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Coronary Heart Disease, Negative Affectivity, and Response Bias.

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Louisiana State University and Agricultural & Mechanical College

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Coronary heart disease, negative affectivity, and response bias

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The Louisiana State University and Agricultural and Mechanical Col., 1990
Coronary Heart Disease, Negative Affectivity, and Response Bias

A Dissertation

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

by

Sharon Elaine Alcock Parisi M.A., Vanderbilt University, 1977 Summer, 1990
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Abstract

The purpose of this study was to examine the relation between negative affectivity, a broad band construct of negative emotions, and coronary heart disease. It was hypothesized the relation had been obscured in previous research by the tendency of some subjects to underreport negative affects. To test this hypothesis, 78 male cardiac patients between to ages of 37 and 65, who had undergone cardiac arteriography procedures within the previous 6 months, were given the Taylor Manifest Anxiety Scale Short Form (TMAS-S; Bendig, 1956) and the Marlowe-Crowne Social Desirability Scale Form C (MCSD-C; Reynolds, 1982). In a series of multiple regression analyses, TMAS-S and MCSD-C scores did not account for a significant degree of variance in degree of cardiac stenosis. Multiple regression of factors of age, family history of heart disease, smoking, and diagnosis of hypertension or diabetes also failed to account for a significant amount of the variance in degree of stenosis, and the addition of TMAS-S and MCSD-C scores did not improve the amount of variance accounted for. In contrast, while TMAS-S scores alone were not related to extent of self-report of pain, the multiple regression of TMAS-S and MCSD-C scores accounted for a significant degree of variance in self-reports of pain, with the interaction of TMAS-S and MCSD-C scores accounting for the greatest amount of variance. The previously noted demographic and medical
factors did not significantly account for variance in self-report of pain. The potential biases in this type of cross-sectional study and their possible impact on the outcome were discussed.
The American Heart Association (1988) estimates that almost five million Americans have coronary heart disease (CHD), a condition in which heart muscle is damaged secondary to inadequate blood flow to the heart tissue. Decreased coronary blood flow can lead to angina pectoris (AP), or chest pain, or myocardial infarction (MI), or heart attack. Approximately 1,500,000 Americans are victims of heart attacks each year, and of these approximately 540,800 do not survive.

A leading cause of decreased coronary blood flow, and hence major contributor to CHD, is atherosclerosis, a process in which plaque, a paste-like fatty substance composed of cholesterol and other materials, builds up on the inner linings of the coronary arteries, resulting in narrowing of the coronary arteries. Most heart attacks occur when blood flow through these arteries is severely or totally reduced, due to obstruction by either the plaque itself or by a blood clot on the surface of the plaque. As the duration of obstruction increases, the affected heart tissue is irreversibly damaged, resulting in disability or death of the individual.

Research on the Type A Behavior Pattern (TABP)

While there has been speculation for many years that psychological factors might be associated with CHD, research in the area was relatively diffuse and lacking in focus.
until 1959, when cardiologists Friedman and Rosenman described a behavior pattern they had noticed in many of their patients, which they referred to as the Type A behavior pattern (TABP). As described by Friedman and Rosenman, this pattern includes aggressively competitive behaviors, very rapid speech and motor behaviors, and a heightened sense of time urgency, or a pressure to accomplish as much as possible in a given amount of time. Since publication of Friedman and Rosenman's book, much of the research on psychological factors and CHD has revolved around the construct of TABP. (For a review of those studies and the issues involved, see Matthews and Haynes, 1986, and Haynes and Matthews, 1988.) In the majority of studies, TABP has been assessed through either the Structured Interview (SI; Rosenman, 1978) or the Jenkins Activity Scale (JAS; Jenkins, Zyzanski, & Rosenman, 1971, 1978, 1979). The SI is a semi-structured interview in which subjects are asked about their usual way of responding to situations that might elicit impatience, competition, or hostility. The interview itself is conducted in a manner to elicit type A behaviors from the subject, and classification of the subject is based on the combination of self-report and actual behaviors within the interview. Rate and volume of speech, verbal explosiveness, and other measures of nonverbal emotion expression are specifically rated. The JAS, on the other hand, is a paper-and-pencil self-report
questionnaire. In addition to the global Type A rating, the JAS includes 3 subscales of Speed and Impatience, Job Involvement, and Hard-driving Competitiveness.

Until very recently, most studies in the area have defined CHD in terms of angina symptoms (with studies differing on exact criteria), the occurrence of a heart attack (with various degrees of medical verification required), and/or the occurrence of sudden cardiac death. Using such criteria for diagnostic classifications, five major longitudinal studies of initially healthy individuals have been conducted over the past 20 years. These have included the Western Collaborative Group Study (Jenkins, Rosenman, & Zyzanski, 1974; Rosenman et al., 1975), the Framingham Heart Study (Haynes & Feinleib, 1982; Haynes, Feinleib, & Kannel, 1980), the Honolulu Heart Study (Cohen & Reed, 1985), the Belgian French Cooperative Heart Study (French-Belgian Cooperative Group, 1982), and the Belgian Heart Disease Prevention Trial (DeBacker, Kornitzer, Kittel, & Dramaix, 1983). As summarized by Haynes and Matthews (1988), four of these studies indicated a positive relation between TABP and CHD. (The exception was the Honolulu Heart Study, which was conducted with Japanese men who demonstrated low incidence rates of both CHD and TABP.) Indeed, it was concluded in 1978 by a National Heart, Lung, and Blood Institute research review panel that TABP was as serious a risk factor for CHD as smoking, hypertension, or
elevated cholesterol levels (Cooper, Detre, & Weiss, 1981).

More recently, however, the association between TABP and CHD has been the subject of increasing debate (e.g., Ragland & Brand, 1988). While longitudinal studies of initially healthy individuals suggest a positive relation between TABP and CHD, studies with individuals who have already sustained an MI suggest that TABP is not a risk factor for recurrent MIs (Case, Heller, Case, & Moss, 1985; Shekelle, Gale, & Norusis, 1985); nor is it an added risk factor for development of CHD in individuals who are considered at risk due to other factors, such as high cholesterol levels, high blood pressure, or cigarette smoking (Dimsdale, Block, Gilbert, Hackett, & Hutter, 1981; Shekelle, Hulley, et al., 1985).

A second source of the current debate has stemmed from the development and widespread use of coronary angiography. In this diagnostic procedure, contrast fluid is injected into the arteries and veins of the heart through a catheter. High-speed x-rays of the flow of the fluid through the heart allow determination of the extent of actual coronary stenosis. Using this procedure, some investigators have reported normal coronary arteries in some angina patients (e.g., Kemp, Elliot, & Gorlin, 1967; Likoff, Segal, & Kasparian, 1967; Wielgosz and Earp, 1986). These results have called into question the conclusions of studies of TABP and CHD in which CHD was defined solely on the basis of
self-reported angina. In addition, the results of recent studies on the associations between TABP and extent of angiographically demonstrated CHD have been considerably less conclusive than were the results of earlier studies that defined CHD on less objective criteria, such as physician judgement, self-report of pain, or EKG results. Of 16 angiography studies reviewed by Haynes and Matthews (1988), and a 17th study by Smith, Korr, Follick, and McCartney (1986), only 6 studies using the SI and 1 using the JAS reported a positive relation between TABP and extent of coronary stenosis. Negative results were found in 4 studies with the SI, 5 with the JAS, and 4 using other measures of TABP. (The total is greater than 17, as some studies used more than 1 measure of TABP). Haynes and Matthews (1988) state, in fact, that there is a "...growing recognition among behavioral scientists that Type A behavior is no longer a risk factor for coronary artery disease" (p. 47).

Booth-Kewley and Friedman (1987) and Haynes and Matthews (1988) suggest several explanations for the shift in results on TABP and CHD. These include subtle methodological changes, such as criteria drift in the SI or changes in experimenter expectancies. These factors could influence the number of people who are classified as Type A with the SI, as such classification is based upon subjective ratings, which in turn could be influenced by how the
interviewer conceptualizes TABP. Another group of explanations involves possible changes in the research climate, such that negative results are now being accepted more readily for publication. And finally, there is the possibility that the phenomena itself may have changed in ways that are more specific to Type A individuals than to Type B individuals. For example, Type A's may have improved their health habits, such as increased exercise or decreased smoking, possibly as a result of self-identification as Type A.

More recent research in this area has been directed towards investigating possible associations between CHD and specific components of the Type A construct. Components investigated have included the 3 JAS subscales of speed and impatience, job involvement, and competitiveness, and over 40 content and response style elements of the SI. The results of some of this research has indicated that anger or hostility may be the toxic factor in the TABP as measured by the SI. For example, Matthews, Glass, Rosenman, and Bortner (1977) analyzed 40 SI response items of CHD subjects and healthy controls who were included in the Western Collaborative Group Study. Of 8 items discriminating between the two groups of subjects, 7 were related to anger and hostility. Barefoot, Dahlstrom, and Williams (1983) found that physicians who had received high scores on the Cook-Medley Hostility Inventory during their years in
medical school were significantly more likely to have developed CHD over the subsequent 25 years than were individuals who had scored lower on the test. And Shekelle, Gale, Ostfeld, and Oglesby (1983) found in a 10-year longitudinal study that high scores on the Cook-Medley were related to subsequent development of CHD. Other research in this area is now in progress. (For thorough reviews of this literature, with discussions of issues and implications, see Chesney & Rosenman [1985] and Schmidt, Dembroski, & Blumchen [1986].)

**CHD and Depression and Anxiety**

Friedman and Booth-Kewley (1987b) argue that, in addition to including components that are not associated with CHD, the Type A construct may exclude some factors that are related to CHD, factors which may have been overlooked in the flurry of Type A research. These investigators (Friedman and Booth-Kewley, 1987b; Booth-Kewley and Friedman, 1987) conducted a series of meta-analyses on articles published between 1945 and 1984, using various diagnostic criteria for CHD, and various combinations of potential psychological factors. Factors under consideration included global Type A behavior (as measured by either the SI or the JAS), subscales of the SI and JAS, other measures of Type A factors (time urgency, job involvement, and competitiveness/hard driving/aggressiveness), anger, hostility, aggression,
depression, anxiety, and extroversion. (Factors were defined on the basis of the terms used by the authors of the original studies.) CHD diagnostic criteria included global cardiovascular disease, myocardial infarction, angina, cardiac death, and atherosclerosis.

Surprisingly, in the meta-analysis of all studies, the largest effect size found was attributable to depression ($r = .225, p < .0000001$), despite the fact that few studies were available that included depression measures (11 independent effect sizes). In comparison, Type A behavior pattern, as measured by the SI, was found to have a combined effect size of .197 ($p < .0000001$), while combined effect size for all measures of TABP was .112 ($p < .0000001$). A combined factor of anger/hostility had a combined effect size of .158 ($p < .0000001$), while anxiety was found to have an effect size of .136 ($p < .0000001$).

Separate meta-analyses were also conducted on cross-sectional and longitudinal studies. Type A behavior pattern, as measured by the SI, was found to have the largest effect size in predicting CHD in cross-sectional studies ($r = .238, p < .0000001$), while depression was found to have a combined effect size of .204 ($p < .0000001$). The combined anger/hostility factor had an effect size of .169 ($p < .0000001$), and anxiety was found to have an effect size of .122 ($p < .00002$).

A very different pattern was found in the meta-
analysis of longitudinal studies. In that analysis, SI-measured TABP was found to have a combined effect size of only .062 (p <.0001). Depression, on the other hand, yielded a combined effect size of .168 (p <.00008), while anxiety was found to have a combined effect size of .136 (p <.000001). Combined anger and hostility was found to have an effect size of only .074 (p <.0130), while hostility alone was found to have an effect size of .135 (p <.00004). Unfortunately, no firm conclusions about the role of depression or anxiety could be drawn from these results, as so few prospective studies have considered these factors. However, the results suggest that depression and anxiety deserve further investigation as potential factors in CHD.

Like other studies in the area of CHD, those studies that have included measures of depression and/or anxiety have differed with respect to sample characteristics, control groups, CHD diagnostic criteria, assessment instruments, and methods of statistical analyses. The majority of the studies have been cross-sectional, and some have been retrospective in nature. Other design weaknesses have included the use of non-standardized assessment procedures and ratings by experimenters who were not blind to the diagnoses of the subjects. In spite of these shortcomings, the results of Booth-Kewley and Friedman suggest that the studies warrant closer inspection.

In the following review, cross-sectional studies that
included measures of depression and/or anxiety will be considered first, with studies separated into those based on myocardial infarct patients and those based upon results of cardiac arteriograms. The review of cross-sectional studies will be followed by a review of longitudinal studies in the area. Studies in which CHD was defined solely on the basis of self-reported angina will not be reviewed, due to diagnostic problems previously noted.

**Cross-sectional Studies**

**Studies of myocardial infarct patients.** The results of four cross-sectional studies have suggested that MI patients are more depressed and/or anxious than are non-cardiac patients. Miller (1965) found that MI subjects were judged to be significantly more depressed, more anxious, and more inwardly hostile than healthy control subjects, when rated via a verbal analysis technique (Gottschalk, 1961). In a multiple regression study of MI subjects and healthy controls, Friedman and Booth-Kewley (1987a) found that maximum predictability was obtained with the combination of either SI Type A behavior and depression, or SI Type A behavior and anxiety. Bianchi, Fergusson, and Walshe (1978) found in a retrospective study that survivors of recent MIs reported significantly more depression and anxiety during the previous 6 months than did age and sex matched controls who had undergone recent surgeries for non-life-threatening conditions (assessed with the State Anxiety Scale;
Spielberger, Gorsuch, & Lushene, 1968; Wakefield Depression Inventory; Snaith, Ahmed, Mehta, & Hamilton, 1971; Cornell Medical Index; Cawte, Bianchi, & Kiloh, 1968). Thiel, Parker, and Bruce (1973) found similar results in another retrospective study that utilized the Bendig Anxiety and Welsh Depression MMPI subscales.

