quick Gauge of Marital Satisfaction

for Phone Administration

Overall, all things considered, I feel that my marriage is:

1	2	3	4	5
Extremely... unhappy	Moderately... unhappy	Neither... unhappy or happy	Moderately... happy	Extremely... happy

Myocardial Infarction Risk Factor Inventory (MIRFI)

Questionnaire

The

MIRFI

How long should it take to complete the MIRFI? 5 to 10 Minutes.

INSTRUCTIONS:

(1) There are 30 short items on this questionnaire.

(2) Each item is a **statement about you.**

(3) You are to **circle the number** under the phrase that best describes how much you **agree** or **disagree** with the statement about yourself.

For example, if you **agree** that the statement describes you, you would circle the number 2 under "Agree". Take a look at the examples below.

Don't worry about adding up your responses or computing a Composite Score!

EXAMPLE ITEMS

Factor 1

	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
(1) My spouse and I have a good marriage.....	1	(2)	3	4	5
(2) My spouse and I agree on a many things.....	1	(2)	3	4	5
(3) Our sex life is satisfactory.....	1	2	3	(4)	5
(4) I often feel like my spouse and I are a team.....	1	2	(3)	4	5
(5) I hardly ever think of leaving my spouse.....	1	(2)	3	4	5

Factor 1 Composite Score

Myocardial Infarction Risk Factor Inventory (MIRFI)

INSTRUCTIONS: Complete each item by circling the number that corresponds to the word or phrase (e.g., *Agree, Disagree*) that best describes your level of agreement with the statement at the left. You do not need to calculate any composite scores or the overall index score, the researchers will take care of that for you. Thank you for your cooperation.

Factor 1

	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
(1) My spouse and I have a good marriage	1	2	3	4	5
(2) My spouse and I agree on a many things	1	2	3	4	5
(3) Our sex life is satisfactory	1	2	3	4	5
(4) I often feel like my spouse and I are a team	1	2	3	4	5
(5) I do not often think of leaving my spouse	1	2	3	4	5

Factor 1 Composite Score ⇩ ☐

Factor 2

	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
(1) I rarely feel sad during the day	1	2	3	4	5
(2) I have as much energy lately as I usually have	1	2	3	4	5
(3) I get as much pleasure out of things than I used to	1	2	3	4	5
(4) My life is worth living	1	2	3	4	5
(5) I do not feel discouraged about the future	1	2	3	4	5

Factor 2 Composite Score ⇩ ☐

Factor 3

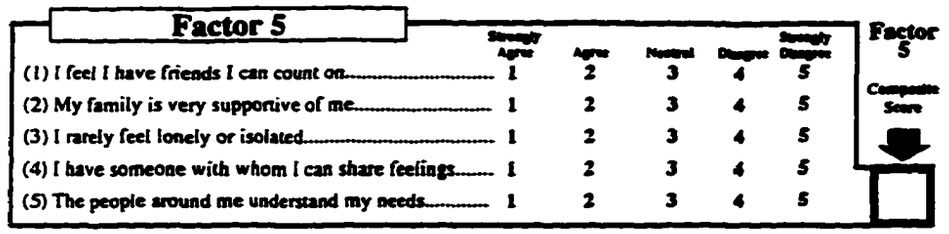
	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
(1) I rarely worry about the time	1	2	3	4	5
(2) I rarely get annoyed when others interrupt me	1	2	3	4	5
(3) It does not bother me to wait in line	1	2	3	4	5
(4) I'm not really very competitive	1	2	3	4	5
(5) I don't eat or speak much faster than others do	1	2	3	4	5

