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Emotion, Facial Expression, and Coronary Artery Disease

by

Erika L. Rosenberg

DISSERTATION

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Emotion, Facial Expression, and Coronary Artery Disease

By

Erika L. Rosenberg

Preface and Acknowledgments

This dissertation serves as an ideal cap to my graduate career not only for its obvious role as the last requirement fulfilled, but also for its topic. The study of emotion and coronary artery disease joins my research experience in the study of facial expression and emotion with the area of interest that first drew me to pursue graduate work in Health Psychology -- the relationship between stress and illness.

Thus, in spirit this project was born early in my graduate career. More practically, however, it was born of opportunity. Around the time I had completed my qualifying exams and was preparing to choose a dissertation topic, Paul Ekman and James Blumenthal (of Duke University) had begun a collaborative project on emotion and transient myocardial ischemia. Ekman and Blumenthal offered me the opportunity of working with their data (thereby offering grant support for my research) to pursue my own questions about emotion and health for a dissertation. This dataset offered me the chance to extend my basic research interests in emotional processes to an applied context *and* to apply my expertise in emotion to an important health question that sorely needed conceptual and methodological clarification.

For this opportunity, and countless other things, I express my deepest gratitude and sincerest respect to Paul Ekman, my mentor and chairman of my dissertation committee. I came to Ekman's lab in my second year of graduate school, because I wanted to learn about emotion. I had no idea that I was entering research powerhouse, a methodological gold mine, and no less than an academic boot camp. From Ekman I have learned not only the art and science of facial measurement, emotion theory, and general research design, but also invaluable lessons about *doing* science, professionalism, ethics in research, and academic politics that were not a formal part of the graduate school

demand it from myself. For that I am ever grateful. I feel truly fortunate to have trained with one of the most important figures in the Psychology of Emotion of the 20th century.

I thank Jim Blumenthal, for his great generosity in sharing a dataset in which he had invested hundreds of hours and dollars before I began even writing my proposal. Additionally, I thank Blumenthal for contributing his expertise in the coronary health domain to my dissertation development and execution. I am grateful also to my other committee members -- Nancy Adler and Len Pearlin -- for their incisive and thoughtful comments on my proposal and a dissertation manuscripts.

My thanks to the following important figures in my life:

• My parents, Lawrence and Sandra Rosenberg, for valuing education, supporting my decisions in life, and providing financial cushion far beyond the call of duty. Most importantly, I thank them for their unwavering, unconditional love. Never have I doubted it, never have I lacked it, and never more than now have I realized how fortunate I am to have it.

• Greg Feist, my husband, best friend, and peer, for his unfaltering love and support; providing me with living proof that graduate school does indeed end; never doubting that I would ever finish even when I wasn't so sure; making me feel special on a daily basis; providing the stability and freedom that enabled my intellectual and spiritual growth as well as the peace of mind to focus on my work; and for picking up the economic slack for just "one more year."

• My sister and brother, Lisa and Greg Rosenberg. Lisa, for being my closest friend for life, an intellectual compatriot, and my poet alter-ego. I also thank her for being a positive role model for academic excellence when it mattered most -- elementary school and junior high school (I wouldn't have *lived* if I didn't get into Algebra early, too!). To Greg -- for great affection, love, camaraderie, and welcome comic relief throughout

child- and adulthood. We share a light-heartedness that we have only begun to realize.

• Pauline Saltman, my grandmother and number-one fan, thanks for loving me undyingly (as only a grandmother can), encouraging my self-reliance, having enormous faith and confidence in my abilities, and providing a fabulous feminist influence. She will always be, in my mind, a woman ahead of her time. And love and thanks to Peter Saltman, my dear grandfather, for enduring Grandma's incessant praise of me, for loving me nonetheless, and for being the only grandfather with whom I have had the privilege of having a conversation as an adult.

• Spalding: for being the fluffiest cat in the world and staying up late with me.

• Sogyal Rinpoche for opening the door; Tarthang Tulku and the Nyingma Institute of Buddhist Studies in Berkeley for helping me develop mindfulness, enabling me with the "skillful means" to get work done, and setting me on the path.

• Jerry, Bobby, et al.: for buoying me to safety and soaring me to the brightest depths more often and in more ways than I could have ever imagined. Once in a while you really *do* get shown the light in the strangest of places if you look at it right.

• To my dear friends and partners in crime: Caroline -- my rock; Mary and Allen (for that first New Year's -- I can *never* say how grateful I am); Barb, for being the exquisite combination of professional role model and first-class goof-ball, for *finally* understanding why they wear those skirts, and for making a commitment to studying the positive emotions; David Galin, first a professor and then a friend (many, many thanks for introducing me to levels of explanation, entertaining my ideas, and being as willing to talk about the perfect fish stock as the perfect theory of consciousness); Aunt Leslie, for being a kindred spirit; and to the rest of my extended-family, Valerie and Bobby ... Wheel to the storm and fly!

EMOTION, FACIAL EXPRESSION, AND CORONARY ARTERY DISEASE

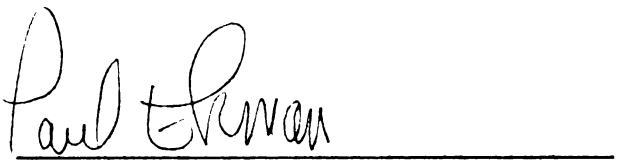
Erika L. Rosenberg

Abstract

In order to examine the role of emotions in heart disease, facial expressions of emotion were studied in 94 (88 male and 6 female) coronary artery disease patients during the Type A Structured Interview. Study 1 examined the relationships between expressions of emotion and the incidence of transient myocardial ischemia, a clinical manifestation of coronary artery disease, in the male subjects. As predicted, facial expressions of anger and non-enjoyment smiling occurred significantly more often in patients who showed ischemia than those who did not. Counter to prediction, expressions of contempt, disgust, and the partial anger expression known as Glare did not discriminate between groups. Linear discriminant analyses showed that the facial behavior pattern of anger expression and non-enjoyment smiling accurately classified subjects as to their ischemic status 69% of the time. This hit-rate increased to 82% when only those subjects who reported experiencing above the median negative emotion were included in the function, which helped identify a sub-group of ischemics who did *not* show the characteristic anger/non-enjoyment smiling pattern. Most importantly, the findings from Study 1 further support the notion that anger can have deleterious coronary consequences, as well as pointing to another behavior -- frequent non-enjoyment smiling -- that should be studied further for its potential usefulness as a marker of patients at risk.

In order to specify the expressive and/or affective components of hostility as measured by a widely-used instrument in the coronary health domain, Study 2 examined how facial expressions of emotion could be used to explain variability in Cook-Medley Hostility scores in the full sample of subjects. Although the hypotheses predicted that anger, contempt, and disgust expression would each account for unique portions of

variability in hostility scores, only contempt accounted for a significant amount of unique variance. This finding is consistent with reports of previous psychometric studies that Cook-Medley hostility scores reflect a cynical form of hostility rather than overt aggressiveness. The findings from both studies are discussed in terms of their implications for both health psychology and basic research on emotion .