Other investigators have failed to find significant differences between MI patients and controls on the IPAT Anxiety Scale (Segers and Mertens, 1977) and on the Taylor Manifest Anxiety Scale (Wardwell, Bahnson, & Caron, 1963), and the results of a factor analytic study by Croog, Koslowsky, and Levine (1976) suggest that MI is associated with low levels of depression and anxiety. However, as the later study included no comparison group, it is unknown how these levels compared with those of non-cardiac individuals.

Studies using heart catheterization results. Like those studies with MI patients, studies based upon information from cardiac arteriograms have also yielded conflicting results. Zyzanski, Jenkins, Ryan, Flessas, and Everist (1976) found that men who were subsequently found to have 2 or more obstructed arteries scored significantly higher on both the Taylor Manifest Anxiety Scale (Bendig Short Form; Bendig, 1956) and the MMPI Depression scale than did men who were subsequently found to have 0 or 1 occluded vessel. In contrast, Blumenthal, Thompson, Williams, and Kong (1979) found no significant differences in trait
anxiety (as assessed with the Lykken Activity Preference Questionnaire; Lykken & Katzenmeyer, 1967) between groups with and without significant artery disease. However, the criteria used to assign subjects to groups in this study is subject to criticism, as "significant disease" was defined as 75% or greater stenosis in at least one of the four major coronary arteries. Other studies (e.g., Zyzanski et al., 1976) have reported that cutoffs of 50% stenosis and 75% stenosis yield comparable groups, suggesting that the "mild or non-significant disease" group of Blumenthal and his colleagues may have included some individuals who were in fact significantly diseased.

The results of a multiple regression study by Dimsdale, Hutter, Hackett, and Block (1981) are also subject to question. These researchers found that depression and anger (as assessed by the Profile of Mood States; McNair, Lorr, & Droppleman, 1971) were negatively associated with extent of stenosis. These results are surprising in view of the recent research on hostility and CHD, and Dimsdale and his colleagues suggested that the negative loadings might reflect lack of awareness of emotional states on the part of the cardiac patients, rather than actual low levels of anger and/or depression. The authors did not, however, provide evidence to support this interpretation. Similar results were reported by Elias and his colleagues (Elias, Robbins, Blow, Rice, and Edgecomb, 1982) who found that Zung
depression scores and Spielberger trait anxiety scores were negatively correlated with extent of coronary occlusion.

**Longitudinal Studies**

Very few longitudinal studies have included measures of anxiety and/or depression, and the results obtained from those studies must be considered in light of such issues as subject inclusion and exclusion criteria. For example, in a 30-year longitudinal study, Gillum, Leon, Kamp, and Becerra-Aldama (1980), found that no MMPI scale or combination of scales significantly predicted life expectancy, disease onset, or death from cardiovascular disease, cancer, or stroke. However, an inclusion criterion for the study was that all subjects be initially "emotionally stable." This criterion was not further defined in the published report. However, if individuals with elevated MMPI scales were excluded from the study, the range of MMPI scores available for analysis would have been restricted, and the possibility of finding a relation between MMPI scale scores and subsequent disease or death would have been greatly diminished.

In some other longitudinal studies, initial reports have yielded similar negative results. However, subsequent reports on more specific analyses of the same data have suggested a relation between depression and/or anxiety and CHD. For example, in a 1964 report of the Western Electric Longitudinal Study, Ostfeld, Lebovits, Shekelle, and Paul
reported no significant differences on initial MMPI and Sixteen Personality Factor Questionnaire scores (16 PF; Cattell, Saunder, & Stice, 1957) between subjects who developed coronary artery disease within the first 4 1/2 years of study and those who did not. However, in a later analysis, Lebovits, Shekelle, Ostfeld, and Paul (1967) found that subjects who died of MI during the first 5 years of the study had significantly higher elevations on every scale of the initial MMPI except the L and Si, as compared with subjects who survived an MI during that time. In addition, significantly more nonsurvivors than survivors had initial MMPI depression scores greater than 70. There were no significant differences between survivors and non-survivors on age, overt health at initial examination, or number of months until onset of coronary heart disease, nor were there significant MMPI score differences between MI-survivors and their controls. Similar results were obtained by Bruhn, Chandler, and Wolf (1969), who found that subjects who died of an MI during the first 7 years of a longitudinal study had significantly higher MMPI depression scores at the time of entry into the study than did subjects who later survived an MI.

Thomas and her colleagues (Thomas & Greenstreet, 1973; Thomas, Ross, & Duszynski, 1975) presented a series of reports on a longitudinal study of students who entered Johns Hopkins Medical School between 1948 and 1964. Among
other measures, the students were administered the Habits of Nervous Tension checklist, a 25-item questionnaire developed for this study, which provided a measure of overall nervous tension, and subscales of depression, anxiety, and anger experienced when under stress. Total HNT was found to be the second predictor variable in a stepwise multiple discriminant function of those students who experienced myocardial infarction, hypertension, tumor, or mental illness, and who committed suicide, between the time of entry into the study and 1971 (Thomas & Greenstreet, 1973). Depression and anxiety were the sixth and eighth factors, respectively, but F values did not reach significance, possibly due to the correlation between depression and anxiety and the HNT factor. Nevertheless, the percentage of coronary patients correctly classified increased from 62.5% with a four factor solution to 100% with the nine factor solution.

In another study based upon the same subjects, Thomas, Ross, and Duszynski (1975) compared characteristics of subjects who were initially hypercholesterolemic with those of subjects who were initially normocholesterolemic. Hypercholesterolemic subjects reported experiencing significantly less depression, less anxiety, and less overall nervous tension under stress than did normocholesterolemic subjects. However, when the investigators compared subjects who subsequently experienced
an MI (most of whom were initially hypercholesterolemic) with hypercholesterolemic subjects who did not experience MIs, it was found that the MI group had significantly higher scores on depression and overall nervous tension under stress than did the non-MI group. Discriminant analysis revealed that depression was significantly associated with MI. (Depression was the fourth factor of the discriminant function, and was preceded by cholesterol level, age, and height.) The investigators suggested that the combination of high cholesterol level and depression is a better predictor of coronary heart disease than is either of the variables alone. In addition to supporting the hypothesis that depression may be associated with CHD, the results of this study provide an example of the importance of multivariate analysis in this area of study.

In summary, there is some evidence that depression and anxiety are related to CHD, although research has yielded mixed results, and the associations, as previously measured, appear to be modest in degree. The results of the previously discussed meta-analysis by Friedman and Booth-Kewley (1987b) strengthen this conclusion.

**CHD and Negative Affectivity**

Several of the studies thus far reviewed have suggested considerable overlap of anxiety and depression in CHD subjects. For example, in their multiple regression study of MI subjects and healthy controls, Friedman and Booth-
Kewley (1987a) found that maximum predictability was obtained with the combination of either SI Type A behavior and depression, or SI Type A behavior and anxiety, and the researchers therefore suggested the concept of a "depression/anxiety cluster" relation with CHD. Bianchi et al. (1978), who found that MI patients scored significantly higher than did controls on the State Anxiety Scale (Spielberger et al., 1968), the Wakefield Depression Scale (Snaith et al., 1971), and the Cornell Medical Index (Cawte et al., 1968), reported that scores on these three measures were highly intercorrelated, and hypothesized that the instruments were measuring an underlying common factor that they suggested be called "anxiety-depression." Similarly, Thomas and Greenstreet (1973) found that the more global Habits of Nervous Tension score (HNT) was the second factor entered in their discriminant analysis, while the depression and anxiety subscale scores derived from the HNT were the 6th and 8th factors, and were not significant after inclusion of the HNT, suggesting a more general factor was involved. Finally, Thiel et al. (1973), noted that many of their subjects reported symptoms of both anxiety and depression, rather than one or the other.

The results of the meta-analysis of Booth-Kewley and Friedman (1987) suggest that anxiety, depression, anger, hostility, and aggression are all related to CHD. Based upon those results, Booth-Kewley and Friedman hypothesize
that the coronary-prone individual might be one who is experiencing one or more of these negative emotions, rather than an individual who is experiencing a single, specific emotion. They therefore recommend that future research on psychological factors and CHD be directed towards a broader dispositional construct, rather than specific, independent psychological factors.

Many researchers have suggested such a construct, using various names and emphasizing various components. Welsh (1954) reviewed factor analytic studies of the MMPI, and noted that two factors had been found consistently. The Welsh A scale was developed from the first factor, and is typically regarded as a measure of "general maladjustment," (Jessor and Hammond, 1957; Kimble and Posnick, 1967). Welsh described high scores on the A scale as related to disorders involving dysthymia and dysphoria, with prominent anxiety. Others have described a similar general construct, with labels such as "neuroticism" (Eysenck & Eysenck, 1968), "emotionality" (Eysenck & Eysenck, 1975), and "repression-sensitization" (Byrne, 1961). Many investigators have noted the high intercorrelation between many self-report measures of depression and anxiety (e.g., Dinning & Evans, 1977; Dobson, 1985a, 1985b; Gotlib, 1984; Meites, Lovallo, & Pishkin, 1980; Mendels, Weinstein, & Cochrane, 1972), and factor analyses of groups of these instruments have consistently yielded a primary factor which resembles the
first factor of the MMPI (Dobson, 1985a; Gotlib, 1984; Mendels et al., 1972). This has prompted investigators to conclude that the instruments are assessing a common general construct of psychological distress.

Watson and Clark (1984) have used the term "negative affectivity" (NA) to describe what appears to be the same general factor. These researchers define NA as a pervasive disposition to experience undesirable mood states, including anxiety, sadness, anger, scorn, and guilt. Watson and Clark present considerable data on construct validity, including intercorrelations among various instruments which they speculate are measuring the common underlying factor of NA. These measures include the Taylor Manifest Anxiety Scale (Taylor, 1953), the first factor of the MMPI (Welsh, 1954), the Byrne Repression-Sensitization Scale (Byrne, 1961), Block's Ego Resiliency - Obvious scale (Block, 1965), the State-Trait Anxiety Inventory A - Trait scale (Spielberger, Gorsuch, & Lushene, 1970), the Eysenck Personality Inventory Neuroticism scale (Eysenck & Eysenck, 1968), the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), and Lowe's Guilt Scale (Lowe, 1964). The authors also report that the Manifest Hostility Scale (Siegel, 1956) and the Buss-Durkee Hostility Inventory (Buss & Durkee, 1957) are correlated with NA measures in the range of .50 to .65, although they do not give specific intercorrelations between these measures and other NA
measures. Among all measures of NA, the Taylor Manifest Anxiety Scale (TMAS) appears to be the most highly intercorrelated measure. Watson and Clark report that the TMAS is correlated .88 with the Repression-Sensitization Scale, .85 with the Welch A scale, .75 with the Lowe Guilt Scale, .74 with the IPAT Anxiety Scale, .73 with the State-Trait Anxiety Inventory A-Trait scale, .72 with the Eysenck Personality Inventory Neuroticism scale, and .64 with the Beck Depression Scale. Although Watson and Clark report the previously noted correlations between the Manifest Hostility Scale and the Buss-Durkee Hostility Inventory with measures of NA, they do not report correlations between the TMAS and scales designed to measure anger.

In summary, in CHD research, as in personality research, there is evidence of considerable overlap in the information provided by self-report measures of depression and anxiety. Friedman and Booth-Kewley have suggested that a more general factor of negative emotions should be addressed in future research. Such a construct has been suggested by numerous investigators, and supported by correlational and factor analytic studies. Watson and Clark (1984) describe the construct as negative affectivity and propose that it is best assessed by the Taylor Manifest Anxiety Scale.

Potential Role of Response Bias in CHD Research

A second issue in the study of CHD and psychological
factors was addressed by Linden (1988) in a rejoinder to Friedman and Booth-Kewley's meta-analytic study (1987b). Linden applauds Friedman and Booth-Kewley's recommendations for future research, but argues that future researchers must also consider the potential impact of response bias in the self-report data used in those studies. He points out that Paulhus (1984) suggested that self-report can be confounded by both impression management, or the attempt to make oneself look a particular way, and by self-deceptive/repressive tendencies, or "the stylistic tendency ... to avoid or ignore threatening information" (Linden, Paulhus, & Dobson, 1986, p. 309). Such confounding has been demonstrated in the self-report of somatic, as well as psychological symptoms (Linden et al., 1986). If some individuals typically underreport psychological symptoms, then the meaning of low scores on self-report measures is unclear, as low scores can reflect either lack of symptoms, or underreporting of symptoms that are indeed experienced. Such response bias, if present, will have a direct impact on the degree of relation found statistically between CHD and psychological factors under consideration. Specifically, if CHD is related to a psychological factor, but some individuals with CHD underreport psychological symptoms, then the statistical correlation between angiographically demonstrated stenosis and self-report of psychological symptoms will be diminished by the extent to which the CHD
Linden also notes that there is some evidence that, in addition to confounding self-reports, self-deception/repression may itself be a correlate of physical disease (Schwartz, Krupp, & Byrne, 1971). He therefore recommends research designs that permit consideration of potential psychological factor by response style interactions, and the effect of those interactions on disease, rather than statistical control of response style through analysis of covariance or partial correlation.