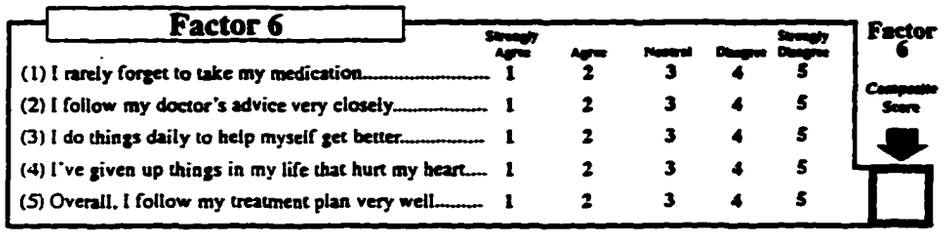
Factor 3 Composite Score ⇩ ☐

Factor 4

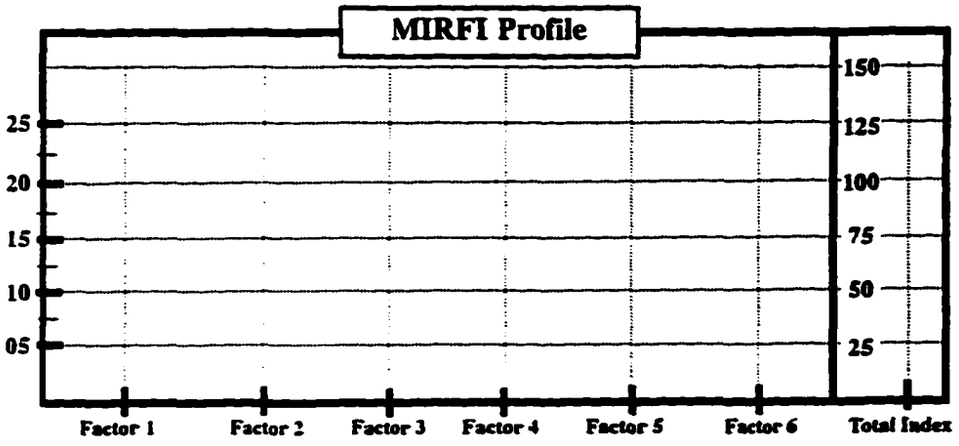
	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
(1) Others would not consider me irritable	1	2	3	4	5
(2) Most people are honest and fair	1	2	3	4	5
(3) I don't often defend my opinions	1	2	3	4	5
(4) I usually forgive and forget	1	2	3	4	5
(5) I enjoy talking with people I do not know well	1	2	3	4	5

Factor 4 Composite Score ⇩ ☐


Factor 5
Strongly Agree
Agree
Neutral
Disagree
Strongly Disagree
(1) I feel I have friends I can count on..... 1 2 3 4 5
(2) My family is very supportive of me..... 1 2 3 4 5
(3) I rarely feel lonely or isolated..... 1 2 3 4 5
(4) I have someone with whom I can share feelings..... 1 2 3 4 5
(5) The people around me understand my needs..... 1 2 3 4 5
Factor 5
Composite Score


Factor 6
Strongly Agree
Agree
Neutral
Disagree
Strongly Disagree
(1) I rarely forget to take my medication..... 1 2 3 4 5
(2) I follow my doctor's advice very closely..... 1 2 3 4 5
(3) I do things daily to help myself get better..... 1 2 3 4 5
(4) I've given up things in my life that hurt my heart..... 1 2 3 4 5
(5) Overall, I follow my treatment plan very well..... 1 2 3 4 5
Factor 6
Composite Score


Total MIRFI Index:


MIRFI Profile
25
20
15
10
05
150
125
100
75
50
25
Factor 1
Factor 2
Factor 3
Factor 4
Factor 5
Factor 6
Total Index

Appendix C

Correlational Matrix for Key Variables for the Follow-Up Group (N=30)