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Chapter 1

Introduction

Heart disease is a problem to which researchers and practitioners have long attributed an important role for emotion. Yet most researchers in health psychology and behavioral medicine have approached the role of emotion in heart disease indirectly, by relating dispositional factors assessed by personality questionnaires or gross behavioral styles coded from interviews to disease status or measurable cardiovascular changes (for review see Matthews & Haynes, 1986; Thoreson & Powell, 1992). A direct approach to emotion in cardiovascular health psychology would involve the measurement of emotional behavior as it occurs, either in daily life or in the laboratory, as well as the measurement of the coronary consequences of such emotions.

A few researchers have taken the direct approach, or they have approximated one. Krantz and his colleagues (1993) have linked fluctuations in daily mood reports with the occurrence of transient myocardial ischemia during ambulatory assessment. Others have tried to evoke emotion in the laboratory and then measured concomitant or subsequent cardiovascular changes (e.g., Suarez & Williams, 1989). For the most part, however, the literature consists of studies that have related the Type A Behavior Pattern or one of its components -- dispositional hostility -- to blood pressure or heart rate reactivity or to coronary disease incidence, progression, or mortality.

Affective dispositions versus emotions. Many people might consider the study of hostility as the study of emotion, and would therefore argue that emotion has been studied in behavioral medicine for years. While hostility and anger are both affective phenomena, however, they may operate at different levels of affective organization. In the emotion literature, hostility has been described primarily as a trait, as a stable predisposition toward anger. Anger, by contrast, is a transient affective state. Ekman (1984), for example, says that hostility is marked by the ease and frequency of

occurrence of anger. Other theorists, such as Izard (1971, 1977), say that hostility might incorporate disgust and contempt as well. What has appeared in more than one theory -- including those of cognitive emotion theorists such as Ortony, Clore, and Collins (1988) -- is that affective traits set the threshold for the occurrence of their corresponding emotions.

Hostility may cause heart disease because this disposition sets the threshold for the ease of occurrence of anger. It may be the physiological effects of anger itself, and possibly other related emotions, which create the physiological conditions that eventually lead to heart disease.

The causal role of emotion in heart disease

Figure 1 depicts my rendition of the psychophysiological reactivity model of heart disease. There have been various implicit and explicit depictions of this model in the coronary health literature (e.g., Krantz & Durel, 1983). According to the general model, there may be certain affective dispositions that lead people to respond in certain emotional ways to psychological stressors. These responses lead to cardiovascular reactivity (CVR), which may take the form of blood pressure or heart rate increases. The people who tend to response this way more often, undergo more repetitive physiological reactivity to stress -- especially blood pressure reactivity -- that eventually takes its toll on the lining of coronary artery walls. The ensuing damage to the arterial lining increases the likelihood of plaque build-up and, eventually, coronary artery disease (CAD) ensues. Eventually, as the arteries get less efficient the blood supply to the heart is reduced, and coronary heart disease (CHD) develops.

Most previous psychological approaches have been dispositionally oriented. The left-most arrow in Figure 1 depicts a dispositional relationship between emotion and heart disease. For example, hostility -- an affective disposition -- could cause heart disease *via* an emotional response to particular types of situations. It is also possible

that an affective disposition could moderate the relationship between emotion and physiology (as indicated by the middle arrow in Figure 1) in terms of the types of coping strategies that the person draws on to regulate the emotional response. Krantz and Durel (1983) also suggest a direct link between personality and reactivity (indicated by the arrow at far right in Figure 1), in which they propose a feedback mechanism, which is *not* depicted here, by which reactivity is manifest in particular types of behaviors under stress.

It is a complex picture that requires extensive research, but by any account clarifying the relationship between emotion and cardiovascular changes is a fundamental aspect of the model. My goal is to bring the concepts and tools of emotion research more directly into the study of heart disease, because although emotions have been fundamental to the research on coronary-prone behavior over the years, researchers have not been explicit in describing or measuring them.

In the following sections, I review the health psychology and behavioral medicine literature relevant to the study of emotion in heart disease. I discuss how the conceptual narrowing of the field has pointed to a causal role for emotion, and how direct measurement of emotions from people's facial expressions can help pinpoint the specific types of emotions that influence cardiac health. I then present the structure of the two studies that comprise this dissertation research, both of which focus on facial expressions of emotion as a basis for clarifying the role of emotions in coronary health. Most broadly this research addresses the question of whether certain negative emotions play a causal role in heart disease. In this dissertation, however, I investigate this question correlationally. My first study examines the relationship between facial expressions of emotion and clinically significant changes in coronary function during a stressful context, and the second uses facial expressions as a measure of emotion that can be used to specify the affective component of hostility. Both studies address limitations of the current literature and were designed to illuminate our understanding of

the role of emotional behavior in cardiac health. The findings from this correlational research can serve as a foundation and justification for future, prospective and/or experimental work.

The Type A Behavior Pattern

The first major research program on the role of behavior in heart disease involved the well-known Type A Behavior Pattern. According to Friedman and Rosenman (1959; 1974), the cardiologists who first described Type A on the basis of anecdotal observations of their patients' behavior, the Type A person is an impatient, aggressive, and competitive workaholic. Friedman and Rosenman emphasized that the Type A pattern is not a stable trait, but rather it emerges in response to stress or environmental challenge in susceptible individuals. The introduction of the construct and a means to assess it (the Type A Structured Interview or SI) spurred a great deal of research over the past two decades. Researchers have taken several different empirical approaches to examining the role of personality and behavior in coronary disease. The emphasis has been on prediction, and in describing a *coronary-prone* behavior pattern.

Assessment of the Type A Behavior Pattern

The original method for classifying people as Type A or Type B was based on clinical observation and evaluation of the extent to which coronary patients showed the characteristic behaviors described by Rosenman and Friedman (1959; c.f. Blumenthal & Kamarck, 1987). The first formal procedure for assessing the Type A pattern was a structured interview, which was designed to elicit signs of some of the major elements of the behavior pattern. Classification of Type A (impatient, hostile, competitive, and time-urgent) versus Type B (the absence of Type A symptoms, an easy-going, relaxed style) was based primarily on the interviewer's observations of the subject's behavioral signs and speech stylistics (Rosenman, 1978) *not* on self-report of Type A

characteristics. Several measures of Type A have appeared in recent years, including some self-report measures.