Few, if any, CHD studies have included Linden's recommendations. However, there has been a parallel line of research within the personality and social psychology literature, the methods and results of which may contribute to our understanding of the relation between psychological factors and CHD.

Like many other researchers, Weinberger, Schwartz, and Davidson (1979) noted the high correlations that have been found between instruments such as the Taylor Manifest Anxiety Scale, the Byrne Repression-Sensitization Scale (Byrne, 1964), and the first factor of the MMPI, and they suggested that all of these instruments are assessing a similar construct. However, in an argument that parallels that of Linden, these investigators hypothesized that among those individuals who score low on these measures, there are actually two heterogeneous groups: one group composed of

subjects underreport psychological symptoms.
individuals who are actually calm, well-adjusted, and self-satisfied, and a second group made up of individuals who deny distress, but demonstrate physiological and other behavioral responses that are indicative of distress. The latter group was referred to as "repressors." (Weinberger et al. retained the use of the term repressor out of deference to convention in the literature. They pointed out, however, that "the extent to which this defensive style is characterized by the use of repression relative to other defenses such as denial, negation, and suppression is not currently known." [pg. 370])

In a test of their hypothesis, Weinberger and his colleagues assigned subjects to experimental groups on the basis of their scores on the Taylor Manifest Anxiety Scale (TMAS-S; Bendig Short Form, Bendig, 1956) and the Marlowe-Crowne Social Desirability Scale (MCSD; Crowne & Marlowe, 1964). Groups were designated as repressor (low TMAS-S, high MCSD), "low anxious" (low TMAS-S, low MCSD), and "high anxious" (high TMAS-S, low MCSD-C). (The terms high anxious and low anxious were used in spite of the fact that the TMAS-S was presumed to measure a broad psychological construct, rather than anxiety per se.) The researchers then conducted psychophysiological assessments of each subject while completing a word phrase association task, the content of which was designed to be neutral, sexual, or aggressive in nature. Dependent variables included heart
rate, spontaneous skin resistance, frontalis EMG, reaction
time to phrases, extent of avoidance of phrase content, and
verbal disturbance scores.

Results suggested that the repressor group was more
distressed than was the low anxious group, and at least as
distressed as the high anxious group, in spite of the fact
that the repressor group scored significantly lower on the
TMAS-S than did either of the other groups. The repressor
group demonstrated significantly more spontaneous skin
resistance responses than did the low anxious group, and
significantly more frontalis region EMG than either the low
anxious or the high anxious group. The high anxious group
demonstrated an intermediate level of SSRR, but was more
similar to the low anxious group than to the repressor
group. Group differences on heart rate approached
significance (p < .06), with the repressor group and the
high anxious group having higher HR than the low anxious
group.

Similar results were found on the verbal indices, where
repressors demonstrated significantly longer overall
reaction times to phrases than did the low anxious group.
Repressors also gave significantly higher verbal disturbance
scores and demonstrated more content avoidance than did
either the low anxious or the high anxious groups.

Following the phrase association task, subjects were
asked to rate their awareness of 16 bodily reactions during
the task. In spite of the differences in psychophysiological responses, there were no group differences in awareness of bodily reactions. In discussing the incongruity in the repressor group between self-reports of low anxiousness and physiological and behavioral indices of distress, Weinberger and his colleagues noted the important implications that such findings may have for models of stress-related illness. They also suggested that a repressive response style to distress may lead to a proneness to physical disease, at the same time that individuals with such a style may avoid seeking medical help.

Weinberger and his colleagues used an incomplete design in their study, as they did not include a group of subjects who scored high on both the TMAS-S and the MCSD (a "defensive high anxious" group). This was corrected in a study by Asendorpf and Scherer (1983). In that study, subjects were assigned to groups according to the criteria used by Weinberger and his colleagues, and psychophysiological recordings were conducted while the subjects performed a free association task to neutral, aggressive, and sexual content phrases, and several control tasks. Dependent measures included heart rate, a measure of subjective anxiety experienced during the task, and degree of anxiety expressed facially, as scored by independent raters of videotapes. During the phrase association task,
both the repressor group and the high anxious group showed significantly greater heart rate increases than did the low anxious group, with no significant differences in HR between the repressor group and the high anxious group. The defensive high anxious group displayed an intermediate level of heart rate increase. The repressor group was also rated as displaying significantly more facial anxiety than was the low anxious group. The high anxious and defensive high anxious groups were rated as demonstrating intermediate levels of facial anxiety, with no significant differences between them or the repressor or low anxious groups. In summary, the investigators noted that within the repressor group there was a distinct incongruence between self-report of subjective anxiety, and measures of HR and facial anxiety.

Gudjonsson (1981) conducted a similar study, but predicted anxiety and defensiveness scores from congruence or incongruence between physiological indices and self-reports of distress. In this study, electrodermal reactivity was assessed as each subject responded orally to emotionally loaded questions. Following the task, subjects were asked to indicate on a visual analogue scale how disturbing they found the questions to be. Subjects were then classified on the basis on congruence or incongruence of subjective distress and assessed electrodermal reactivity. Those subjects displaying high electrodermal
reactivity but low subjective distress were labelled the repressor group, those reporting high subjective distress but demonstrating low electrodermal responses were labelled "sensitizers," and those who reported subjective distress that was congruent with electrodermal reactivity were labelled "congruents." Subjects were then administered the Marlowe-Crowne Social Desirability Scale and the Eysenck Personality Inventory (EPI; Eysenck & Eysenck, 1964). The EPI neuroticism subscale (which correlates .78 with the Taylor Manifest Anxiety Scale) was used to assess trait anxiety.

As predicted, Gudjonsson found significant differences between the three groups in both trait anxiety and defensiveness. The repressor group had the highest MCSD scores and the lowest EPI-N scores, while the sensitizer group had the lowest MCSD scores and the highest EPI-N scores.

Other investigators have compared similarly grouped subjects on verbal responsiveness and on the ability to recall affective memories. Schill, Emanuel, Pedersen, Schneider, and Wachowiak (1970) found that repressor subjects were significantly less verbally responsive to double-entendre phrases than were high anxious or low anxious subjects. Defensive high anxious subjects responded at an intermediate level. Davis and Schwartz (1987) and Davis (1987) found that repressor subjects were able to
recall fewer affective memories from childhood than did either low anxious or high anxious subjects, while defensive high anxious subjects were able to recall an intermediate number of affective memories. This was not found to be related to a general deficit in memory recall.

As noted previously, there are few, if any, studies that have looked specifically at the impact of response bias on CHD research, or at the potential relation between response bias and CHD per se. Nevertheless, there has been some research on CHD from the psychodynamic perspective, whose results could be re-interpreted within the context of response bias, and several non-dynamically oriented investigators have noted incidental findings which might also be explained through a response bias interpretation. These findings will be reviewed briefly.

Rime and Bonami (1979) conducted a study based upon the psychodynamic theory of Arlow, which suggests that cardiac patients behave in a responsible, adult-like manner, while desiring to behave in a more passive, immature manner, with resulting constant inner stress. These researchers found no significant differences between MI subjects and healthy controls on responses to questions about overt behaviors and attitudes, except for a set of questions reflecting lowered energy in the MI subjects. In contrast, MI subjects scored significantly higher on questions designed to measure attitudes and preferences that were presumed to be under
less control of social norms, and which suggested passivity and dependence. The researchers interpret these results as suggesting that cardiac subjects do not differ from non-cardiac subjects on overt, easily measured characteristics, but that they do differ on less obvious characteristics, which are in contrast to the overt characteristics. The authors speculate that these individuals need to disguise these less obvious traits, as they are socially less desirable.

In another psychodynamically oriented study, Cleveland and Johnson (1962) compared projective test results of MI patients with those of pre-surgery patients. The MI subjects gave significantly more responses that suggested dysphoria, hopelessness, and depression than did the control group. In contrast, their scores on a test of self-concept were very close to the test norms mean, suggesting that they had relatively high self-esteem. Interestingly, those items endorsed by at least two thirds of the coronary subjects suggested that the subjects were independent and success-oriented, but that they were very concerned about behaving in a socially acceptable manner and about conforming to conventional norms.

In the Wardwell et al. (1963) study previously reviewed, cardiac subjects did not differ significantly from seriously ill, non-cardiac subjects on scores on the Taylor Manifest Anxiety Scale, or on a measure of self-esteem.
However, cardiac subjects reported more often that they worried frequently and could not relax, that they became anxious when they experienced anger, and that they frequently "blew up" when angered. These subjects were also significantly more likely to be rated by the interviewer as "does not know about himself and his world." These researchers suggested that the cardiac subjects appeared to attempt to "maintain face and a favorable conscious self-image in spite of underlying anxiety and insecurity about themselves" (p. 162).

Siltanen et al. (1975) conducted a discriminant analysis on data from three subgroups of a longitudinal study of 1326 policemen in Helsinki, Finland. The subgroups were healthy men (Group A), men with ECG signs of CHD, but without symptoms (Group B), and men with ECG signs of CHD, plus angina and/or history of chest pain of 30 minutes duration or longer, with or without verification of MI (Group C). Results indicated that subjects in Group B (symptom-free, in spite of ECG evidence of CHD) were inhibited in their expressions, controlled, and tended to be submissive, compared to subjects in Groups A (healthy) and C (ECG evidence of CHD, plus symptoms). Based upon these results, the authors suggested that the inhibition of expression in Group B subjects might be related to difficulty in recognizing and expressing symptoms of CHD that they might be experiencing.
In the previously reviewed longitudinal study of hypercholesteremic subjects by Thomas et al. (1975) the combination of high cholesterol level and depression was found to be a better predictor of coronary heart disease than either of the variables alone. However, while 6 of the 10 cardiac subjects scored within the top quartile of the depression scale, 4 did not endorse any of the depression items. These 4 subjects did report that they were usually tired upon awakening (a common symptom of depression), and one was later hospitalized for depression. The authors noted that all 4 of these subjects were World War II veterans, and speculated whether the failure to endorse depression items may have been related to their military training and experience with stressful situations, or to chronic depression such that depressed reactions to stress were not different from their normal state.

In the study by Friedman and Booth-Kewley (1987a), the authors investigated the differences in predicting CHD between the SI and the JAS. The investigators found that better discrimination between cardiac and non-cardiac subjects was achieved when JAS scores were combined with scores on a test of emotional expressiveness (Affective Communication Test; ACT; Friedman, Prince, Riggio, & DiMatteo, 1980), while the addition of ACT scores did not improve the ability of the SI to discriminate between the groups, suggesting that the SI includes this type of
information. Specifically, subjects who appeared reserved and quiet, but reported exhibiting high levels of positive nonverbal expressiveness (low JAS, high ACT), and subjects who appeared hurried and spoke quickly, but reported demonstrating low level of positive nonverbal expressiveness (high JAS, low ACT) were significantly more likely to have CHD than subjects in the other two groups. In addition, the low JAS, high ACT subjects were found to be the most depressed and the most anxious of the four subject groups.

Several investigators have noted the relation between CHD, hostility, and the non-expression of anger. Dembroski, MacDougall, Williams, Haney, and Blumenthal (1985) found that the interaction of potential for hostility and the tendency to not express anger was significantly correlated with the extent of cardiac stenosis. This finding was independently replicated by MacDougall, Dembroski,Dimsdale, and Hackett (1985). Similarly, in an eight year report on the Framingham study, Haynes et al. (1980) found that low scores on expressing anger outwardly were related to incidence of heart disease in white collar men.

In summary, previous research in the area of CHD and psychological factors has not considered the possible impact of response bias on the results of such investigations, even though such bias has been demonstrated within other areas of psychology, and incidental findings in CHD research would suggest such an effect. If, as suggested by Linden (1988),
some CHD subjects underreport subjective distress, the relation that is found between CHD and psychological factors will be diminished to an unknown extent. In addition, such underreporting may itself be related to CHD. Research within the field of personality and social psychology provides a methodology for investigating this issue, and suggests some implications for CHD research.

Summary

Since the publication of Friedman and Rosenman's book in 1959, much of the research on psychological factors and CHD has centered on the examination of the relation between the Type A behavior pattern (TABP) and CHD. While several early longitudinal studies demonstrated a relation between TABP and CHD (e.g., DeBacker et al., 1983; French-Belgian Cooperative Group, 1982; Haynes et al., 1980; Rosenman et al., 1975), the results of more recent studies have brought that conclusion into question, and TABP is no longer considered to be a risk factor for CHD (Haynes & Matthews, 1988). Much of the subsequent research has been directed towards discovering the "toxic" components of the TABP, and recent research has focused on the role of anger and hostility in CHD (e.g., Matthews et al., 1977).

The results of a meta-analytic study by Friedman and Booth-Kewley (1987b) suggest that depression and anxiety are also related to CHD, though there have been few studies that included measures of either. Friedman and Booth-Kewley
(1987a) also found that depression and anxiety are not independent in their relation to CHD, as has been suggested by the results of other researchers (e.g., Bianchi et al., 1978; Thiel et al., 1973; Thomas & Greenstreet, 1973). They suggest therefore that future research investigate the relation between CHD and a broader psychological factor, rather than more specific factors such as depression or anxiety per se. This suggestion is consistent with the findings of high intercorrelations between many self-report questionnaires that are assumed to assess either anxiety or depression (e.g., Dobson, 1985a, 1985b; Gotlib, 1984; Meites et al., 1980). Several investigators have described similar broad psychological constructs, such as general maladjustment (e.g., Jessor & Hammond, 1957), and emotionality (Eysenck & Eysenck, 1975). Watson and Clark (1984) have used the term negative affectivity, and their research suggests that one of the best measures of this construct is the Taylor Manifest Anxiety Scale (Taylor, 1953).