Correlations Among the Major Variables

	AGE	DAS1	DAS2	DAS3	DAS4	DAS5	IDD Tot	T1 MIRFI 1	T1 MIRFI 2	T1 MIRFI 3	T1 MIRFI 4	T1 MIRFI 5
AGE	1 000	193	230	- 154	118	182	- 050	- 131	152	093	049	- 011
DAS 1	193	1 000	827**	503*	170	885**	- 395	- 712**	- 313	- 128	- 093	- 577*
DAS 2	230	827**	1 000	503*	410	928**	- 347	- 806**	- 412	- 067	022	- 490*
DAS 3	- 154	503*	503*	1 000	385	676**	- 208	- 570**	- 204	- 116	- 066	- 371
DAS 4	118	170	410	385	1 000	558**	089	- 262	- 146	424	- 188	- 275
DAS ODA	182	885**	928**	676**	558**	1.000	- 315	- 777**	- 363	015	- 100	- 577*
IDD Total	- 050	- 395	- 347	- 208	089	- 315	1 000	222	721**	485**	436*	543*
TIMIRFI 1 (Mar. Sat.)	- 131	- 712**	- 806**	- 570**	- 262	- 777**	222	1.000	313	179	- 078	543*
TIMIRFI 2 (Dep.)	152	- 313	- 412	- 204	- 146	- 363	721**	313	1 000	563**	425*	657*
TIMIRFI 3 (Type A)	093	- 128	- 067	- 116	424	015	485**	179	563**	1 000	550**	414*
TIMIRFI 4 (Hostility)	049	- 093	022	- 066	- 188	- 100	436*	- 078	425*	550**	1 000	498**
TIMIRFI 5 (Social S.)	- 011	- 577**	- 490*	- 371	- 275	- 577**	543**	543*	657**	414*	498**	1 000
TIMIRFI 6 (Tx Comp)	023	- 416	- 296	- 136	- 132	- 357	353	- 016	273	284	348	061
TIMIRFI Adjusted	127	- 484*	- 403	- 291	- 102	- 443*	608**	332	682**	528**	586**	654*
TIMIRI Chest Pain	- 120	035	- 152	097	- 234	- 091	- 229	058	- 038	- 219	- 276	- 183
T2 Dep. Gauge	117	428	147	268	- 122	264	- 444*	- 168	- 080	- 028	- 129	- 278
T2 Marital Gauge	- 009	178	322	042	312	382	210	- 288	398*	267	183	025
T2 MIRFI 1 (Mar. Sat.)	- 369	- 468*	- 463*	- 094	- 151	- 437*	- 078	437*	- 176	- 254	- 328	172
T2 MIRFI 2 (Dep.)	- 083	- 399	- 104	- 119	- 105	- 278	420*	- 032	193	084	387*	415*
T2 MIRFI 3 (Type A)	- 180	- 036	006	260	442*	148	418*	- 216	281	437*	303	184
T2 MIRFI 4 (Hostility)	125	184	289	151	033	219	381*	- 450*	288	284	499**	071
T2 MIRFI 5 (Social S.)	- 003	- 305	- 277	- 130	- 182	- 311	620**	063	410*	284	491**	507*
T2 MIRFI 6 (Tx Com)	- 445*	- 463*	- 371	- 100	- 015	- 372	166	084	- 049	- 112	028	- 046
T2 MIRFI Adjusted	- 192	- 356	- 177	000	036	- 220	621**	- 110	349	250	480**	366*
T2 MIRI Total	084	268	054	- 107	120	155	- 442*	070	- 221	122	- 216	- 420*
AGE		301	296	506	609	431	796	570	431	633	801	955
DAS 1	301		000	021	462	000	077	000	167	581	687	006
DAS 2	296	000		020	065	000	123	000	065	775	924	024
DAS 3	506	021	020		085	001	367	007	376	616	778	098
DAS 4	609	462	065	085		009	700	252	527	056	414	227
DAS ODA	431	000	000	001	009		164	000	106	950	668	006
IDD Total	796	077	123	367	700	164		334	000	007	016	002
TIMIRFI 1 (Mar. Sat.)	570	000	000	007	252	000	334		167	438	737	011
TIMIRFI 2 (Dep.)	431	167	065	376	527	106	000	167		001	019	000
TIMIRFI 3 (Type A)	633	581	775	616	056	950	007	438	001		001	023
TIMIRFI 4 (Hostility)	801	687	924	778	414	668	016	737	019	001		005
TIMIRFI 5 (Social S.)	955	006	024	098	227	006	002	011	000	023	005	
TIMIRFI 6 (Tx Comp)	905	061	193	555	569	112	056	944	145	128	059	747
TIMIRFI Adjusted	512	026	070	201	654	044	000	142	000	003	001	000
TIMIRI Chest Pain	552	885	536	692	334	712	242	814	849	263	155	351
T2 Dep. Gauge	547	093	923	240	598	248	014	465	673	884	496	157
T2 Marital Gauge	066	092	154	858	168	087	314	205	049	197	382	695
T2 MIRFI 1 (Mar. Sat.)	099	012	015	000	514	047	712	048	399	220	109	412
T2 MIRFI 2 (Dep.)	008	071	055	001	029	230	021	000	307	059	035	023
T2 MIRFI 3 (Type A)	190	878	900	256	045	521	022	347	133	016	004	329
T2 MIRFI 4 (Hostility)	520	425	205	513	887	340	038	041	122	128	005	709
T2 MIRFI 5 (Social S.)	087	178	228	573	431	170	000	786	024	128	006	004
T2 MIRFI 6 (Tx Com)	015	035	098	008	879	000	381	716	797	557	882	808
T2 MIRFI Adjusted	319	113	343	1 000	877	337	000	636	059	183	006	047
T2 MIRI Total	007	280	497	644	000	501	014	763	240	522	252	021
AGE	29	21	21	21	21	21	29	21	29	29	29	29
DAS 1	21	21	21	21	21	21	21	21	21	21	21	21
DAS 2	21	21	21	21	21	21	21	21	21	21	21	21
DAS 3	21	21	21	21	21	21	21	21	21	21	21	21