The Structured Interview. Rosenman and Friedman (Rosenman et al., 1964) developed the Type A Structured Interview (SI) for use in the first prospective study of Type A behavior and cardiac health -- the Western Collaborative Group Study (WCGS). The original SI consisted of 26 questions about peoples' characteristic ways of responding to a variety of work and social situations, with some questions administered in an intentionally provocative manner. Trained auditors made global ratings of Type A on the basis of subjects' speech content and stylistics during the interview. The initial procedure allowed for 4 levels of classification between A and B: A1 (totally developed Type A), A2 (incompletely developed Type A), B (an absence of Type A characteristics), and X (equal representation of Type A and Type B characteristics). For all practical purposes, however, the classification was dichotomous: A versus B, in which a Type B person was characterized by an absence of any Type A symptoms. Rosenman and Friedman (1978) emphasized that speech style and motoric behavior were equally if not more important to Type A vs. Type B classification than the actual verbal content of interviewee's responses. Indeed, non-verbal speech characteristics such as voice emphasis, latency in answering questions, and speed of speaking have been shown to distinguish between Type As and Type Bs (Blumenthal, O' Toole, & Haney, 1984; Scherwitz, Berton, & Leventhal, 1977, Schucker & Jacobs, 1977).

Other forms of non-verbal behavior, such as facial expressions of certain negative emotions, can be useful in distinguishing Type As from Type Bs (Chesney, Ekman, Friesen, Hecker, & Black, 1990). Chesney and her colleagues pointed out that in Rosenman's (1978) writings on the use of the SI, he "drew attention" to the information that facial behavior reveals about the Type A person, as well. The original WCGS study included data on the observation of such facial characteristics in making the

classification, although this approach has not been taken since. The utility of studying facial expressions during the SI will be discussed in greater detail later in this chapter.

Researchers have adopted several approaches to scoring the SI in recent years, one of the most notable being the Component Scoring System (CSS; Chesney, Hecker, & Black, 1989; Hecker, Chesney, Black, & Frautschi, 1988). The CSS was developed in an effort to move beyond the limited dichotomous classification of the SI, as well as to look at effects of the specific components on coronary heart disease (Chesney, Hecker, & Black, 1989). The CSS offers scores on 14 operationally defined components, as well as a global Type A score.[1]

Another variation on the SI is the Videotaped Structured Interview (VSI; Powell et al., 1984). The VSI differs from the SI in that it is videotaped, is administered in an empathic rather than challenging manner, and is scored on a 38 individual indicators as well as a global score (whereas the traditional SI scoring yields a global score only).

Self-report methods

There are several self-report methods for assessing the Type A pattern, most of which were developed because the SI was cumbersome and difficult to use with large samples.

The Jenkins Activity Survey (JAS; Jenkins, Rosenman, & Friedman, 1964; Jenkins, Zyzanski, & Rosenman, 1979). The JAS is an easily administered, approximately 50 item (depending on which form is used), multiple-choice questionnaire that measures competitiveness, work habits, and a rapid lifestyle. In addition to a global Type A score, the JAS yields three subscores: Factor S (speed and impatience), Factor H (hard-driving & competitiveness), and Factor J (job involvement).

The JAS has been used for Type A-Type B classification in psychophysiological studies with some success (Goldbland, 1980; Manuck & Garland, 1979) and some

[1] The CSS will be discussed in more detail in the section on interview methods for hostility assessment.

failures (Krantz, Arabian, Davia, & Parker, 1982). The failure of JAS Type A classification to predict heart disease in prospective research was demonstrated in the large-scale Multiple Risk Factor Intervention Trial (MRFIT; Shekelle et al., 1985), although the MRFIT also found null findings with the SI classification. Several researchers have attributed this to JAS's failure to assess the affective aspects of the Type A pattern (Ivancevich & Matteson, 1988; Matthews, 1982; Booth-Kewley & H. Friedman, 1987). Thoresen and Powell (1992) said that the JAS captures competitiveness and work involvement aspects of the Type A pattern, while the SI captures aspects more relevant to emotion "anger, impatience or competitiveness as well as suspicious and distrustful attitudes" (p. 596).

Other self-report measures of Type A include the Framingham Type A Scale (Haynes, Levine, Scotch, Feinleib, & Kannel, 1978), the Bortner Type A Scale (Bortner, 1969), and the Type A Self-Report Inventory (TASRI; Blumenthal et al., 1985). These questionnaires have been used much less frequently than the JAS, although the Framingham and Bortner scales were used in large-scale, prospective studies: the Framingham Heart study and the French-Belgian Cooperative Studies, respectively. The TASRI is comparable to the JAS in classifying people as Type A or B, but it takes less time to administer and score (Blumenthal et al., 1985).

Methodological limitations of Type A assessment procedures

Problems with the Structured Interview. The SI has been criticized on the grounds that the scoring techniques (especially systems that derive only a global score) are too subjective (Powell, 1982). This subjectivity leads to inconsistent use of rating scales, and subsequent problems with interrater reliability (Thoresen & Powell, 1992). Also, the A and B classification is limited, with Type B generally connoting an absence of Type A characteristics. Thus, the Type B classification may include people who were "depressed or suppressed" in their interview behavior, which creates a potential

confound, because depression may be independently linked to CHD (Barefoot et al., 1990; H. Friedman & Booth-Kewley, 1987). Research has shown that interviewer behavior can influence Type A classification as well (Scherwitz, 1988). Operational coding systems that score multiple components such as the CSS help remedy both the subjectivity and classification problems to some extent, but they do not address the problem of interviewer effects. Another potential problem is whether Structured Interview based A/B classification of women is valid, as the SI was developed for use with middle-aged, white-collar men (Evans & Moran, 1987).

Problems with Type A self-report instruments. In general, SI assessed Type A is more predictive of CHD than questionnaire assessed Type A (Booth-Kewley & H. Friedman, 1987; Matthews, 1982). The JAS, Bortner, and Framingham scales have shown poor concordance with the SI in Type A and Type B classification (Edwards, Baglioni, & Cooper, 1990). The JAS has shown only modest test-retest reliability (.60 -.70). Correspondences between JAS, Bortner, and Framingham classifications average about .60, which is quite poor for a dichotomous classification scheme (Feuerstein, Labbe, & Kuczmierczyk, 1986). The TASRI, though easier to use than the other self-report techniques, shows misclassification rates similar to those of the JAS. Researchers have noted that self-report instruments tend to tap into the competitiveness and achievement-oriented aspects of the Type A pattern, while the Structured Interview captures responsiveness to stress and the emotional components of the behavior pattern (Blumenthal et al., 1985; Thoresen & Powell, 1992). This problem may result from the fact that self-report procedures cannot draw on behavioral characteristics such as speech style and gestures, which are important aspects of interview-based classification (Scherwitz et al., 1987). Self-report instruments in general are subject to the effects of presentation bias as well, which might also influence the results seen with inventory approaches to Type A assessment.