Linden (1988) agrees that CHD research should address a broad band psychological factor, but argues that future research should also consider the possible effects of response bias on the data obtained. This issue has not been directly addressed in CHD research. However, the findings from some psychodynamically oriented CHD studies lend themselves to a response bias interpretation, and the same
factor might also explain some incidental findings in other CHD studies. A series of studies based upon similar arguments has been conducted in the area of social psychology (e.g., Asendorpf & Scherer, 1983; Davis, 1987; Davis & Schwartz, 1987; Weinberger et al., 1979). The results of these studies indicate that there are some individuals who report low levels of psychological distress on self-report questionnaires, but who exhibit distress-related physiological and verbal behaviors at a level as high or higher than individuals who acknowledge psychological distress. Several of these studies successfully identified these groups through administration of the Taylor Manifest Anxiety Scale and the Marlowe-Crowne Social Desirability Scale. This methodology was incorporated in the present investigation.

Purpose of this Study

The purpose of this study was to investigate the relation between CHD and a general psychological factor, referred to as negative affectivity (NA; Watson and Clark, 1984), and to investigate the role of response bias in predicting CHD from NA. Negative affectivity was assessed with the Taylor Manifest Anxiety Scale - Bendig Short Form (TMAS-S), and response bias was assessed with the Marlowe-Crowne Social Desirability Scale - Short Form (MCSD-C). These choices parallel the methods developed by Weinberger et al. (1979). CHD was indicated by the presence of
36

coronary stenosis, as measured by coronary arteriography. The relations between negative affectivity and response bias, and self-reports of chest pain and impairment in daily functioning secondary to chest pain were also assessed. Six related questions were addressed. (In this study, the terms "prediction" and "predictor variable" refer only to the statistical procedure of entering a set of variables, called predictor variables, into a regression equation to determine the extent to which that set of variables is related to a single "criterion" variable. Given the design of this study, it cannot be assumed that the factors measured by the predictor variables caused the phenomena measured by the criterion variable.)

1. Is there a significant positive relation between scores on the TMAS-S and severity of cardiovascular stenosis, self-reports of chest pain, and/or self-reports of impairment in daily functioning secondary to chest pain? Based upon previous research findings, it was expected that the relation between the TMAS-S scores and degree of stenosis would be modest, while the relation between TMAS-S scores and self-reports of pain and impairment in daily functioning would be of greater magnitude.

2. Does the combination of TMAS-S scores, MCSD-C scores, and the TMAS-S by MCSD-C interaction improve the prediction of degree of cardiovascular stenosis, self-reports of chest pain, and/or self-reports of impairment in daily functioning
secondary to chest pain? While TMAS-S scores alone were expected to account for only a modest amount of variability in degree of stenosis, it was hypothesized that the addition of MCSD-C scores and the interaction of TMAS-S by MCSD-C scores would improve the prediction of degree of cardiovascular stenosis, while having little effect on the prediction of self-reports of chest pain or self-reports of impairment in daily functioning secondary to chest pain.

3. Do scores on the TMAS-S and MCSD-C add to the prediction of heart disease over and above demographic and medical factors of age, positive family history of heart disease, diagnosis of hypertension, diagnosis of diabetes, or current cigarette use? It was predicted that the addition of TMAS-S and MCSD-C scores and the TMAS-S by MCSD-C interaction would increase the amount of variability accounted for in degree of stenosis, as NA was hypothesized to be significantly related to CHD, and the combination of TMAS-S and MCSD-C scores was hypothesized to be a better measure of NA than TMAS-S scores alone.

4. Of those factors contributing to the prediction of degree of stenosis, which are the best predictors? As there had been no prior investigation of this issue, no hypothesis was proposed.

5. Are there differences in degree of stenosis, level of self-reported pain, and degree of self-reported functional impairment secondary to pain, between groups that are
classified by high versus low scores on the TMAS-S and MCSD-C? As TMAS-S scores were hypothesized to be correlated with self-reports of pain and impairment in daily living secondary to pain, it was predicted that subjects with low TMAS-S scores would report less pain or impairment than would groups of subjects with high TMAS-S scores. It was also predicted that subjects with low TMAS-S scores and high MCSD-C scores (repressors) would demonstrate greater levels of stenosis than would the other three groups, as those subjects would not be expected to seek out medical help as quickly as would the other three groups.

6. Are there significant differences in TMAS-S scores and MCSD-C scores between individuals whose self-reports of pain are congruent with degree of stenosis, and individuals whose self-reports of pain are incongruent with degree of stenosis? Based upon the results of Gudjonsson (1981), it was predicted that individuals whose self reports were congruent with degree of stenosis would have low MCSD-C scores. Those who reported relatively little pain, but demonstrated a relatively high degree of stenosis were predicted to have low TMAS-S scores and high MCSD-C scores. Those who reported relatively high levels of pain, while demonstrating relatively low degrees of stenosis, were predicted to have high TMAS-S scores, and high MCSD-C scores.
Method

Design

Independent Variables

This study included 2 independent (predictor) variables of primary interest. These were scores on the Taylor Manifest Anxiety Scale - Short Form (TMAS-S; Bendig, 1956), as a measure of negative affectivity, and scores on the Marlowe-Crowne Social Desirability Scale - Short Form C (MCSD-C; Reynolds, 1982), as a measure of response bias. Other predictor variables included in the multiple regression analyses were age, family history of CHD, smoking history, diagnosis of hypertension, and diagnosis of diabetes.

Dependent Variables

Dependent variables consisted of two measures of degree of cardiac stenosis, a measure of chest pain, and a measure of impairment in daily activities secondary to chest pain.

Subjects

Subjects were drawn from those patients of the Cardiology Clinic at the Jackson, MS, VA Medical Center, who had had cardioarteriography studies within the previous 6 months. Patients who were older than 65 years of age, or who had medical or other evidence of active psychosis, dementia, or organic brain syndrome were excluded from the study, as were those with serious transportation problems,
Table 1

Sample Demographic and Medical Characteristics (N=78)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Yes</th>
<th>No</th>
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<tbody>
<tr>
<td>Married</td>
<td>61 (78%)</td>
<td>17 (22%)</td>
</tr>
<tr>
<td>Diagnosis of hypertension</td>
<td>30 (38%)</td>
<td>48 (62%)</td>
</tr>
<tr>
<td>Diagnosis of diabetes</td>
<td>9 (12%)</td>
<td>69 (88%)</td>
</tr>
<tr>
<td>Family history of heart disease</td>
<td>51 (65%)</td>
<td>27 (35%)</td>
</tr>
<tr>
<td>Smoker</td>
<td>27 (35%)</td>
<td>51 (65%)</td>
</tr>
</tbody>
</table>
and those who were unable to read at a level sufficient to complete necessary questionnaires. The sample included 78 men with an average age of 56.8 years (range of 37 to 65 years). The average education level was 11.2 years of school, with a range of 4 to 16 years. Seventy of the subjects were white (93%) and the remainder were black (7%). Other pertinent demographic and medical characteristics of the subjects are presented in Tables 1 and F-1.

**Measures**

**Taylor Manifest Anxiety Scale - Short Form (TMAS-S).** (See Appendix A.)

The Taylor Manifest Anxiety Scale (Taylor, 1953) is a self-report scale consisting of 50 true-false questions that were drawn from the MMPI. It was originally designed for selecting subjects in human motivation studies, but has since been used in both research and clinical settings. Test-retest reliability reported by the author (Taylor, 1953) is .88 over 4 weeks, and internal consistency has been reported to be approximately .82 (Bendig, 1956; Watson & Clark, 1984; Tanaka-Matsumi & Kameoka, 1986). An estimate of convergent validity is provided in the study by Tanaka-Matsumi and Kameoka (1986), who reported intercorrelation of .79 between the TMAS-S and the Spielberger Trait Anxiety Scale (Spielberger, Gorsuch, & Lushene, 1970) and .72 with the Zung Self-Rating Anxiety Scale (Zung, 1971). On the other hand, Meites et al. (1980) found that the TMAS
correlates .79 with the Eysenck Personality Inventory - Neuroticism Scale, and .64 with the Beck Depression Scale, suggesting that it is a measure of a more general construct. As reviewed earlier, Watson and Clark (1984) consider the TMAS to be one of the best instruments for assessing negative affectivity (NA). In addition to the above noted relations, they report that it correlates .85 with the first factor of the MMPI and .88 with the Byrne Repression-Sensitization Scale (Byrne, 1961).

The short form of the TMAS (TMAS-S; Bendig, 1956) is comprised of the 20 most consistently valid items from the original scale. Bendig reports that the intercorrelation between the TMAS-S and the TMAS is .93, while the internal consistency of the TMAS-S is .76. Weinberger et al. (1979), Davis and Schwartz (1987), and Davis (1987) used the short form of the TMAS, in conjunction with the Marlowe-Crowne, to differentiate high anxious, low anxious, and repressor groups in their studies.

Marlowe-Crowne Social Desirability Scale - Short Form C (MCSD-C). (See Appendix B.)

The Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1960) is a 33 item true-false questionnaire that was designed to assess the tendency of subjects to respond in a culturally appropriate manner, without implication of maladjustment or psychopathology. The items refer to behaviors that are judged to be culturally sanctioned, but
unlikely to occur. The authors report a Kuder-Richardson estimate of internal consistency of .88, and report test-retest reliability of .89. The Short Form - C was developed by Reynolds (1982) and is comprised of 13 items from the original 33. Reynolds reports intercorrelation with the full scale MCSD-C of .93, and Kuder-Richardson estimate of internal reliability of .76. Zook and Sipps (1985) report similar results with the Form C, and also report test-retest reliability of .74 over 4 weeks.

Other Assessment Data

Medical records. Medical records were reviewed for diagnoses of hypertension or diabetes, for family history of heart disease, and for history of smoking. Subjects who had not smoked for the 60 days preceding the angiographic studies were considered to be non-smokers.

The extent of cardiac stenosis was determined through review of angiography reports, and two measures of stenosis were computed. The first of these consisted of the number of major cardiac vessels with greater than 50% occlusion, a measure that has been widely used in previous research (e.g., Bass & Wade, 1984; Costa, Zonderman, et al., 1985; Elias et al., 1982; Frasure-Smith, 1987; Katon et al., 1988; MacDougall et al., 1985; Schocken, Greene, Worden, Harrison, & Spielberger, 1987; Zyzanski et al., 1976). The major vessels are considered to be the left main, the right coronary, the left anterior descending, and the circumflex
coronary arteries. Fifty per cent reduction in arterial diameter is often used as the criterion for diagnosis of significant atherosclerosis, based upon the fact that in normal subjects, who demonstrate no reduction in coronary blood flow while at rest, blood flow reduction occurs when the cross-sectional area of a coronary artery is reduced by 70%, in response to stimuli such as exercise or emotion. As 50% reduction in diameter yields 75% reduction in area, this is considered to be a logical criterion for diagnosis of disease (Pearson, 1984).

As Jenkins, Stanton, Klein, Savageau, and Harkin (1983) have suggested that the previously described measure of stenosis, though widely used, may not be sufficiently sensitive, a second measure of stenosis was calculated. This measure was based upon the method developed by Jenkins and his colleagues (1983). Arteries were assigned a score on the basis of degree of occlusion, with 4 points for 100% occlusion, 3 points for 75-99% occlusion, 2 points for 50-74% occlusion, 1 point for any occlusion less than 50%, and 0 points for no occlusion. Scores were then totaled for the four main arteries.

**Chest Pain Questions.** (See Appendix C.) Subjects completed 2 questions concerning frequency and intensity of chest pain, which was defined as pain, heaviness, tightness, or discomfort. The questions were 9-point likert-type questions, and were based upon questions developed by Rose,
McCartney, and Reid (1977). Anchors concerning the frequency of pain ranged from never to more than 5 times a day, and anchors for the intensity of pain ranged from barely noticeable to most severe pain possible. The responses were scored on a scale of 1 to 9, and the results were multiplied to yield a pain intensity x frequency index.

**Interference in Daily Living Questions.** (See Appendix D.) Subjects completed two 9-point likert-type questions from the Rose questionnaire (Rose et al., 1977), the first of which assessed the frequency with which chest pain interferes with activities, and the second of which assessed frequency with which the subject avoids activities because the activities might bring on chest pain. The anchors for these questions ranged from less than once a year to more than once a day. The responses were scored on a scale of 1 to 9 and totaled to provide one measure of interference in daily living.

**Procedure**

Data were collected in the context of a larger study on behavioral intervention for chest pain. All patients who met inclusion and exclusion criteria (as noted above) and who had other scheduled appointments were asked to attend the Behavioral Cardiology Clinic on the day of their other appointment.

Teams of two psychology interns and/or master's level
research associates conducted the group meetings of 12-20 subjects. The subjects were informed that the study was being conducted by the Behavioral Cardiology Service in conjunction with the Cardiology Department, and that the data were being collected to further understanding of chest pain, and to improve development of treatment programs for chest pain. As part of the larger study, half of the subjects were asked to participate in a three-session behavioral intervention program. Informed consent was obtained from all subjects (See Appendix E.)
Results

Three main predictions were made in this study. These were: (1) that scores on the Taylor Manifest Anxiety Scale Short Form (TMAS-S) would be more highly correlated with self reports of pain and interference in daily living than with extent of stenosis; (2) that the combination of Marlowe-Crowne Social Desirability Scale - C (MCSD-C) scores, TMAS-S scores, and the TMAS-S by MCSD-C interaction would increase the amount of variance accounted for in degree of stenosis, while having little effect on the prediction of pain and interference levels; and (3) that the combination of TMAS-S and MCSD-C scores and the TMAS-S by MCSD-C interaction would improve the prediction of level of stenosis, over and above demographic and medical factors. It was also hypothesized that subjects with low TMAS-S and high MCSD-C scores would report less pain and impairment in daily living, while demonstrating higher levels of stenosis, than would groups of subjects with other combinations of TMAS-S and MCSD-C scores. Finally, it was hypothesized that if subjects were grouped by degree of stenosis and self-report of pain, that those subjects reporting relatively little pain, but demonstrating relatively high degrees of stenosis would have low TMAS-S scores and high MCSD-C scores. Those who reported relatively high levels of pain, while demonstrating relatively low levels of stenosis, were
predicted to have high TMAS-S scores.