Correlations Among the Major Variables

	AGE	DAS1	DAS2	DAS3	DAS4	DAS5	IDD Tot	T1 MIRFI 1	T1 MIRFI 2	T1 MIRFI 3	T1 MIRFI 4	T1 MIRFI 5
DAS 4	21	21	21	21	21	21	21	21	21	21	21	21
DAS ODA	21	21	21	21	21	21	21	21	21	21	21	21
IDD Total	29	21	21	21	21	21	30	21	30	30	30	30
T1MIRFI 1 (Mar. Sat.)	21	21	21	21	21	21	21	21	21	21	21	21
T1MIRFI 2 (Dep.)	29	21	21	21	21	21	30	21	30	30	30	30
T1MIRFI 3 (Type A)	29	21	21	21	21	21	30	21	30	30	30	30
T1MIRFI 4 (Hostility)	29	21	21	21	21	21	30	21	30	30	30	30
T1MIRFI 5 (Social S.)	29	21	21	21	21	21	30	21	30	30	30	30
T1MIRFI 6 (Tx Comp)	29	21	21	21	21	21	30	21	30	30	30	30
T1MIRFI Adjusted	29	21	21	21	21	21	30	21	30	30	30	30
T1MIRI Chest Pain	27	19	19	19	19	19	28	19	28	28	28	28
T2 Dep. Gauge	29	21	21	21	21	21	30	21	30	30	30	30
T2 Marital Gauge	25	21	21	21	21	21	25	21	25	25	25	25
T2 MIRFI 1 (Mar. Sat.)	25	21	21	21	21	21	25	21	25	25	25	25
T2 MIRFI 2 (Dep.)	29	21	21	21	21	21	30	21	30	30	30	30
T2 MIRFI 3 (Type A)	29	21	21	21	21	21	30	21	30	30	30	30
T2 MIRFI 4 (Hostility)	29	21	21	21	21	21	30	21	30	30	30	30
T2 MIRFI 5 (Social S.)	29	21	21	21	21	21	30	21	30	30	30	30
T2 MIRFI 6 (Tx Com)	29	21	21	21	21	21	30	21	30	30	30	30
T2 MIRFI Adjusted	29	21	21	21	21	21	30	21	30	30	30	30
T2 MIRI Total	29	21	21	21	21	21	30	21	30	30	30	30