Evidence for a Relationship Between Type A Behavior and Coronary Disease

In the search for causal mechanisms there is a preference for prospective, predictive studies that follow healthy people over time, assess them psychologically, note their habits, and document heart disease incidence and mortality. Two major prospective studies have looked at the Type A Behavior Pattern and coronary disease outcomes. One is the Western Collaborative Group Study (WCGS), which was initiated in 1960 by M. Friedman and Rosenman and was the first prospective study focusing specifically on Type A behavior and heart disease (c. f., Rosenman et al., 1964). This project has examined psychological factors and coronary heart disease (CHD) incidence and mortality in 3000 men. This research showed that Type A males are twice as likely as Type B to develop heart disease, *over and above* the influence of such strong risk factors as smoking, blood pressure, cholesterol level. Early follow-up studies showed that the TABP continued to predict CHD incidence (Rosenman et al., 1975), but the 22 year follow-up failed to find a relationship between the Type A behavior pattern and CHD mortality (Ragland & Brand, 1988).

The other notable prospective study is the well known Framingham Heart Study, which was a large, population-based prospective project conducted in a small community of Massachusetts, begun in the late 1940's. In the 1970's, the Framingham Type A Scale was developed and administered to approximately 1,800 of the subjects enrolled in the Framingham study (Haynes et al., 1978). The results from this research substantiated the relationship between aspects of the Type A Behavior Pattern and coronary heart disease in both men *and* women (Haynes et al., 1978). The French-Belgian Collaborative Study (French-Belgian Collaborative Group, 1982), in which Type A classification was made from Bortner Scale ratings, also provides evidence that Type A behavior predicts CHD.

In a recent meta-analysis of prospective and cross-sectional research on the Type A pattern and coronary outcomes, Booth-Kewley and H. Friedman (1987) found an effect

size for the relationship between SI assessed Type A behavior and heart disease of .20, which according to Cohen (1977) is a small to moderate effect size. In a qualitative review, Ivancevich and Matteson, (1988) claimed that there is overall support that the Type A behavior pattern is a coronary risk factor. However, they emphasized that certain conflicting findings bring into question conclusions reached from studies of Type A as a coronary risk factor. Ivancevich and Matteson suggested that a more componential approach to measuring Type A in relation to CHD may help resolve some of these problems.

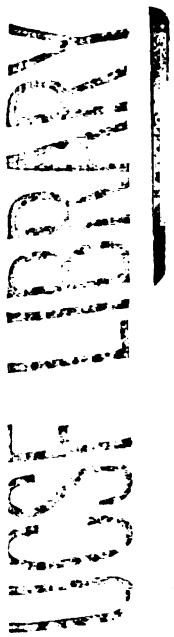
Null findings on the Type A - coronary heart disease relationship

Some prospective studies have failed to find a significant relationship between Type A and CHD. The Multiple Risk Factor Intervention Trial (MRFIT; Shekelle et al., 1985) was a large intervention trial study of 12,700 men who were CHD-free at entry into the study. Type A behavior was unrelated to seven year incidence of CHD, regardless of whether Type A was assessed by the SI or JAS. Also, as mentioned above, in a 20 year follow-up of the WCGS, there were no significant differences in the number of heart disease deaths between Type A and Type B males (Ragland & Brand, 1988). The Aspirin Myocardial Infarction Study (Shekelle, Gale, & Norusis, 1985) was another prospective study in which JAS classified Type A behavior did not independently predict heart disease.

Unfortunately, prospective studies are costly and difficult to conduct. Thus, prospective studies with coronary endpoints by far constitute the minority of research on the Type A pattern. The bulk of the literature on Type A behavior and coronary disease consists of cross-sectional studies that measure intermediate coronary outcomes. The prevailing theoretical model for the causal relationship between Type A behavior and heart disease -- the physiological reactivity model -- has dictated the choice of intermediate endpoints.

Intermediate cardiovascular and coronary endpoints

According to the physiological reactivity model, the Type A person is physiologically reactive to psychological provocations that are potentially frustrating or challenging. This reactivity results in chronic stimulation of the sympathetic nervous system. Chronic elevations of sympathetic activity, in turn, lead to deterioration of the cardiovascular system, by damaging the inner layer of the coronary arteries (c.f., Ivancevich & Matteson, 1988; Krantz & Durel, 1983). Researchers have examined the relationship between Type A status and measures of cardiovascular reactivity (CVR) and neuroendocrine changes, which can support of the sympathetic activation portion of the model, as well as measures of coronary artery disease incidence and progression, the underlying disease process that leads to CHD.



Cardiovascular reactivity outcomes . The psychophysiological study of Type A and Type B individuals in a variety of stressful situations can inform the reactivity model. Several researchers have shown that the Type A behavior pattern is related to autonomic arousal in the context of the SI. Type As and Bs typically do not differ in baseline levels of heart rate, blood pressure, or catecholamines; rather the differences emerge in terms of responsiveness to certain challenging or annoying situations (Krantz & Manuck, 1984; Matthews & Haynes, 1986). Type As show greater blood pressure increases (Dembroski, MacDougall, & Lushene, 1979; Krantz, Schaeffer, Davia, Dembroski, MacDougall, & Shaffer, 1981; Lake, Suarez, Schneiderman, & Tocci, 1985), and faster pulse transit times (Baker, Hastings, and Hart, 1984) during the SI than Type Bs. Similar patterns have emerged in response to moderately stressful tasks. Types A's show greater cardiovascular responsiveness than Type B's, in terms of blood pressure reactivity in particular (Baker et al., 1984; Dembroski, MacDougall, Shields, Petitto, & Lushene, 1978; Ward et al., 1986). Studies in which A/B status was classified in terms of the SI have shown more consistent cardiovascular reactivity differences than those classified by self-report instruments (Matthews, 1982).

In their critical review of the research on psychophysiological reactivity and cardiovascular disease, Krantz and Manuck (1984) stated that although there is much evidence that cardiovascular reactivity is linked to heart disease, it is still not clear whether those people who show increased cardiovascular responsiveness to stressors are more likely to develop coronary heart disease. There is a need for prospective studies that measure reactivity and heart disease in the same group of people, preferably starting with a healthy sample. Krantz and his colleagues (1991) have shown recently that mental stress induced reactivity is associated with the occurrence of myocardial ischemia (a clinical manifestation of coronary artery disease) in coronary patients. This finding is supportive of the reactivity model for the development of heart disease.

Neuroendocrine support for the reactivity model. Endocrinological studies have provided support for the psychophysiological reactivity model of coronary disease. Friedman, Byers, Diamant, and Rosenman (1975) found increased norepinephrine but not epinephrine during stressful tasks in Type As. Williams et al. (1982) measured cardiovascular responses and neuroendocrine changes in health male college students who participated in mental arithmetic and reaction time tasks. Type A's showed greater forearm vascular resistance (which is indicative of greater active muscle vasodilation) and greater increases in norepinephrine, epinephrine, and cortisol than Type Bs during the mental arithmetic. Type As and Bs did not differ in blood pressure or heart rate reactivity, nor in testosterone or prolactin levels. During the reaction time task, Type As showed elevations in testosterone levels. Recently, Williams and his colleagues (1991) found elevations in both epinephrine and norepinephrine in middle-aged Type A men in measurements taken on two separate days. These plasma differences were consistent with differences in urinary excretion of both of these hormones on a third day of the study. Thus, cardiovascular and neuroendocrine evidence supports the psychophysiological reactivity model, although much further prospective research is still

needed, especially studies that measure coronary disease endpoints in people for whom reactivity to stress has already been measured.