A correlation matrix was computed to test the hypothesis that the relation between TMAS-S scores and the two measures of stenosis is modest, while the relations between TMAS-S scores and self-reported pain and impairment in daily functioning are of greater magnitude. (See Table F-2.) The hypothesis was not supported, as none of the correlations were significant. (Table F-3 presents the correlations between the other predictor variables and the criterion variables.)

A series of hierarchal multiple regression analyses were conducted to test the hypothesis that the addition of MCSD-C scores and the TMAS-S by MCSD-C interaction to TMAS-S scores would increase the amount of variance accounted for in degree of stenosis, while having little effect on the prediction of level of self-reported pain or interference in daily living. TMAS-S scores were entered on the first step of these analyses, followed by MCSD-C scores on the second step, and the interaction of TMAS-S by MCSD-C on the third step. (Summaries of the results of these analyses are presented in Tables F-4 through F-7.)

The results of these analyses failed to support the hypothesis, as the combination of TMAS-S and MCSD-C scores and their interaction did not improve the prediction of either stenosis measure. In contrast, the combination of MCSD-C scores and TMAS-S scores in the second step of the
multiple regression yielded a significant equation in the prediction of pain index scores ($R^2 = .0937; p < .028$). The addition of the TMAS-S x MCSD-C interaction increased the amount of variance accounted for ($R^2$ change = .094, $p < .005$), yielding a significant overall model $R^2 = .1873$ ($p < .002$). As predicted, the combination of TMAS-S scores, MCSD-C scores, and their interaction did not substantially increase the amount of variance accounted for in interference in daily living.

A second series of multiple regression analyses were conducted to test the hypothesis that the combination of TMAS-S scores, MCSD-C scores, and the TMAS-S by MCSD-C interaction would add to the prediction of extent of stenosis, over and above demographic and medical factors. In these analyses, age, family history of CHD, smoking, diagnosis of hypertension, and diagnosis of diabetes were entered simultaneously into a regression analysis for each of the stenosis measures. TMAS-S scores and MCSD-C scores were entered hierarchically following the demographic and medical variables (on the sixth and seventh steps of the analysis, respectively), followed by the TMAS-S by MCSD-C interaction term. (Summaries of these analyses are presented in Tables F-8 and F-9.)

These results did not support the above hypothesis. The demographic and medical factors did not significantly predict the extent of either measure of stenosis, and the
addition of TMAS-S scores, MCSD-C scores, and the TMAS-S by MCSD-C interaction did not improve the amount of variance accounted for. In the regression to the first measure of stenosis (the number of major arteries with greater than 50% occlusion), none of the factors had beta weights that were significant. When the second measure of stenosis was used as the dependent variable (the total of occlusion ratings from the major arteries), only family history of heart disease carried a significant beta weight (p < .04).

Methods similar to those of Davis and Schwartz (1987) were used to test the hypothesis that there are significant differences in degree of stenosis, self-report of pain, and impairment in daily living between subjects grouped by TMAS-S and MCSD-C scores. Median splits on both measures were used to define four groups of subjects. Preliminary analyses were conducted to test that the groups were equivalent on relevant demographic variables. There were no significant age differences, but the groups were found to be non-equivalent on level of education. However, no significant linear relations were found between education and the dependent measures, and therefore education was not covaried in the main analysis. Loglinear analyses were conducted to verify group equivalences on factors of marital status, race, family history of heart disease, smoking, and diagnosis of hypertension or diabetes. No significant differences were found. MANOVAS were then conducted to
determine whether the groups differed on the dependent variables of degree of stenosis, self report of pain, and/or impairment in daily living. The assumption of homogeneity of dispersion matrices was not rejected ($F (30, 10806) = 1.080, p = .349$). The overall MANOVA was not significant. One univariate $F$ test was significant (main effect for TMAS-S scores on pain index; $p < .025$). However, as the overall MANOVA was not significant, this result was considered to be attributable to random chance, and was not interpreted. (See Table F-10 for a summary of this analysis.)

To test the hypothesis that subjects who differ on congruency or incongruency of self-report of pain and degree of stenosis also differ on TMAS-S scores and MCSD-C scores, subjects were grouped by median split into high and low levels of self-report of pain, and high and low levels of stenosis, based on the second stenosis measure. As in the previous analysis, a MANOVA was conducted to determine whether there were group differences on age and education, and loglinear methods were applied to determine whether there were significant group differences in marital status, race, family history of heart disease, smoking, or diagnosis of hypertension or diabetes. No significant group differences on these factors were revealed. MANOVAs were then conducted to determine whether the groups differed on TMAS-S and MCSD-C scores. The assumption of homogeneity of dispersion matrices was not rejected ($F (9, 56685) = 1.409, p$
=.178). The overall MANOVA was not significant, nor were any univariate F tests significant. (See Table F-11 for a summary of this analysis.)
Discussion

Results of this study failed to support the hypothesis that negative affectivity is related to coronary stenosis, or that a tendency on the part of some subjects to underreport negative affectivity may obscure such a relation. TMAS-S scores alone were not found to be significantly related to either of two measures of stenosis, nor did multiple regressions including only TMAS-S and MCSD-C scores and their interaction, or TMAS-S and MCSD-C scores and their interaction in combination with demographic and medical factors, account for significant amounts of variance in extent of stenosis. Similarly, no significant differences in stenosis were found between subjects who were grouped by TMAS-S and MCSD-C scores, nor were differences in TMAS-S and MCSD-C scores found between subjects who were grouped by extent of stenosis and level of self-report of pain.

There are several possible explanations for the failure to find significant results. The most obvious is the possibility that there may not be a relation between negative affectivity and CHD. Certainly, the results of previous studies have been mixed, and several researchers (e.g. Siegel, 1985) have suggested that future research be directed towards areas other than personality constructs.

On the other hand, there are several factors inherent
in cross-sectional studies in general, and those investigating medical problems in particular, that may have influenced the results. Pearson (1984) has discussed some potential biases that may be particularly problematic in cross-sectional studies of arteriographically defined coronary heart disease, and any of these sources of bias may contribute to spurious non-associations, as well as to spurious associations. The most serious methodological problem in this type of study is that of very restricted subject sample, with resultant decreased variance of dependent variables, and hence diminished chances of finding significant associations. As noted by Pearson, the choice of individuals who undergo arteriography is not random, as the procedure is invasive, expensive, and involves some degree of risk, and hence only individuals with suspected disease are typically studied. Furthermore, those people who do undergo arteriography are not completely representative of all individuals with coronary artery disease. For example, individuals with mild angina or silent MIs are less likely to seek medical attention for cardiac problems, and those individuals in whom coronary artery disease is not diagnosed prior to sudden cardiac death or fatal initial myocardial infarct will obviously not be included in such studies (prevalence-incidence bias). The later group may be of particular relevance in studies of negative affectivity, given that Lebovits et al. (1967) and
Bruhn et al. (1969) found significant differences in initial measures of depression between subjects who did not survive subsequent MIs and those who did survive. Admission rate bias may also affect research outcome, as many studies include a prevalence of married individuals, while vital statistics and prospective studies suggest that non-married individuals are at greater risk for coronary heart disease (e.g., Cramer, Paulin, & Werko, 1966). In the current study, 78% of the sample was married, suggesting that the sample was not adequately representative of non-married individuals with CHD. Other potential sources of bias include differential rates of referral for arteriography for individuals who have a positive history for known risk factors, such as smoking (diagnostic suspicion bias) or for individuals who have certain characteristics, such as those associated with type A behavior pattern, due to physician beliefs about symptoms associated with CHD (detection signal bias). Finally, there is the possibility of nonresponder bias, which exists not only at the level of agreeing to participate in a psychological study on angiography patients, but more basically at the level of deciding whether or not to undergo angiography. Any of these factors may decrease the variance in extent of cardiac stenosis in the sample. The possibility that the lack of significant results in the current study may be due to methodological issues is supported by the fact that family history of heart
disease was the only demographic or medical factor to have a significant positive beta weight in either regression to level of stenosis. As reviewed by Pearson (1984), previous cross-sectional studies have indicated that age and diabetes are each positively related to prevalence and extent of stenosis, and cigarette smoking and family history of heart disease are positively related to presence of heart disease. The lack of positive association in this study between CHD and three of these factors calls into question the adequacy of the sample in this study.

The research findings on the relation between hypertension and heart disease points out another potential bias in cross-sectional studies. As noted by Pearson (1984), by the time of arteriography, the evidence for some factors that are associated with the development of CHD may no longer be present. For example, although most longitudinal and autopsy studies have documented a positive association between hypertension and coronary heart disease, negative associations between the two have been found in 10 out of 12 cross-sectional studies. Pearson states that during an MI, the myocardium can be damaged to the extent that a previously hypertensive individual can become clinically normotensive or even hypotensive, thereby biasing any study of the concurrent presence of hypertension and CHD. He also hypothesizes that evidence of hypertension is frequently obscured at the time of arteriography by the
fact that many cardiology patients take medications for angina that also reduces blood pressure. A similar factor may have affected the current study, in that it was not possible to control the types of medications taken by the subjects. It is possible that these medications may have had a significant effect on the experience of negative affectivity.

Another problem that may have affected the results of this study is the fact that after having been diagnosed with CHD, some subjects may have modified their behaviors and reactions to emotional situations in an attempt to decrease their chances of further heart difficulties. Examples of such behavior changes might be learning to avoid or walk away from emotional situations, learning to use cognitive coping skills for dealing with emotional situations, learning specific relaxation procedures, or increasing exercise. To the extent that these changes affected the experience of negative affectivity, they might also affect the results of this study. The current study did not assess whether subjects had attempted to change their behaviors in such manners.

The method used to rate extent of stenosis may have further contributed to the lack of positive findings. Jenkins et al. (1983) have discussed the problems with measures of stenosis, and have pointed out that the measures now in use may not be sufficiently sensitive to determine
different levels of stenosis. While the present study attempted to use a more sensitive measure, there are also components of stenosis that were not included. For example, the systems of scoring currently in use do not take into consideration the location of lesions within an artery, the fact that some arteries have more than one lesion, the fact that some patients develop collateral vessels that may also develop lesions, or the fact that lesions may be either discrete or tubular in nature. Scoring systems for incorporating such information have not yet been developed.

This study did yield some results that were unexpected. While TMAS-S scores were not significantly correlated with self-report of pain, the combination of TMAS-S and MCSD-C scores in the regression analysis accounted for a significant amount of variance in the pain index, with the greatest amount of variance being carried by the TMAS-S by MCSD-C interaction. Costa and his colleagues (Costa, 1986; Costa & McCrae, 1980, 1985, 1987; Costa et al., 1985) have described the construct of "neuroticism," which Costa believes is equivalent to negative affectivity. However, instead of viewing neuroticism as a cause of disease, Costa suggests that people who score high on measures of neuroticism are more concerned about health issues, seek medical attention earlier, and are more willing to complain of pain and other symptoms than are people who score lower on measures of neuroticism. In those situations where
medical diagnosis is based largely on patient report of symptoms (such as is often the case with angina), Costa predicts that there will be a larger proportion of individuals who are high in neuroticism, as those people would be more likely to complain of somatic problems. On the other hand, when diagnosis involves expensive or invasive diagnostic procedures (such as angiography), Costa predicts that people who are tested are either very ill, or complain a lot (i.e., would score very highly on measures of neuroticism). In these cases, since some of the high neuroticism individuals will not be truly ill, and will subsequently be found to be free of "hard" signs of disease, the degree of impairment might be found to be inversely related to neuroticism. The results of this study did not clearly support Costa's hypothesis, as there were no significant differences in level of stenosis between subjects with high TMAS-S scores, and those with low TMAS-S scores. There was also not a significant association between TMAS-S scores and self-report of pain or interference in daily living secondary to pain. However, the combination of TMAS-S scores and MCSD-C scores in a multiple regression was significantly related to self-report of pain. The exact nature of this relation is beyond the scope of this study. However, the results suggest that social desirability and the interaction between negative affectivity and social desirability may be an important
consideration in explaining self-reports of pain, independent of medical factors.

Until the development of diagnostic techniques that are less invasive than cardiac arteriography, but equally objective, it is likely that researchers using cross-sectional methods in the study of CHD will have difficulty obtaining adequate subject samples and control groups. Some of the potential biases introduced by this limitation could be minimized by determining the extent to which a given sample is representative of CHD patients in general. However, other sources of potential bias, such as prevalence-incidence bias, cannot be controlled within the context of any cross-sectional study. Future studies of negative affectivity and CHD should therefore use longitudinal designs with initially health individuals, and should incorporate measures of response bias.