Correlations Among the Major Variables

	T1 MIRFI 6	T1 MIRFI Adj	T1 MIRI Chest	T2 Dep Gauge	T2 Gauge Mar Sat	T2 MIRFI 1	T2 MIRFI 2	T2 MIRFI 3	T2 MIRFI 4	T2 MIRFI 5	T2 MIRFI 6	T2 MIRFI Adj
AGE	023	127	-120	117	-009	-369	-083	-180	125	-003	-445*	-192
DAS 1	-416	-484*	035	428	378	-468*	-390	-036	184	-305	-463*	-356
DAS 2	-296	-403	-152	147	322	-465*	-106	006	289	-277	-371	-177
DAS 3	-136	-291	097	268	042	-094	-119	260	151	-130	-100	000
DAS 4	-132	-102	-234	-122	312	-151	-105	442*	033	-182	-035	036
DAS ODA	-357	-443*	-091	264	382	-437*	-274	148	219	-311	-372	-220
IDD Total	353	488**	-229	-444*	210	-078	420*	418*	381*	620**	166	621**
T1MIRFI 1 (Mar Sat.)	-016	332	058	-168	-288	437*	-032	-216	-450*	063	084	-110
T1MIRFI 2 (Dep.)	273	682**	-038	-080	398*	-176	193	281	288	410*	-049	349
T1MIRFI 3 (Type A)	284	528**	-219	-028	267	-254	084	437*	284	284	-112	250
T1MIRFI 4 (Hostility)	348	586**	-276	-159	183	-328	387*	303	499**	491**	028	489**
T1MIRFI 5 (Social S.)	061	654**	-183	-278	025	172	415*	184	071	507**	-046	366*
T1MIRFI 6 (Tx Comp)	1 000	374*	037	-135	-108	-083	325	031	242	360	314	356
T1MIRFI Adjusted	374*	1 000	-108	-216	371	-209	284	193	121	386*	052	309
T1MIRI Chest Pain	037	-108	1 000	306	-051	211	-272	-268	-366	-323	177	-273
T2 Dep. Gauge	-135	-216	306	1 000	255	-257	-731**	-356	-211	-403*	-230	-581**
T2 Marital Gauge	-108	371	-051	255	1.000	-811**	-472*	112	-081	-382	-028	-259
T2 MIRFI 1 (Mar Sat.)	-083	-209	211	-257	-811**	1 000	312	-103	-282	266	294	155
T2 MIRFI 2 (Dep.)	325	284	-272	-731**	-472*	312	1.000	292	351	609**	330	762**
T2 MIRFI 3 (Type A)	031	193	-268	-356	112	-103	292	1 000	472**	449*	185	684**
T2 MIRFI 4 (Hostility)	242	121	-366	-211	-081	-282	351	472**	1 000	597**	085	698**
T2 MIRFI 5 (Social S.)	360	386*	-323	-403*	-382	266	609**	449*	597**	1 000	048	788**
T2 MIRFI 6 (Tx Com)	314	052	177	-230	-028	294	330	185	085	048	1 000	468**
T2 MIRFI Adjusted	356	309	-273	-581**	-259	155	762**	684**	698**	788**	468**	1 000
T2 MIRI Total	-147	-350	160	581**	380	-325	-699**	-173	-364*	-567**	-215	-614**
AGE	905	512	552	547	906	069	068	350	520	987	015	319
DAS 1	061	026	885	053	092	032	073	878	425	178	035	113
DAS 2	193	070	536	524	154	035	655	980	205	224	098	443
DAS 3	555	201	692	240	858	686	607	256	513	573	068	1 000
DAS 4	569	659	354	598	166	515	649	045	887	431	879	877
DAS ODA	112	043	712	248	087	047	230	521	340	170	096	337
IDD Total	056	000	242	014	314	712	021	022	038	000	381	000
T1MIRFI 1 (Mar Sat.)	044	142	814	465	205	048	890	347	041	786	716	616
T1MIRFI 2 (Dep.)	145	000	849	673	049	369	307	133	122	024	797	059
T1MIRFI 3 (Type A)	128	003	263	884	197	220	659	016	128	128	557	183
T1MIRFI 4 (Hostility)	059	001	155	496	382	109	035	104	005	006	882	006
T1MIRFI 5 (Social S.)	747	000	351	137	905	412	023	329	709	004	808	047
T1MIRFI 6 (Tx Comp)		042	851	477	048	692	079	870	198	051	091	053
T1MIRFI Adjusted	042		585	252	068	317	128	306	525	035	786	097
T1MIRI Chest Pain	851	585		292	818	335	162	168	055	094	367	159
T2 Dep Gauge	477	252	292		218	215	000	054	263	027	221	001
T2 Marital Gauge	906	068	818	218		000	017	595	699	060	894	212
T2 MIRFI 1 (Mar Sat.)	692	317	335	215	000		129	623	172	199	154	459
T2 MIRFI 2 (Dep.)	079	128	162	000	017	129		117	057	000	078	000
T2 MIRFI 3 (Type A)	870	306	168	054	595	623	117		008	013	327	000
T2 MIRFI 4 (Hostility)	198	525	055	263	699	172	057	008		000	654	000
T2 MIRFI 5 (Social S.)	051	035	094	027	060	199	000	013	000		801	000
T2 MIRFI 6 (Tx Com)	091	786	367	221	894	154	078	327	654	801		009
T2 MIRFI Adjusted	053	097	159	000	212	459	000	000	000	000	009	
T2 MIRI Total	417	058	415	000	060	113	000	362	048	001	253	000
AGE	29	29	27	29	25	25	29	29	29	29	29	29
DAS 1	21	21	19	21	21	21	21	21	21	21	21	21
DAS 2	21	21	19	21	21	21	21	21	21	21	21	21
DAS 3	21	21	19	21	21	21	21	21	21	21	21	21