Coronary artery disease outcomes

The physiological reactivity model implies that chronic sympathetic reactivity to stress or challenge ultimately takes its toll on the cardiovascular system itself, especially arterial walls. Thus, we would expect a positive relationship between Type A characteristics and coronary artery disease (CAD). There have been several cross-sectional, angiographic studies of coronary patients that have sought to document a relationship between Type A and CAD. Several studies offer evidence of a positive relationship between angiographically determined coronary artery occlusion and Type A characteristics (Blumenthal, Williams, Kong, Schanberg, & Thompson, 1978; Frank, Heller, Kornfeld, Sporn, & Weiss, 1978; Williams et al., 1980; Zyzanski, Jenkins, Ryan, Flessas, & Everist, 1976). The nature of this relationship may differ for different age groups. Williams et al. (1988) found that the Type A pattern (as measured by the SI), was significantly associated with angiographically measured coronary occlusion, after controlling for traditional risk factors such as gender, age, hyperlipidemia, smoking, and hypertension. This positive relationship was most pronounced for patients under age 45, however. Type A and CAD were unrelated for patients between the ages of 46-64 and inversely related for patients over age 55. Williams et al. (1988) attributed the null to inverse relationship between Type A and coronary artery disease among older patients to survival effects. They argued that Type A may be involved in the "...*premature* development of heart atherosclerosis" (p. 148, italics theirs).

Shortcomings of the findings on the Type A pattern and coronary disease

Cumulatively, the prospective studies on Type A as well as the cross-sectional psychophysiological and angiographic studies all point to a mechanism by which a

coronary-prone behavior pattern may lead to heart disease. What is missing, however, is research that tracks all of these phenomena in the same individuals over time. Many studies have found support for a relationship between the Type A behavior pattern and cardiovascular reactivity, coronary artery disease, and coronary heart disease, but other studies have produced negative findings. Of most concern, of course, are the large prospective studies in which Type A failed to predict heart disease incidence (i. e., The MRFIT study, Shekelle et al., 1985) and mortality (the 22 year follow-up on the WCGS study; Ragland & Brand, 1988). Some studies have found no evidence of an association between Type A and coronary artery disease (Dimsdale, Hackett, Black & Hutter, 1978; Dimsdale, Hackett, & Hutter, 1979; Krantz et al., 1981; Scherwitz et al., 1983). Although the findings on Type A and cardiovascular reactivity have been fairly consistent, there have been reports of null findings in this domain as well. Evans and Moran (1987) observed positive relationships between Type A and cardiovascular reactivity for women but not for men, although the use of the Framingham Type A Scale for classification may have contributed to these results.[2] Matthews and Haynes (1986) noted that although the findings on cardiovascular reactivity and the Type A pattern have been quite consistent for male subjects, there have been some inconsistent findings women.

In an effort to understand the inconsistent findings on the Type A behavior pattern and coronary outcome measures, recent research has focused on breaking the Type A construct down into its constituent parts; namely, impatience, hostility, competitiveness, and time-urgency. Matthews et al. (1977) conducted a componential re-analysis of the WCGS data. They factor analyzed the ratings on various interview items, and found that two factors predicted heart disease in the sample: one that they labeled Competitive Drive, and the other that they labeled Impatience. The three items on the Competitive Drive factor that were significantly related to heart disease outcomes were "vigor, drive,

[2] Type A classification using this scale, however, was successful in predicting CHD *incidence* in the Framingham Heart Study, as previously mentioned.

and hostility," while the only item from the Impatience factor that was a significant predictor of heart disease reflected "irritability at being forced to wait in line" (p. 496). Matthews' results provoked researchers to focus not only on Type A components rather than the global pattern, but on the hostility component in particular. Several studies have appeared in the past 10 to 15 years, including some major prospective studies, which indicate that it is the hostility component -- not the global pattern -- that is most reliably predictive of coronary heart disease incidence and mortality (Barefoot, Dahlstrom, & Williams, 1983; Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985; MacDougall, Dembroski, Dimsdale, & Hackett, 1985; Shekelle, Gale, Ostfeld, & Paul, 1983; for review see Dembroski & Costa, 1987).

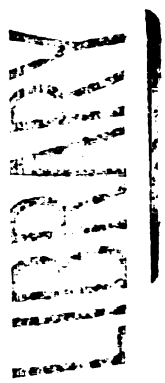
The recent emphasis on hostility in coronary disease forms the foundation of my interest in emotion and coronary outcomes. Thus, I now turn to an elaboration of the construct of hostility, the means by which it has been measured in coronary health research, and a review of the findings on hostility and cardiovascular disease prior to presenting the dissertation studies.

Hostility

There are several interpretations of the construct of hostility. For the most part, conceptualizations of hostility in the coronary health arena have been framed by the tools that people have used to assess the characteristic -- especially by those that have shown predictive relationships with coronary events. Only recently have researchers in behavioral medicine attempted to define the construct of hostility, or at least to understand what is measured by each of the varying hostility assessment techniques. In the coronary health literature, researchers have stressed that hostility is a complex trait, comprising cognitive, behavioral, and affective components (Barefoot, 1992; Contrada & Jussim, 1992; Smith, Sanders, & Alexander, 1990; Williams, Barefoot, & Shekelle, 1985). According to Barefoot (1992), the cognitive component reflects negative

attitudes and beliefs about others as well as hostile attributions; the affective component may comprise "[s]everal emotional states, including anger, resentment, contempt, and disgust, and annoyance" (p. 14); and the behavioral component consists of aggressive behavior towards others (more reactive aggression than proactive aggression).

Although Barefoot offers a more refined description of the affective aspect of hostility than has appeared elsewhere in the behavioral medicine literature, to the eyes of an emotion researcher his definition is somewhat confused. Anger, contempt, and disgust are discrete emotions that have consistently recognizable facial signals and proscribed antecedent events. Annoyance and resentment are of a different status. Annoyance is a variant of anger, and resentment may be more complex, involving the emotions of anger, contempt, and disgust as well as beliefs.