Research should also be conducted to develop more sensitive measures of stenosis, and to determine to what extent these measures are related to the probability of subsequent MI and death secondary to CHD. Finally, there is a need for research on the neural and hormonal mechanisms of emotion and stress, as this could provide hypotheses on the relation between negative affectivity and CHD.
Bibliography


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Appendix A

Taylor Manifest Anxiety Scale

Short Form (Bendig, 1956)
TMAS-S

For each statement please indicate whether the statement is more true, or more false, for you. Circle the T for "true" or the F for "false."

1. I believe that I am no more nervous than most others. T F
2. I work under a great deal of tension. T F
3. I cannot keep my mind on one thing. T F
4. I am more sensitive than most other people. T F
5. I frequently find myself worrying about something. T F
6. I am usually calm and not easily upset. T F
7. I feel anxiety about something or someone almost all the time. T F
8. I am happy most of the time. T F
9. I have periods of such great restlessness that I cannot sit long in a chair. T F
10. I have sometimes felt that difficulties were piling up so high that I could not overcome them. T F
11. I find it hard to keep my mind on a task or job. T F
12. I am not unusually self-conscious. T F
13. I am inclined to take things hard. T F
14. Life is a strain for me much of the time. T F
15. At times I think I am no good at all. T F
16. I am certainly lacking in self-confidence. T F
17. I certainly feel useless at times. T F
18. I am a high-strung person. T F
19. I sometimes feel that I am about to go to pieces. T F
20. I shrink from facing a crisis. T F
Appendix B

Marlowe-Crowne Social Desirability Scale

Form C (Reynolds, 1982)
Listed below are a number of statements concerning personal attitudes and traits. Read each item and decide whether the statement is true or false as it relates to you, then circle T for "True" or F for "False.

True  False

1. It is sometimes hard for me to go on with my work if I am not encouraged.  T  F
2. I sometimes feel resentful when I don't get my way.  T  F
3. On a few occasions, I have given up doing something because I thought too little of my ability.  T  F
4. There have been times when I felt like rebelling against people in authority even though I knew they were right.  T  F
5. No matter who I'm talking to, I'm always a good listener.  T  F
6. There have been occasions when I took advantage of someone.  T  F
7. I'm always willing to admit it when I make a mistake.  T  F
8. I sometimes try to get even rather than forgive and forget.  T  F
9. I am always courteous, even to people who are disagreeable.  T  F
10. I have never been irked when people expressed ideas very different from my own.  T  F
11. There have been times when I was quite jealous of the good fortune of others.  T  F
12. I am sometimes irritated by people who ask favors of me.

13. I have never deliberately said something that hurt someone's feelings.
Appendix C

Pain Questions
Pain Questions

1. Over the past 4 weeks, how often have you experienced chest pain, heaviness, tightness, or discomfort?
   
   a. Never
   b. 1-3 times a month
   c. Once a week
   d. 2-3 times a week
   e. 4-6 times a week
   f. Once a day
   g. 2-3 times a day
   h. 4-5 times a day
   i. More than 5 times a day

2. In general, when you have had chest pain or discomfort, how strong or intense has it been?
   
   a. Barely noticeable
   b. Very mild pain
   c. Mild pain
   d. Moderate pain
   e. Fairly strong pain
   f. Strong pain
   g. Very strong pain
   h. Severe pain
   i. Most severe pain
   possible
Appendix D

Questions on Interference with Daily Activities
Interference with Daily Activities Questions

1. How often has chest pain interfered with your daily activities?
   a. Less than once a year           f. Once a week
   b. Once or twice a year           g. More than once a week
   c. Less than once a month         h. Once a day
   d. Once a month                   i. More than once a day
   e. 2-3 times a month

2. How often do you avoid activities because the activity might bring on chest pain or discomfort?
   a. Less than once a year           f. Once a week
   b. Once or twice a year           g. More than once a week
   c. Less than once a month         h. Once a day
   d. Once a month                   i. More than once a day
   e. 2-3 times a month
Appendix E

Consent Forms
# Part I: Agreement to Participate in Research

**by or under the direction of the Veterans Administration**

## 1. I, [Subject's Name], voluntarily consent to participate as a subject in the investigation entitled **Self Management of Chest Pain**

**Title of Study**

2. I have signed one or more information sheets with this title to show that I have read the description including the purpose and nature of the investigation, the procedures to be used, the risks, inconveniences, side effects and benefits to be expected, as well as other matters all action open to me and my rights to withdraw from the investigation at any time. Each of these items has been explained to me by the investigator in the presence of a witness. The investigator has answered my questions concerning the investigation and I believe I understand what is intended.

3. I understand that no guarantees or assurances have been given to me since the results and risks of an investigation are not always known beforehand. I have been told that this investigation has been carefully planned, that the plan has been reviewed by knowledgeable people, and that every reasonable precaution will be taken to protect my well-being.

4. In the event I sustain physical injury as a result of participation in this investigation, if I am eligible for medical care as a veteran, all necessary and appropriate care will be provided. If I am not eligible for medical care as a veteran, humanitarian emergency care will nevertheless be provided.

5. I realize I have not released this institution from liability for negligence. Compensation may or may not be payable, in the event of physical injury arising from such research, under applicable federal laws.

6. I understand that all information obtained about me during the course of this study will be made available only to doctors who are taking care of me and to qualified investigators and their assistants where their access to this information is appropriate and authorized. They will be bound by the same requirements to maintain my privacy and anonymity as apply to all medical personnel within the Veterans Administration.

7. I further understand that, where required by law, the appropriate federal officer or agency will have access to information obtained in this study should it become necessary. Generally, I may expect the same respect for my privacy and anonymity from these agencies as is afforded by the Veterans Administration and its employees. The provisions of the Privacy Act apply to all agencies.

8. In the event that research in which I participate involves certain new drugs, information concerning my response to the drug(s) will be supplied to the sponsoring pharmaceutical house(s) that made the drug(s) available. This information will be given to them in such a way that I cannot be identified.

### Name of Volunteer

I ________________________________

Have read this consent form. All my questions have been answered, and I freely and voluntarily choose to participate. I understand that my rights and privacy will be maintained. I agree to participate as a volunteer in this program.

9. Nevertheless, I wish to limit my participation in the investigation as follows:

## Witness Information

- **Witness's Name and Address (print or type):**
- **Witness's Signature:**

## Investigator Information

- **Investigator's Name (print or type):** Thomas J. Payoe, Ph.D.
- **Investigator's Signature:**

---

**Date:**

**VA Facility:** Veterans Administration Medical Center, Jackson

**Subjects Signature:**

**Signed Information:**
- [ ] Sheet Attached
- [ ] Sheet Available

**Subjects Identification (D.O.B., place of birth, etc.):**

**Subjects I.D. No.:**

**Hand:**

---

**Agreement to Participate in Research by or under the direction of the Veterans Administration**

VA Form 10-1086

SEP 1979, which will not be used.
INFORMED CONSENT

TITLE OF STUDY: Self-Management of Chest Pain

The Cardiology and Psychology Services of the Jackson Veterans Administration Medical Center are currently involved in research exploring the most effective methods of managing chest pain. Similar methods have been found to be useful with a variety of pain conditions. This research has been designed to explore whether such an intervention may improve upon or supplement current medical practices.

If you choose to participate, you will be asked to simply fill out some forms providing information about yourself which is related to heart disease. Over the following 12 months, you will be contacted (usually by mail, or during your regularly scheduled CV Clinic appointment), and asked to fill out a brief questionnaire checking on your progress. This information will be made available to your cardiologist.

If you choose not to participate in this study, this decision will not affect your relationship with Cardiology Service, Psychology Service or the VA Medical Center in any way. In addition, if you decide to participate, you are free to withdraw at any time, for any reason.

All of the information you provide during this study will be kept strictly confidential, and will be available only to the investigators of the project unless we obtain written permission from you. No information which may identify you will be used in any reports of the results obtained from this study.

Risks

There are no known risks associated with participation in this study. As mentioned above, any personal information you reveal to us will be kept in the strictest confidence.

Consent

Participants must be at least 18 years of age. By signing below, you are indicating a willingness to participate in this study. It further indicates you have read and understood the information above, and have had the opportunity to ask any questions you have about this research project. If you wish to have a copy of this form, one will be provided for you. If any questions or concerns related to this study should arise in the future, you may contact Dr. Thomas J. Payne at the telephone number listed below.

Principal Investigator
Thomas J. Payne, Ph.D.
Director, Behavioral Cardiology Program
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VA Medical Center
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Jackson, MS 39216
(601) 364-1350

Institutional Review Board
James L. Achord, M.D.
Chairman
University of Mississippi Medical Center
2500 N. State Street
Jackson, MS 39216

_________________________  __________________________
Patient's Signature          Date

_________________________  __________________________
Witness                   Date
TITLE OF STUDY: Self-Management of Chest Pain

The Cardiology and Psychology Services of the Jackson Veterans Administration Medical Center are currently involved in research exploring the most effective methods of managing chest pain. Similar methods have been found to be useful with a variety of pain conditions. This research has been designed to explore whether such an intervention may improve upon or supplement current medical practices.

If you choose to participate, you will be asked to report for 3 sessions which are approximately 1-1/2 hours long, to be held weekly at the Jackson VAMC. (Today’s session will be the first of these three.) You will participate with a number of other individuals in a small group (5-10) where you will be taught skills designed to help you manage your chest pain. During these meetings, you will be asked to fill out questionnaires and forms. Finally, we will be following your progress over the course of the 12 months after your treatment, either at your regularly scheduled CV Clinic appointment, or by mail.

If you choose not to participate in this study, this decision will not affect your relationship with Cardiology Service, Psychology Service or the VA Medical Center in any way. In addition, if you decide to participate, you are free to withdraw at any time, for any reason.

All of the information you provide during this study will be kept strictly confidential, and will be available only to the investigators of the project unless we obtain written permission from you. No information which may identify you will be used in any reports of the results obtained from this study.

Risks

There are no known risks associated with participation in this study. As mentioned above, any personal information you reveal to us will be kept in the strictest confidence.

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Institutional Review Board
James L. Achord, MD
Chairman
University of Mississippi
Medical Center
2500 N. State Street
Jackson, MS 39216

Patient’s Signature:
______________
Date:
______________

Witness:
______________
Date:
______________
Table F-1

Means, Ranges, and Standard Deviations of Continuous Dependent and Independent Measures

<table>
<thead>
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<th>Variable</th>
<th>Mean</th>
<th>Range</th>
<th>SD</th>
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<td>0 - 18</td>
<td>4.55</td>
</tr>
<tr>
<td>MCSD-C</td>
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<td>0 - 13</td>
<td>3.02</td>
</tr>
<tr>
<td>Age</td>
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<td>37 - 65</td>
<td>6.91</td>
</tr>
<tr>
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<td>0 - 4</td>
<td>1.07</td>
</tr>
<tr>
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<td>3.46</td>
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<td>0 - 40</td>
<td>9.99</td>
</tr>
<tr>
<td>Interference</td>
<td>5.41</td>
<td>2 - 10</td>
<td>2.91</td>
</tr>
</tbody>
</table>

Note. TMAS-S = Taylor Manifest Anxiety Scale, Short Form (Bendig, 1956); MCSD-C = Marlowe-Crowne Social Desirability Scale Form C (Reynolds, 1982); Stenosis 1 = number of major cardiac arteries with more than 50% stenosis; Stenosis 2 = total occlusion rating for major cardiac arteries; Pain Index = frequency of pain x intensity of pain; Interference = interference in daily activities.
Table F-2

Correlations of Stenosis Measures, Pain Index, and Impairment in Daily Living with TMAS-S

<table>
<thead>
<tr>
<th>Stenosis 1</th>
<th>Stenosis 2</th>
<th>Pain Index</th>
<th>Interference</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.01</td>
<td>-0.02</td>
<td>0.22</td>
<td>0.09</td>
</tr>
</tbody>
</table>

Note. TMAS-S = Taylor Manifest Anxiety Scale, Short Form (Bendig, 1956); Stenosis 1 = number of major cardiac arteries with more than 50% stenosis; Stenosis 2 = total occlusion rating for major cardiac arteries; Pain Index = frequency of pain x intensity of pain; Interference = interference in daily activities.
Table F-3

Correlations Between Criterion and Predictor Variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>Sten 1</th>
<th>Sten 2</th>
<th>Pain Index</th>
<th>Interference</th>
</tr>
</thead>
<tbody>
<tr>
<td>TMAS-S</td>
<td>.01</td>
<td>-.02</td>
<td>.22</td>
<td>.09</td>
</tr>
<tr>
<td>MCSD-C</td>
<td>.07</td>
<td>.15</td>
<td>.10</td>
<td>.07</td>
</tr>
<tr>
<td>Age</td>
<td>.13</td>
<td>.14</td>
<td>-.13</td>
<td>-.10</td>
</tr>
<tr>
<td>Fam Hx</td>
<td>.19</td>
<td>.26</td>
<td>.03</td>
<td>.02</td>
</tr>
<tr>
<td>Smoker</td>
<td>-.15</td>
<td>-.18</td>
<td>.19</td>
<td>.37*</td>
</tr>
<tr>
<td>HIN</td>
<td>.02</td>
<td>.01</td>
<td>.05</td>
<td>.07</td>
</tr>
<tr>
<td>DM</td>
<td>-.16</td>
<td>-.10</td>
<td>-.03</td>
<td>-.08</td>
</tr>
</tbody>
</table>

Note. TMAS-S = Taylor Manifest Anxiety Scale, Short Form (Bendig, 1956); MCSD-C = Marlowe-Crowne Social Desirability Scale Form C (Reynolds, 1982); Sten 1 = number of major arteries with more than 50% occlusion; Sten 2 = total of occlusion ratings for 4 major cardiac arteries; Pain Index = frequency of pain X severity of pain; Fam Hx = positive family history of heart disease; HIN = diagnosis of hypertension; DM = diagnosis of diabetes mellitus.