Correlations Among the Major Variables

	T1 MIRFI 6	T1 MIRFI Adj	T1 MIRI Chest	T2 Dep Gauge	T2 Gauge Mar Sat	T2 MIRFI 1	T2 MIRFI 2	T2 MIRFI 3	T2 MIRFI 4	T2 MIRFI 5	T2 MIRFI 6	T2 MIRFI Adj
DAS 4	21	21	19	21	21	21	21	21	21	21	21	21
DAS ODA	21	21	19	21	21	21	21	21	21	21	21	21
IDD Total	30	30	28	30	25	25	30	30	30	30	30	30
T1MIRFI 1 (Mar. Sat.)	21	21	19	21	21	21	21	21	21	21	21	21
T1MIRFI 2 (Dep.)	30	30	28	30	25	25	30	30	30	30	30	30
T1MIRFI 3 (Type A)	30	30	28	30	25	25	30	30	30	30	30	30
T1MIRFI 4 (Hostility)	30	30	28	30	25	25	30	30	30	30	30	30
T1MIRFI 5 (Social S.)	30	30	28	30	25	25	30	30	30	30	30	30
T1MIRFI 6 (Tx Comp)	30	30	28	30	25	25	30	30	30	30	30	30
T1MIRFI Adjusted	30	30	28	30	25	25	30	30	30	30	30	30
T1MIRI Chest Pain	28	28	28	28	23	23	28	28	28	28	28	28
T2 Dep. Gauge	30	30	28	30	25	25	30	30	30	30	30	30
T2 Marital Gauge	25	25	23	25	25	24	25	25	25	25	25	25
T2 MIRFI 1 (Mar. Sat.)	25	25	23	25	24	25	25	25	25	25	25	25
T2 MIRFI 2 (Dep.)	30	30	28	30	25	25	30	30	30	30	30	30
T2 MIRFI 3 (Type A)	30	30	28	30	25	25	30	30	30	30	30	30
T2 MIRFI 4 (Hostility)	30	30	28	30	25	25	30	30	30	30	30	30
T2 MIRFI 5 (Social S.)	30	30	28	30	25	25	30	30	30	30	30	30
T2 MIRFI 6 (Tx Com)	30	30	28	30	25	25	30	30	30	30	30	30
T2 MIRFI Adjusted	30	30	28	30	25	25	30	30	30	30	30	30
T2 MIRI Total	30	30	28	30	25	25	30	30	30	30	30	30

Correlations Among the Major Variables

	T2 MIRI Tot
AGE	084
DAS 1	268
DAS 2	034
DAS 3	-107
DAS 4	120
DAS ODA	155
IDD Total	-442*
T1MIRFI 1 (Mar Sat.)	070
T1MIRFI 2 (Dep)	-221
T1MIRFI 3 (Type A)	122
T1MIRFI 4 (Hostility)	-216
T1MIRFI 5 (Social S)	-420*
T1MIRFI 6 (Tx Comp)	-147
T1MIRFI Adjusted	-350
T1MIRI Chest Pain	160
T2 Dep. Gauge	581*
T2 Marital Gauge	380
T2 MIRFI 1 (Mar. Sat.)	-325
T2 MIRFI 2 (Dep)	-699*
T2 MIRFI 3 (Type A)	-173
T2 MIRFI 4 (Hostility)	-364*
T2 MIRFI 5 (Social S.)	-567*
T2 MIRFI 6 (Tx Com)	-215
T2 MIRFI Adjusted	-614*
T2 MIRI Total	1 000
AGE	667
DAS 1	240
DAS 2	885
DAS 3	644
DAS 4	606
DAS ODA	501
IDD Total	014
T1MIRFI 1 (Mar Sat)	763
T1MIRFI 2 (Dep)	240
T1MIRFI 3 (Type A)	522
T1MIRFI 4 (Hostility)	252
T1MIRFI 5 (Social S)	021
T1MIRFI 6 (Tx Comp)	437
T1MIRFI Adjusted	058
T1MIRI Chest Pain	415
T2 Dep Gauge	001
T2 Marital Gauge	061
T2 MIRFI 1 (Mar Sat)	113
T2 MIRFI 2 (Dep)	000
T2 MIRFI 3 (Type A)	362
T2 MIRFI 4 (Hostility)	048
T2 MIRFI 5 (Social S)	001
T2 MIRFI 6 (Tx Com)	253
T2 MIRFI Adjusted	000
T2 MIRI Total	
AGE	29
DAS 1	21
DAS 2	21
DAS 3	21

Correlations Among the Major Variables

	T2 MIRI Tot
DAS 4	21
DAS ODA	21
IDD Total	30
T1MIRFI 1 (Mar. Sat.)	21
T1MIRFI 2 (Dep.)	30
T1MIRFI 3 (Type A)	30
T1MIRFI 4 (Hostility)	30
T1MIRFI 5 (Social S.)	30
T1MIRFI 6 (Tx Comp)	30
T1MIRFI Adjusted	30
T1MIRI Chest Pain	28
T2 Dep. Gauge	30
T2 Marital Gauge	25
T2 MIRFI 1 (Mar. Sat.)	25
T2 MIRFI 2 (Dep.)	30
T2 MIRFI 3 (Type A)	30
T2 MIRFI 4 (Hostility)	30
T2 MIRFI 5 (Social S.)	30
T2 MIRFI 6 (Tx Com)	30
T2 MIRFI Adjusted	30
T2 MIRI Total	30

* Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).