Emotion and personality theorists have discussed hostility in a different context, one that is not guided by the measurement procedures used in behavioral medicine. As such, their perspective might inform research and thinking on the role of hostility in heart disease by not showing a bias towards the instruments that have been so predictive in coronary research. Not surprisingly, emotion theorists have focused primarily on the affective aspects of hostility. Hostility has been described as an affective trait that is characterized primarily by anger, although theorists differ on whether or not hostility may involve other negative emotions. As previously mentioned, Ekman (1984) describes hostility as an emotional trait that is marked by the frequency and ease of occurrence of anger: "A hostile person becomes angry about matters that do not usually elicit anger in others, and when a hostile person is angry, the anger expression and the social consequences of the anger are likely to be more severe than is so when a non-hostile person is angry. Hostile characters may also be less able to dampen anger expressions, and they may have a longer recovery time. Hostile characters are know to others by their anger, it is what is salient about them, central to the organization of the personality" (p.33).

It is possible that hostility may involve other negative emotions as well. Izard (1977) has suggested that "the emotions of anger, disgust, and contempt interact in hostility" (p. 95). Izard describes different types of hostility, based on the differential contribution of each of the emotions in this triad. In his view, hostility in its most aggressive manifestation is more characterized by anger than the other two emotions. Hostility marked by the prominence of disgust or contempt, are characterized, respectively, by the tendency to avoid or shun another (disgust) or to hostile acts of prejudice (contempt) (Izard, p. 95, 1977). Spielberger, who has contributed to research on emotions in personality as well as in cardiovascular health, argues that while hostility usually involves angry feelings "... this concept has the connotation of a complex set of attitudes that motivate aggressive behaviors directed toward destroying objects or injuring other people" (Spielberger, Jacobs, Russell, & Crane, 1983, as cited in Spielberger et al., 1985, p. 7). The concept of anger is central to most definitions of hostility, but views on what else is involved clearly varies between theorists.

Consideration of the ways in which specific emotions are related to hostility is relevant to the present research for two reasons. First, the fact that many studies have found a relationship between hostility and coronary outcomes justifies the investigation of how specific negative affects are related to coronary events (Study 1). Second, the conceptual ambiguity in behavioral medicine as to what hostility is serves as the rationale for Study 2, an investigation of the relationship between measures of actual emotional behavior and a widely used measure of hostility -- the Cook-Medley Hostility Scale (Cook & Medley, 1954).

In the sections below, I review the methods for hostility assessment that have been used most often in research on coronary disease and discuss the findings of research using these techniques. For the purposes of the review, I use the term "hostility" to refer to the general construct of a complex affective trait characterized primarily by anger (but may involve other negative emotions, thoughts, and behavioral tendencies). Given

the extent to which the meaning of "hostility" varies depending on the way in which it was measured, I take care to specify the type of assessment procedure on which hostility was based for each study.

Assessment of Hostility

There are two approaches to hostility measurement in the research on coronary disease: interview methods and inventory techniques.

Interview methodologies. Early criticism of Type A's predictive ability focused on the fact that the Type A ratings were quite global. On the basis of the client's gestures, voice, and speech exhibited during the Structured Interview, interviewers used a 5-point scale to classify whether someone was Type A or B (Feuerstein, Labbe, & Kuczmierczyk, 1986). Newer interview rating techniques focus specifically on the Type A component of hostility. All of the techniques described below involve trained judges' ratings made from audio tapes of the Structured Interview. Voice stylistics and verbal content are the primary bases for the ratings.

The Component Scoring System (CSS; Chesney, Hecker, & Black, 1989; Hecker, Chesney, Black, & Frautschi, 1988) rates 14 components of the Type A behavior pattern (including hostility) on a question-by-question basis. Although this is generally a Type A assessment technique, it yields a score on a hostility component that can be independently related to various outcome measures, as well as anger-relevant components on the expression or suppression of anger (anger-out and anger-in, respectively). Each component is operationally defined in order to minimize subjectivity of judgments (Chesney et al., 1989). The CSS assessment approach served as the model or later interview based assessment of hostility, such as those described below (Barefoot, 1992).

Although the CSS is a great improvement over traditional SI classification and its hostility component can independently predict coronary heart disease (Chesney et al.,

1989), it possesses some limitations. The CSS may be a componential system in terms of Type A, but it rates hostility on a single dimension only. Thus, there may be a considerable amount of information consolidated into one category. Also, as an audio-only technique, it necessarily omits an important source of affective information -- facial behavior. There is evidence that facial behaviors can discriminate between Type A and Type B males (Chesney et al., 1990). Ratings based on audio recordings can only make use of data derived from vocalizations and speech, yet much can occur on the face in the absence of both of these events. Given that facial expressions are a rich source of information about experienced emotions (Ekman, Friesen, & Ellsworth, 1972; Ekman, Friesen, & Ancoli, 1980; Rosenberg & Ekman, in press), audio-only approaches are limited.[3]

Dembroski's Hostility Facet Scoring System (HFSS; Dembroski, MacDougall, Costa, & Grandits, 1989) provides separate ratings on the content, style, and intensity of hostility or the *potential for hostility*. Ratings are based on the entire interview; that is, the judge does not score in increments or on a question-by-question basis as in the CSS. The major limitation of the HFSS approach is that potentially large amounts of behavior contribute to the judgments. The judges must keep the entire interview in mind, thus creating an opportunity for memory biases to influence ratings. Even though the HFSS yields component scores on style, content, and intensity, the categories of behavior are still so broad that each one might be quite heterogeneous.

Barefoot's Interpersonal Hostility Assessment Technique (IHAT: Barefoot, 1992) is one of the most recently developed hostility rating schemes. The IHAT provides the most extensive hostility description of any of the rating techniques available, by scoring hostile behavior from each interview question in terms on four categories: evasiveness,

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[3] These criticisms about audio ratings apply to all of the hostility rating schemes described in this section.

direct challenge, indirect challenge, and irritation. This allows for a more detailed description of hostility, which should help clarify which aspects of the construct are related to health outcomes.

The interview assessment methods are commendable in that they assess actual behavior, but these methods are limited. First, they break down hostility into only a few broad categories. Several different types of emotions and behaviors may contribute to raters' judgments. Second, most interview assessments are based on audio recordings, thus omitting potentially informative affective behavior from the face.

Inventory methodologies. A good portion of the research on hostility and coronary health involves no behavioral study, but instead examines the relationship between people's scores on hostility inventories and coronary disease measures. Most of the longitudinal studies that have found relationships among psychological factors and heart disease took advantage of early assessments of large groups of people using the MMPI. A subscale of the MMPI -- The Cook-Medley Hostility Scale (Ho; Cook & Medley, 1954) -- has been found to predict heart disease outcome variables. Thus, much of the recent research has favored using the Cook-Medley: in part because of its moderate predictive success in longitudinal research, and in part because inventories are easier to use than interviews and require considerably less time to score. Also, inventory procedures may be less subjective than interviewer ratings.

Several psychometric concerns have been raised about the measurement of hostility with the Cook-Medley inventory. Although it shows good internal consistency and moderate test-retest reliability and convergent validity, there are questions regarding its construct validity (Barefoot, 1992; Smith, 1992). This might be due to the fact that the Cook-Medley was originally developed to identify teachers who had difficulty getting along with their students, not for the measurement of hostility, *per se* (Barefoot, 1992; Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989).