*p < .001.
Table F-4

Summary Table: Multiple Regression TMAS-S and MCSD-C Scores, and TMAS-S X MCSD-C Interaction. Dependent Variable of Stenosis 1: Number of Major Cardiac Arteries with Greater than 50% Stenosis

<table>
<thead>
<tr>
<th>Step</th>
<th>Var.</th>
<th>Beta In</th>
<th>$R^2$ change</th>
<th>F</th>
<th>p</th>
<th>Cumulative $R^2$</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>TMAS-S</td>
<td>.01</td>
<td>.00</td>
<td>.00</td>
<td>&lt;.95</td>
<td>.00</td>
<td>.00</td>
<td>&lt;.95</td>
</tr>
<tr>
<td>2</td>
<td>MCSD-C</td>
<td>.09</td>
<td>.01</td>
<td>.51</td>
<td>&lt;.48</td>
<td>.01</td>
<td>.26</td>
<td>&lt;.77</td>
</tr>
<tr>
<td>3</td>
<td>TxMC</td>
<td>.05</td>
<td>.00</td>
<td>.03</td>
<td>&lt;.87</td>
<td>.01</td>
<td>.18</td>
<td>&lt;.91</td>
</tr>
</tbody>
</table>

Note. TMAS-S = Taylor Manifest Anxiety Scale, Short Form (Bendig, 1956); MCSD-C = Marlowe-Crowne Social Desirability Scale Form C (Reynolds, 1982); TxMC = TMAS-S X MCSD-C interaction.
Table F-5

Summary Table: Multiple Regression TMAS-S and MCSD-C Scores, and TMAS-S X MCSD-C Interaction. Dependent Variable of Stenosis 2: Total Rating of Extent of Stenosis in Major Cardiac Arteries

<table>
<thead>
<tr>
<th>Step</th>
<th>Var.</th>
<th>Beta In</th>
<th>$R^2$ change</th>
<th>F</th>
<th>p</th>
<th>$R^2$</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>TMAS-S</td>
<td>-0.02</td>
<td>0.00</td>
<td>0.02</td>
<td>&lt;.88</td>
<td>0.00</td>
<td>0.02</td>
<td>&lt;.88</td>
</tr>
<tr>
<td>2</td>
<td>MCSD-C</td>
<td>0.17</td>
<td>0.02</td>
<td>1.75</td>
<td>&lt;.19</td>
<td>0.02</td>
<td>0.89</td>
<td>&lt;.42</td>
</tr>
<tr>
<td>3</td>
<td>TxMC</td>
<td>0.21</td>
<td>0.01</td>
<td>0.49</td>
<td>&lt;.49</td>
<td>0.03</td>
<td>0.75</td>
<td>&lt;.53</td>
</tr>
</tbody>
</table>

Note. TMAS-S = Taylor Manifest Anxiety Scale, Short Form (Bendig, 1956); MCSD-C = Marlowe-Crowne Social Desirability Scale Form C (Reynolds, 1982); TxMC = TMAS-S X MCSD-C interaction.
Table F-6

Summary Table: Multiple Regression TMAS-S and MCSD-C Scores, and TMAS-S X MCSD-C Interaction. Dependent Variable of Interference in Daily Activities Due to Pain

<table>
<thead>
<tr>
<th>Step Var.</th>
<th>Beta In</th>
<th>$R^2$ change</th>
<th>F</th>
<th>p</th>
<th>$R^2$</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>TMAS-S</td>
<td>.09</td>
<td>.01</td>
<td>.59</td>
<td>&lt;.45</td>
<td>.01</td>
<td>.59</td>
</tr>
<tr>
<td>2</td>
<td>MCSD-C</td>
<td>.13</td>
<td>.01</td>
<td>1.01</td>
<td>&lt;.32</td>
<td>.02</td>
<td>.80</td>
</tr>
<tr>
<td>3</td>
<td>TxMC</td>
<td>.45</td>
<td>.03</td>
<td>2.32</td>
<td>&lt;.13</td>
<td>.05</td>
<td>1.31</td>
</tr>
</tbody>
</table>

Note. TMAS-S = Taylor Manifest Anxiety Scale, Short Form (Bendig, 1956); MCSD-C = Marlowe-Crowne Social Desirability Scale Form C (Reynolds, 1982); TxMC = TMAS-S X MCSD-C interaction.
### Table F-7

**Summary Table: Multiple Regression TMAS-S and MCSD-C Scores, and TMAS-S X MCSD-C Interaction. Dependent Variable of Pain Frequency x Intensity Index**

<table>
<thead>
<tr>
<th>Step</th>
<th>Var.</th>
<th>Beta In</th>
<th>$R^2$ change</th>
<th>F</th>
<th>p</th>
<th>$R^2$</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>TMAS-S</td>
<td>.22</td>
<td>.05</td>
<td>3.78</td>
<td>&lt;.06</td>
<td>.05</td>
<td>3.78</td>
<td>&lt;.06</td>
</tr>
<tr>
<td>2</td>
<td>MCSD-C</td>
<td>.23</td>
<td>.05</td>
<td>3.63</td>
<td>&lt;.06</td>
<td>.09</td>
<td>3.77</td>
<td>&lt;.03</td>
</tr>
<tr>
<td>3</td>
<td>TxMC</td>
<td>.78</td>
<td>.09</td>
<td>8.30</td>
<td>&lt;.01</td>
<td>.19</td>
<td>5.53</td>
<td>&lt;.01</td>
</tr>
</tbody>
</table>

Note. TMAS-S = Taylor Manifest Anxiety Scale, Short Form (Bendig, 1956); MCSD-C = Marlowe-Crowne Social Desirability Scale Form C (Reynolds, 1982); TxMC = TMAS-S X MCSD-C interaction.
Table F-8

Summary Table: Multiple Regression of Demographic and Medical Factors, TMAS-S and MCSD-C Scores, and TMAS-S X MCSD-C Interaction. Dependent Variable of Stenosis 1: Number of Major Cardiac Arteries with Greater than 50% Stenosis

<table>
<thead>
<tr>
<th>Step</th>
<th>Var.</th>
<th>Beta In</th>
<th>$R^2$ change</th>
<th>$F$</th>
<th>$p$</th>
<th>$R^2$</th>
<th>$F$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>FamHx</td>
<td>.19</td>
<td></td>
<td></td>
<td></td>
<td>.11</td>
<td>1.77</td>
<td>&lt;.13</td>
</tr>
<tr>
<td>2</td>
<td>Age</td>
<td>.13</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>HIN</td>
<td>.02</td>
<td></td>
<td></td>
<td></td>
<td>.11</td>
<td>1.77</td>
<td>&lt;.13</td>
</tr>
<tr>
<td>4</td>
<td>Smoker</td>
<td>-.14</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>DM</td>
<td>-.21</td>
<td>.11</td>
<td>1.77</td>
<td>&lt;.13</td>
<td>.11</td>
<td>1.77</td>
<td>&lt;.13</td>
</tr>
<tr>
<td>6</td>
<td>TMAS-S</td>
<td>-.07</td>
<td>.00</td>
<td>.29</td>
<td>&lt;.59</td>
<td>.12</td>
<td>1.51</td>
<td>&lt;.19</td>
</tr>
<tr>
<td>7</td>
<td>MCSD-C</td>
<td>.08</td>
<td>.00</td>
<td>.37</td>
<td>&lt;.54</td>
<td>.12</td>
<td>1.33</td>
<td>&lt;.25</td>
</tr>
<tr>
<td>8</td>
<td>TxMC</td>
<td>-.06</td>
<td>.00</td>
<td>.04</td>
<td>&lt;.84</td>
<td>.12</td>
<td>1.16</td>
<td>&lt;.34</td>
</tr>
</tbody>
</table>

Note. Fam Hx = positive family history of heart disease; HIN = diagnosis of hypertension; DM = diagnosis of diabetes mellitus; TMAS-S = Taylor Manifest Anxiety Scale, Short Form (Bendig, 1956); MCSD-C = Marlowe-Crowne Social Desirability Scale Form C (Reynolds, 1982); TxMC = TMAS-S x MCSD-C interaction.
Table F-9

Summary Table: Multiple Regression of Demographic and Medical Factors, TMAS-S and MCSD-C Scores, and TMAS-S X MCSD-C Interaction. Dependent Variable of Stenosis 2: Total Rating of Extent of Stenosis in Major Cardiac Arteries

<table>
<thead>
<tr>
<th>Step</th>
<th>Var.</th>
<th>Beta In</th>
<th>$R^2$ change</th>
<th>$F$</th>
<th>$p$</th>
<th>Cumulative $R^2$</th>
<th>$F$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>FamHx</td>
<td>.26</td>
<td></td>
<td></td>
<td></td>
<td>.26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Age</td>
<td>.14</td>
<td></td>
<td></td>
<td></td>
<td>.13</td>
<td>2.12</td>
<td>&lt;.07</td>
</tr>
<tr>
<td>3</td>
<td>HIN</td>
<td>.01</td>
<td></td>
<td></td>
<td></td>
<td>.13</td>
<td>.41</td>
<td>&lt;.53</td>
</tr>
<tr>
<td>4</td>
<td>Smoker</td>
<td>-.16</td>
<td></td>
<td></td>
<td></td>
<td>.14</td>
<td>1.24</td>
<td>&lt;.27</td>
</tr>
<tr>
<td>5</td>
<td>DM</td>
<td>-.15</td>
<td>.13</td>
<td>2.12</td>
<td>&lt;.07</td>
<td>.13</td>
<td>1.82</td>
<td>&lt;.11</td>
</tr>
<tr>
<td>6</td>
<td>TMAS-S</td>
<td>-.08</td>
<td>.01</td>
<td>.41</td>
<td>&lt;.53</td>
<td>.14</td>
<td>1.74</td>
<td>&lt;.11</td>
</tr>
<tr>
<td>7</td>
<td>MCSD-C</td>
<td>.14</td>
<td>.02</td>
<td>1.24</td>
<td>&lt;.27</td>
<td>.15</td>
<td>1.52</td>
<td>&lt;.17</td>
</tr>
<tr>
<td>8</td>
<td>TxMC</td>
<td>.09</td>
<td>.00</td>
<td>.09</td>
<td>&lt;.77</td>
<td>.15</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. Fam Hx = positive family history of heart disease; HIN = diagnosis of hypertension; DM = diagnosis of diabetes mellitus; TMAS-S = Taylor Manifest Anxiety Scale, Short Form (Bendig, 1956); MCSD-C = Marlowe-Crowne Social Desirability Scale Form C (Reynolds, 1982); TxMC = TMAS-S x MCSD-C interaction.
Table F-10

Summary Table - MANOVA. Comparisons of Degree of Stenosis, Pain, and Impairment in Daily Activities Between Subjects Grouped by TMAS-S and MCSD-C

<table>
<thead>
<tr>
<th>Effect</th>
<th>Value</th>
<th>Approx F (df=4)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>TMAS-S</td>
<td>.91</td>
<td>1.71</td>
<td>&lt;.16</td>
</tr>
<tr>
<td>MCSD-C</td>
<td>.92</td>
<td>1.52</td>
<td>&lt;.21</td>
</tr>
<tr>
<td>TMAS-S x MCSD-C</td>
<td>.96</td>
<td>.76</td>
<td>&lt;.56</td>
</tr>
</tbody>
</table>

(table continues)
Table F-10 (continued)

Univariate F-Tests

<table>
<thead>
<tr>
<th>Effect</th>
<th>Dependent Variable</th>
<th>F (1,72)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>TMAS-S</td>
<td>Stenosis 1</td>
<td>.18</td>
<td>&lt;.671</td>
</tr>
<tr>
<td></td>
<td>Stenosis 2</td>
<td>.51</td>
<td>&lt;.48</td>
</tr>
<tr>
<td></td>
<td>Interference</td>
<td>1.03</td>
<td>&lt;.31</td>
</tr>
<tr>
<td></td>
<td>Pain Index</td>
<td>5.24</td>
<td>&lt;.03</td>
</tr>
<tr>
<td>MCSD-C</td>
<td>Stenosis 1</td>
<td>.10</td>
<td>&lt;.76</td>
</tr>
<tr>
<td></td>
<td>Stenosis 2</td>
<td>1.12</td>
<td>&lt;.29</td>
</tr>
<tr>
<td></td>
<td>Interference</td>
<td>1.22</td>
<td>&lt;.27</td>
</tr>
<tr>
<td></td>
<td>Pain Index</td>
<td>2.19</td>
<td>&lt;.14</td>
</tr>
<tr>
<td>TMAS-S x MCSD-C</td>
<td>Stenosis 1</td>
<td>.01</td>
<td>&lt;.91</td>
</tr>
<tr>
<td></td>
<td>Stenosis 2</td>
<td>.17</td>
<td>&lt;.68</td>
</tr>
<tr>
<td></td>
<td>Interference</td>
<td>.09</td>
<td>&lt;.76</td>
</tr>
<tr>
<td></td>
<td>Pain Index</td>
<td>1.09</td>
<td>&lt;.30</td>
</tr>
</tbody>
</table>

Note. TMAS-S = Taylor Manifest Anxiety Scale, Short Form (Bendig, 1956); MCSD-C = Marlowe-Crowne Social Desirability Scale Form C (Reynolds, 1982); TmMC = TMAS-S x MCSD-C interaction; Stenosis 1 = number of major cardiac arteries with more than 50% stenosis; Stenosis 2 = total occlusion rating for major cardiac arteries; Interference = interference in daily activities; Pain Index = frequency of pain x intensity of pain.
Table F-11