Appendix D

Figures

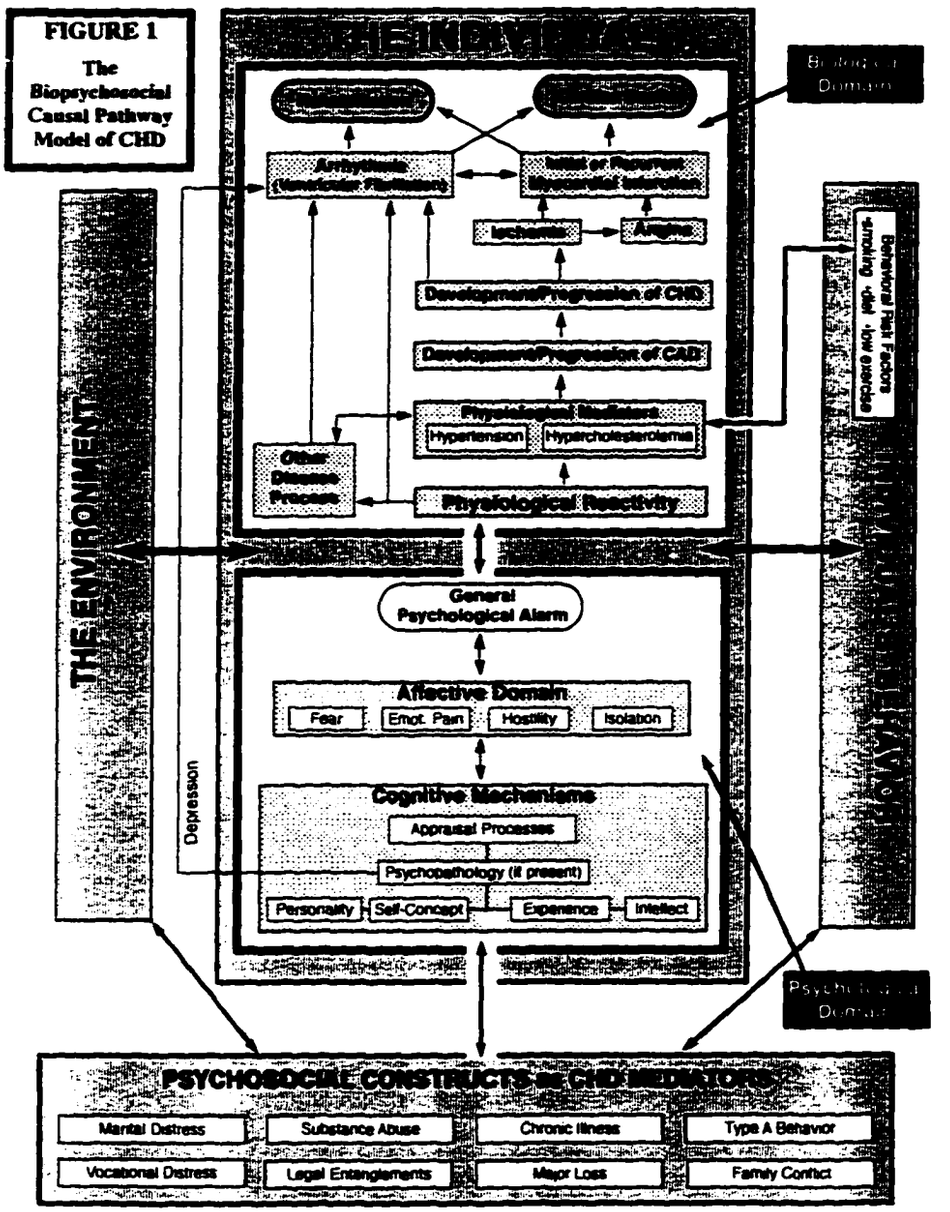
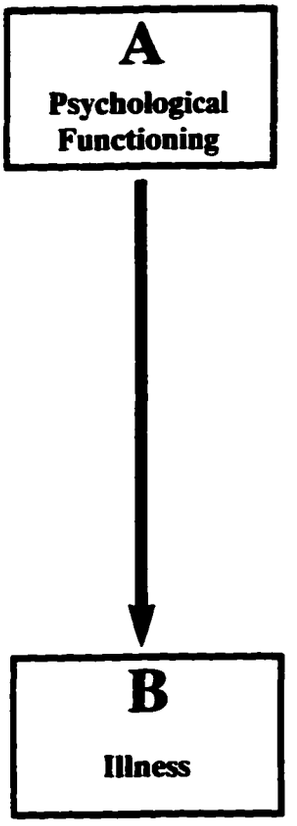