Other researchers (e. g., Siegman et al., 1987) have used the Buss-Durkee Hostility Inventory (BDHI; Buss & Durkee, 1957). This scale may be a more sensitive index of self-reported aggressive behavior than the Cook-Medley, and it more directly measures such affective components of hostility as irritation and resentment (Barefoot, 1992).

Inventory methods are convenient, but as self-report measures they are plagued by self-presentation biases. There is evidence that people sometimes underreport hostility on the Cook-Medley (c.f., Barefoot, 1992 for discussion). Barefoot (1992) reports that it has been difficult to replicate correlations between inventory measured hostility (from a variety of inventories) and CAD severity. This suggests inconsistency in how people respond to these scales, be it for reasons of self-presentation bias, question difficulty, or both.

Inventory methods for measuring hostility purportedly measure both expressive and experiential aspects of the hostility (Barefoot, et al., 1989; Dembroski & Costa, 1987; Smith, 1992). Smith (1992) says that expressive hostility items measure assaultiveness and verbal aggression, whereas experiential hostility items involves resentment and suspicion. Describing questionnaire assessed hostility in terms of both expressive and experiential aspects is misleading, however. It would be more accurate to refer to the "expressive" items as indicative of "verbal aggression" rather than aggressiveness and assaultiveness in general, because no one has shown whether these items relate to non-verbal expressions of anger or other assaultive behaviors. The term "experiential" is may be appropriate because some hostility scale items have been related to self-reports of emotion (c.f., Blumenthal, Barefoot, Burg, & Williams. 1987), but not enough research has been done to discriminate which *types* of emotions are most captured by scales scores or particular items.

However one parses hostility from inventory assessment, clearly some inventories are better at measuring one aspect of hostility than the other. The Buss-Durkee Hostility Inventory appears measure primarily "assaultiveness and verbal aggression" (Smith,

1992). Cook-Medley scores are correlated with self-reports of anger (Blumenthal et al., 1987; Smith & Frohm, 1985). The Cook-Medley, however, has not correlated well with some behavioral hostility measures (Dembroski, et al., 1985).

Evidence for a Relationship between Hostility and Coronary Heart Disease

Several studies have found an association between hostility and coronary health (e. g., Dembroski, et al., 1985; Williams, et al., 1980). In a cross-sectional study, Williams et al. (1980) studied approximately 400 patients who were undergoing coronary arteriography. Type A classification and Cook-Medley Ho scores were related independently to coronary artery disease, but Ho scores showed a stronger relationship to the degree of atherosclerosis. Barefoot, Dahlstrohm, and Williams (1983) examined the relationship between Cook-Medley Hostility scores and coronary heart disease fatalities in a sample of University of North Carolina Medical School graduates, who had completed the MMPI and were followed-up over several years. Across the 20 year period, the percentage of low hostile men who survived was relatively steady, but there was a sharp decrease in the proportion of high hostile men who survived.

Hecker, Chesney, and Frautschi (1988) used the Component Scoring System for the Structured Interview in a study of the WCGS men. In univariate analyses, hostility, speech rate, immediateness, competitiveness, and Type A "content" were all significantly related to CHD incidence, but only hostility remained a significant risk factor when all 12 CSS components were in the model. Shekelle, Gale, Ostfeld, and Paul (1983) found higher 20 year heart disease mortality in men with high Ho scores than in those with low Ho scores. Barefoot et al.'s (1989) study of lawyers revealed that high Ho scores were related to poorer survival rates. Dembroski and his colleagues (1989) reanalyzed the data of the MRFIT study -- a major prospective study that did not yield significant predictive effects for Type A on heart disease -- with a special emphasis on SI components of hostility experience, intensity, and expression (measured by a

version of the HFSS). Expression of hostility was positively related to heart disease incidence.

The physiological reactivity model has also been adopted in linking hostility to coronary heart disease. Subsequently, research has focused on measuring hostility in relation to the various cardiovascular endpoints of cardiovascular reactivity and coronary artery disease, as well as coronary heart disease.

Cardiovascular reactivity outcomes

Research on hostility and cardiovascular reactivity in healthy adults (usually males) has yielded much the same pattern of results as that on Type A and cardiovascular reactivity. Hostility, rated by the Cook-Medley, Buss-Durkee Hostility Inventory, and interview techniques is related to increases in blood pressure reactivity during stressful social interaction (Christensen & Smith, 1993), increases in cardiovascular reactivity during a word identification task combined with anger provocation (Suarez & Williams, 1989), and increases in systolic blood pressure, epinephrine, and norepinephrine across a variety of laboratory stressors (Lundberg, Hedman, Melin, & Frankenhaeuser, 1989). Siegman and his colleagues (1992) recently showed that Buss-Durkee measured expression of hostility was positively correlated with blood pressure reactivity in healthy males.

Coronary artery disease outcomes

Ever since Williams et al. (1980) showed that both Type A behavior and Ho scores were independently associated with atherosclerosis (with Ho scores as the stronger predictor), there has been an enormous amount of research on hostility and coronary artery disease. The emphasis in this area has been on cross-sectional, angiographic studies. These have far outnumbered the studies on hostility and cardiovascular reactivity. Several studies conducted by Dembroski, MacDougall, and their colleagues

have shown that hostility is associated with coronary artery disease. Potential for Hostility and "anger-in" scores from HFSS scoring of the SI were significant and independent predictors of coronary disease severity in a sample of 125 male patients in Massachusetts, while global Type A was unrelated to coronary artery disease in the same sample (MacDougall, Dembroski, Dimsdale, & Hackett, 1985). In a separate sample of 131 men and women in North Carolina, Dembroski and his colleagues (1985) found that the anger-in dimension of the potential for hostility ratings of the SI were related to an increase in coronary artery disease. The inventory approach has yielded significant associations as well. Siegman, Dembroski, and Ringel (1987) used the Buss-Durkee in a cross-sectional study of male and female coronary patients in Maryland. The Buss-Durkee yields scores on neurotic and non-neurotic hostility. For patients up to age 60, the researchers found an inverse relationship between hostility and severity of coronary artery disease, but for non-neurotic hostility, they found a positive relationship. Joesoef, Wetterhall, DeStefano, Stroup, and Fronek (1989) found a relationship between Cook-Medley Ho scores and *peripheral* artery disease in a large scale, cross-sectional study of male, US Army veterans.[4]

The hostility research has had its null findings with which to contend as well. Kamarck, Manuck, and Jennings (1990) found that Ho scores were unrelated to blood pressure responses during SI. Some studies have reported null findings on the relationship between Cook-Medley Ho scores and coronary artery disease. These include cross-sectional studies of angiographic patients (Dembroski, MacDougall, Williams, Haney & Blumenthal, 1985; Helmer, Ragland, & Syme, 1991). Also, as with some of the Type A findings, the relationship between hostility and coronary artery disease may be age dependent (Siegman et al., 1987; Williams et al., 1980).