Summary Table - MANOVA. Comparisons of TMAS-S and MCSD-C Scores Between Subjects Grouped by Pain Level and Stenosis Level

<table>
<thead>
<tr>
<th>Effect</th>
<th>Value</th>
<th>Approx F (df=2)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stenosis 2</td>
<td>.99</td>
<td>.37</td>
<td>&lt;.69</td>
</tr>
<tr>
<td>Pain Index</td>
<td>.96</td>
<td>1.59</td>
<td>&lt;.21</td>
</tr>
<tr>
<td>Sten x Pain</td>
<td>.96</td>
<td>1.41</td>
<td>&lt;.25</td>
</tr>
</tbody>
</table>

(table continues)
Table F-11 (continued)

<table>
<thead>
<tr>
<th>Effect</th>
<th>Dependent Variable</th>
<th>F (1,72)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stenosis 2</td>
<td>TMAS-S</td>
<td>.30</td>
<td>&lt;.59</td>
</tr>
<tr>
<td></td>
<td>MCSD-C</td>
<td>.13</td>
<td>&lt;.72</td>
</tr>
<tr>
<td>Pain Index</td>
<td>TMAS-S</td>
<td>.89</td>
<td>&lt;.35</td>
</tr>
<tr>
<td></td>
<td>MCSD-C</td>
<td>.91</td>
<td>&lt;.34</td>
</tr>
<tr>
<td>Sten x Pain</td>
<td>TMAS-S</td>
<td>2.00</td>
<td>&lt;.16</td>
</tr>
<tr>
<td></td>
<td>MCSD-C</td>
<td>.04</td>
<td>&lt;.84</td>
</tr>
</tbody>
</table>

Note. Stenosis 2 = total occlusion rating for major cardiac arteries; Pain Index = frequency of pain x intensity of pain; Sten x Pain = Stenosis x Pain interaction; TMAS-S = Taylor Manifest Anxiety Scale, Short Form (Bendig, 1956); MCSD-C = Marlowe-Crowne Social Desirability Scale Form C (Reynolds, 1982).
Appendix G

Vita
Vita

SHARON ELAINE ALCOCK PARISI

ADDRESS:  3239 Holly Mill Run
           Marietta, GA  30062
Home Phone:  (404) 565-6564

PERSONAL DATA:  Date of Birth:  March 12, 1949
                 Place of Birth:  Hampton, VA
                 Married, 2 children

EDUCATION:

1986 - 1987  Clinical Psychology Resident
             University of Mississippi Medical Center/Veterans Administration Medical Center
             Jackson, MS
             (APA Approved)

1982 - Present  PhD Candidate - Clinical Psychology
                Louisiana State University
                Baton Rouge, LA
                (APA Approved)
                Specialty Area:  Behavioral Medicine
                Minor:  Behavioral Neurology
                Dissertation:  "Coronary Heart Disease, Negative Affectivity, and Response Bias."
                Degree Expected:  August, 1990

1973 - 1975  MA, Clinical Psychology
             Vanderbilt University
             Nashville, TN
             (APA Approved)
             Thesis:  "Five-, Seven-, and Nine-month-old Infants' Facial Responses to Twenty Stimulus Situations."
             Degree awarded:  August, 1977

1971 - 1973  BA, Psychology
             Rhode Island College
             Providence, RI
             Degree awarded:  June, 1973

1967 - 1969  University of New Orleans
             New Orleans, LA
             Majors:  Psychology and Philosophy
CLINICAL EXPERIENCE:

8/89 - present  Psychology Associate. Conduct comprehensive neuropsychological evaluations of brain injured individuals and behavioral pain assessments of individuals with chronic pain difficulties. Also provide psychotherapy to brain injured patients and their families. Supervisor: Stephen J. Johnson, PhD

8/87 - 9/88  Psychology Assistant - Behavioral Medicine. Conducted psychological assessments of cardiac, cancer, and gastric disease patients who were involved in various research studies. Led behavioral intervention groups for cardiac patients. Provided long-term psychotherapy to Vietnam Veterans with combat-related post-traumatic stress disorder. Supervisor: Phillip Godding, PhD

8/86 - 8/87  Clinical Psychology Resident. University of Mississippi Medical Center/ Jackson VA Medical Center Internship Consortium. APA Approved.

Behavioral Neuropsychology (3 months). Provided psychological consultation to neurology, neurosurgery, and psychiatry services. Conducted comprehensive neuropsychological assessments and functional analyses of patients with various medical and psychiatric disorders. Provided extensive reports to referral sources and detailed feedback to the patients and their families, with recommendations for modifying living arrangements to accommodate the limitations of the patients. Supervisor: Kathryn L. Kerr, PhD.

Behavioral Gerontology (6 months). Provided psychological consultation to medical/surgical units, especially the Geriatric Evaluation Unit and Nursing Home Care Unit. Member of an interdisciplinary planning and treatment team. Assessed patients' cognitive and functional abilities, and conducted neuropsychological screenings for differential diagnosis of
depression and dementia. Developed and managed behavioral intervention programs for patients and nursing home residents presenting with a variety of problems. Conducted individual behavior therapy with patients for treatment of depression and for improvement of compliance with medical regimens, and provided supportive therapy to families of patients. Involved in research on depression and aging, and on the detection of depression in geriatric patients by primary care physicians. Supervisor: Stephen R. Rapp, PhD

**Trauma Recovery Program** (3 months). Conducted intensive, multimethod assessments of Vietnam Veterans for presence of combat-related post-traumatic stress disorders (PTSD). Provided individual behavior therapy to PTSD patients, with emphasis on anxiety reduction, stress management, anger control, and depression. Provided consultation to psychiatry inpatient wards. Also worked in conjunction with local Vet Center (Vietnam Veterans Outreach Program). Supervisor: John A. Fairbank, PhD.

4/86 - 8/86 **Psychology Assistant**, VA Medical Center, Jackson, MS. Conducted diagnostic evaluations of patients with sexual dysfunction who were being considered for surgical intervention. Supervisor: Patricia Dubbert, PhD

6/84 - 6/85 **Medical Psychology Trainee - Medical Consultation/Liaison**, Earl K. Long Memorial Hospital, Baton Rouge, LA. Conducted psychological assessments and brief treatment for adult medical inpatients and outpatients, including those in intensive care and in the emergency room. Presenting problems included depression, anxiety secondary to hospitalization and/or medical procedures, medical non-compliance, and trauma recovery. Patients seen included those with cardiovascular disease, renal disease, diabetes, myasthenia gravis, and those who were victims of physical abuse. Supervisor: Phillip J. Brantley, PhD
6/83 - 6/84  **Medical Psychology Trainee - Family Practice**, Earl K. Long Memorial Hospital, Baton Rouge, LA. Conducted psychological assessments and therapy with adult outpatients referred from family practice physicians. Also provided biofeedback treatment to patients with migraine headaches.
Supervisor: Phillip J. Brantley, PhD

9/82 - 6/83  **Adult Psychology Trainee**. Psychological Services Center, Louisiana State University, Baton Rouge, LA. Psychological assessment and treatment of adult outpatients. Problems treated included depression, anxiety, and personality disorders.
Supervisor: Don Williamson, PhD

9/81 - 12/82  **School Psychological Assistant**. St. Tammany Parish Pupil Appraisal, Covington, LA. Conducted psycho-educational assessments of gifted, learning disabled, and behavior disordered children.
Supervisor: Barbara Lehrman, PhD

1/79 - 7/79  **Psychological Assistant**. Hammond State School, Hammond, LA. Provided intellectual and behavioral assessments of mentally retarded individuals, and developed recommendations for individualized behavior modification programs.
Supervisor: Harold Katz, PhD

4/76 - 1/79  **Vocational Rehabilitation Senior Counselor**. Louisiana State Department of Health and Human Resources, Hammond, LA. Counseled physically handicapped individuals, with ultimate goal of placement in satisfactory employment. Defined training and educational needs, set up individual vocational development programs, located employment opportunities, and arranged for state and federal financial assistance in cases of bona fide indigence and disability. Streamlined, organized, and managed previously neglected caseload of 300+ individuals.
Supervisor: Wayne T. Kitchens, MA
9/74 - 8/75  Psychology Trainee. Interuniversity Psychological and Counseling Center, Vanderbilt University, Nashville, TN. Conducted individual, marital, and group therapy for university undergraduates, staff members, and their families. Also conducted group therapy with juvenile offenders from local residential program. Supervisors: Lawrence Weitz, PhD and Kenneth Anchor, PhD

9/74 - 6/75  Psychological Assessment Trainee. Psychology Division, Department of Psychiatry, Vanderbilt University Medical School Hospital, Nashville, TN. Conducted psychological assessments of adult psychiatric inpatients. Supervisor: Warren Webb, PhD

1/74 - 6/74  Child Psychology Trainee. John F. Kennedy Child Study Center, Vanderbilt University, Nashville, TN. Developed and carried out individual behavior modification plans with retarded, autistic, and "high risk" children under 5 years of age. Supervisor: Jan Odom, MS

RESEARCH EXPERIENCE:

12/87 - present  VA Medical Center, Jackson, MS. Member of research team investigating predictors of treatment outcome in stress management training for chest pain patients. Contributed to development of research design, treatment manual, and construction of behavioral measures. Administered assessment packages to patients, and led behavioral intervention groups. Currently participating in data analysis and manuscript preparation.

1/88 - 7/90  VA Medical Center, Jackson, MS. Primary investigator on study of negative affectivity and cardiovascular heart disease, with emphasis on the role of response bias in CHD research. Developed hypotheses, based upon integration of previous research, planned research design, selected appropriate measures, and developed and implemented research protocols, analyzed and interpreted data, and prepared manuscripts.
Primary Investigators: Thomas Payne, PhD, Donald Penzein, PhD, and Cheryl Johnson, PhD

8/87 - 9/88 Co-project Director. VA Medical Center, Jackson, MS. Investigation of anxiety and coping styles in patients who are undergoing invasive medical procedures. Funded by VA Regional Grant. Established research procedures and protocols, modified existing psychometric instruments as appropriate, developed and piloted new measures. Conducted psychological assessments of patients both prior to and after medical procedures, observed patients and collected behavioral measures during the medical procedures. Developed and implemented data management systems. Also participated in design and implementation of study on memory function in patients undergoing chemotherapy.

Primary Investigator: Phillip R. Godding, PhD

4/85 - 8/87 Research Assistant. VA Medical Center, Jackson, MS. Investigation of depression in elderly medical patients, and detection of depression in these patients by primary health care physicians. Funded by VA Health Systems Research and Development grant. During initial and 1-year follow-up phases, administered cognitive screening instruments and conducted diagnostic interviews of all subjects, using Schedule for Affective Disorders and Schizophrenia (SADS). Administered additional battery of psychometric instruments, and provided appropriate clinical feedback to physicians. Developed and managed data collection. Prepared and edited manuscripts. Trained research assistant for second and third year follow-ups.

Primary Investigator: Stephen R. Rapp, PhD

9/84 - 6/85 Research Assistant. University of Mississippi Medical Center, Jackson, MS. Conducted study on patterns of referrals to Psychology Department. Also conducted psychophysiological assessments of patients.
during relaxation induction procedures.  
Primary Investigator: Ellie Sturgis, PhD

1/84 - 6/85  
Louisiana State University, Baton Rouge, LA.  Member of a research team investigating the correlates of successful outcome in biofeedback treatment of migraine headaches.  
Primary Investigator: Phillip J. Brantley, PhD

1/84 - 6/84  
Louisiana State University, Baton Rouge, LA.  Member of a research team investigating the relative effectiveness of different group methods for teaching communication skills to couples.  
Primary Investigator: Bernard J. Jensen, PhD

9/73 - 6/75  
Vanderbilt University, Nashville, TN.  Member of a research team investigating the development of emotion expression and perception of emotion in infants and children.  
Primary Investigator: Carroll Izard, PhD

5/74 - 8/74  
Research Assistant.  George Peabody College, Nashville, TN.  Longitudinal study of the development of auditory and visual perception in infants and young children.  
Primary Investigator: Peter Vietze, PhD

9/72 - 6/73  
Rhode Island College, Providence, R.I.  Co-investigator on study of the effects of stress on creativity in children.  
Primary Investigator: Terence Belcher, PhD

TEACHING EXPERIENCE:

9/74 - 6/75  
Teaching Assistant.  Psychology Department, Vanderbilt University, Nashville, TN.  Undergraduate statistics course.  
Supervisor: Leland Thune, PhD

PROFESSIONAL AFFILIATIONS:

Association for the Advancement of Behavior Therapy  
Southeastern Psychological Association
HONORS AND AWARDS:

1973 - 1975
NIMH Clinical Psychology Fellowship,
Vanderbilt University

1972 - 1973
Rhode Island College Honors Scholarship

1967 - 1969
University of New Orleans Centennial Honor Award

PUBLICATIONS:


PROFESSIONAL PRESENTATIONS:


WORKSHOPS PRESENTED:


MANUSCRIPTS UNDER EDITORIAL REVIEW:


RESEARCH IN PROGRESS:

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Stephen R. Rapp, PhD
Department of Epidemiology and Public Health
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Candidate: Sharon Elaine Parisi

Major Field: Psychology

Title of Dissertation: Coronary Heart Disease, Negative Affectivity, and Response Bias

Approved:

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Major Professor and Chairman

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EXAMINING COMMITTEE:

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Date of Examination:

July 10, 1990