FIGURE 1
The Biopsychosocial Causal Pathway Model of CHD
Arrhythmia
Ischemia
Angina
Development/Progression of CHD
Development/Progression of CAD
Hypertension
Hypercholesterolemia
Physiological Reactivity
Behavioral Risk Factors
smoking diet low exercise
THE ENVIRONMENT
General Psychological Alarm
Affective Domain
Fear
Emot. Pain
Hostility
Isolation
Depression
Cognitive Mechanisms
Appraisal Processes
Psychopathology (if present)
Personality
Self-Concept
Experience
Intellect
PSYCHOSOCIAL CONSTRUCTS as CHD MEDIATORS
Marital Distress
Substance Abuse
Chronic Illness
Type A Behavior
Vocational Distress
Legal Entanglements
Major Loss
Family Conflict

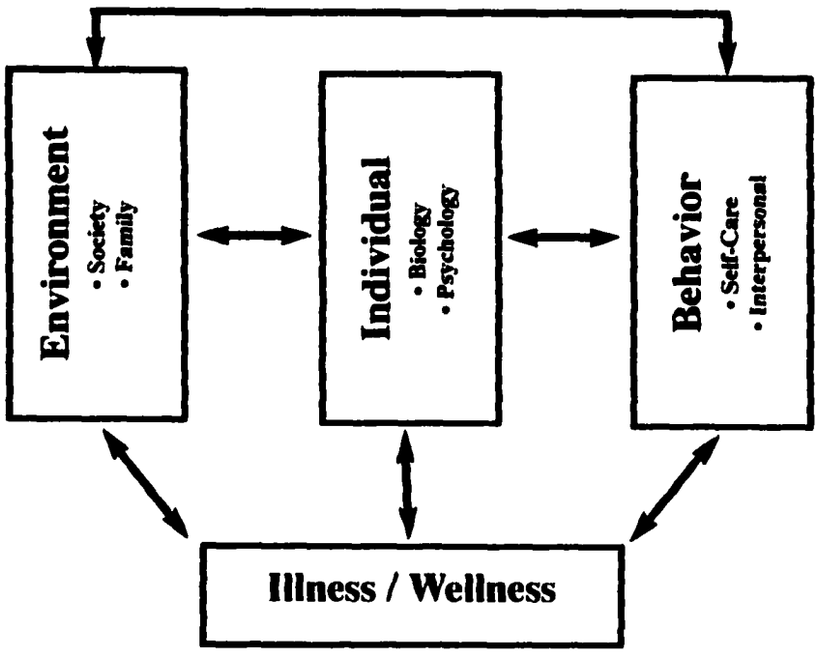
Causal Pathway Modeling

How do you get from Point A to Point B?

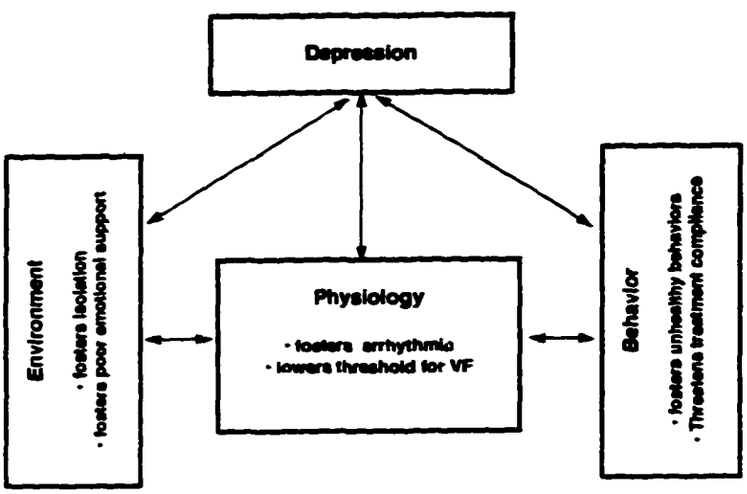


Biopsychosocial Model of Illness/Wellness

(Closely resembles Bandura's Triadic Reciprocality Model)



Depression's Link to Poor Cardiac Recovery



Link Between Marital Satisfaction & Cardiac Recovery