Some studies have found no relationship between hostility and coronary heart disease outcomes. Hearn, Murray, & Luepker, (1989) showed that Ho measured at age

[4] Subjects were defined as having peripheral artery disease if they had a resting index (ratio of the ankle to brachial systolic blood pressure) < 0.9, an absent posterior tibial waveform, or a femoral bruit.

19 did not predict heart disease mortality or morbidity. Similar null findings with the Ho have been reported by others (Leon, Finn, & Bailey, 1987; McCranie, Watkins, Brandsma, & Sisson, 1987). Some researchers have attributed the inconsistent findings between hostility and cardiovascular health to problems in the conceptualization and measurement of hostility, and have called for clarification of the construct (Barefoot, 1992; Smith, 1992).

The Present Research

There are many shortcomings to the extant body of research on Type A behavior, hostility, and heart disease. This dissertation focuses on three specific shortcomings within the context of two studies: 1) a need for the measurement of specific emotions and emotional behavior during the SI; 2) a need for a clinically-relevant coronary outcome measure that is sensitive to changes in affective state; and 3) a need to conceptually refine our understanding of an existing, widely-used measurement tool -- the Cook-Medley Hostility Scale.

The progressive narrowing of the concepts from Type A to its components, and the subsequent focus on hostility, has sharpened the understanding of how psycho-behavioral factors may lead to heart disease. The emphasis on hostility has provoked claims that anger, specifically responding to stress or challenge with anger, is dangerous to coronary health (Chesney & Rosenman, 1985). There are at least two reasons why this inference is premature. First, as explained earlier, hostility is a dispositional construct that may be marked by the ease and occurrence of anger (Ekman, 1984), but this construct is not equivalent to the emotion of anger. Second, while research has shown that spontaneous instances of anger cause physiological effects consistent with the psychophysiological reactivity, such as heart rate and blood pressure increases from baseline (Levenson, Ekman, & Friesen, 1990, Schwartz, Weinberger, & Singer, 1981), it has not been established that these changes are substantive enough to eventually lead to

coronary artery disease. What we need is high-fidelity measurement of spontaneous emotion, and further, the examination of the relationships of measured emotion to clinically significant coronary outcomes. I take such an approach in Study 1 of this dissertation.

The direct study of emotion can also illuminate our understanding of the construct of hostility as measured by the Cook-Medley, so that we might gain a better understanding of what is being measured by an instrument that has shown predictive relationships with coronary artery disease severity, coronary heart disease incidence, and coronary heart disease death in prospective research (e. g., Barefoot, Dahlstrom, & Williams, 1983; Williams et al., 1980). Study 2 examines how emotions are related to Cook-Medley scores, and it will be described after the rationale and hypotheses for Study 1.

Bringing the conceptualization and measurement of emotion into the study of coronary -prone behavior

The first componential studies on Type A helped clarify which aspect of the behavior pattern was most relevant to coronary health, namely hostility. The hostility findings point to a role for specific negative emotions in the etiology of heart disease. Thus, the task at hand is to either breakdown the construct of hostility, or to study directly emotional responses that lead to cardiac change. If one takes the view that the hostility findings might not be an endpoint but rather indicative of a more fundamental link between emotion and the cardiovascular system, and that the fact that a hostile disposition predicts heart disease incidence is merely indicative of the cumulative effects of repeated anger (and/or other negative emotion) arousal, then the decision to study emotion directly is obvious. There are many potential paths by which emotion could cause heart disease, as depicted in Figure 1.

Researchers in health psychology rarely talk about the contributions of specific emotions to heart disease, although many have claimed that anger is toxic to cardiac

health because of the hostility findings (Chesney & Rosenman, 1985; Spielberger et al., 1985). Advances in emotion research, however, make it possible to measure emotions from various sources during an interview or other potentially stressful contexts. Thus, the techniques are available for a thorough investigation of the role of specific emotions in coronary health.

An argument for measuring emotions from facial expressions. If one is interested in the relationship between emotion and coronary outcome measures there are several potential sources of affective information from the Structured Interview context (which is the context studied in the present research) from which one could draw. There is the verbal content of the patient's responses to the questions, vocal style and intonation characteristics from speech, facial behavior, and reports or ratings of experience that can be obtained prior to, during, or after the interview. I have chosen to focus on facial behavior, with supplemental emotion information from subjective ratings.

The face offers information on type, intensity, and timing of specific emotional events. Although speech content can offer similar information, there are some drawbacks to obtaining emotional information from speech that do not plague facial measures. First, the verbal content of speech is limited by the fact that people might censor their answers to questions (Ekman, 1985), social desirability might influence their choice of words, and even if people choose to speak about what they feel, they might not be able to adequately convey the subjective experience through words. The Structured Interview context is especially limiting, in that the it contains very few questions that call for open discourse on the interviewee's present emotional state. Thus, there would not be much text to study.

Speech also offers non-verbal vocal content, from which intonation changes can reveal information about affective state (c. f., Scherer, 1989 for a review). The vocal channel is useful, because of the three primary channels through which emotion can be communicated -- verbal, vocal, and facial -- the vocal channel is the one that people are

probably least able to control (Ekman, 1985; Ekman & Friesen, 1969). The face is next most difficult to control, followed by speech content.

However, the vocal channel is limited. One cannot obtain information about as many different emotions from the voice as one can from the face (Scherer, 1981). Additionally, vocal changes are not always accessible. Verbal and vocal emotional information from speech can only be studied when speech occurs. Thus, it is impossible to learn about the emotional state of a person during his or her silent periods. Facial behavior, on the other hand, can be recorded (or measured) continuously, even when the subject is silent.

One strength of facial measurement is that facial behavior may reveal both conscious and non-conscious information about emotion. Facial behavior may reveal emotions that people may be neither *willing* nor *able* to disclose verbally. People do not always report experiencing the emotions that they show on their faces, although measures of experience and expression cohere in time at non-chance levels (Rosenberg & Ekman, in press). This advantage may be shared by vocal intonation as well.

Considering that facial behavior is continuously available, provides information about specific categories of emotion, and is moderately difficult to censor, I chose facial expression as my primary measure of emotion. There are several means by which to measure facial behavior, which are discussed in detail below.

Options for facial measurement. One method for measuring facial behavior is electromyography (EMG), which involves the actual recording of muscular activity via electrodes placed on the skin of the face (needle electrodes inserted into facial muscles can be used, but rarely are). Facial EMG is excellent for detecting subtle muscular movements that are not visible to the naked eye (Fridlund, Ekman, & Oster, 1987). Also, the temporal resolution of electrophysiological measurement far surpasses that of coding schemes that rely on the observation of behavior. Unfortunately, there are some drawbacks to the use of facial EMG. The presence of electrodes is obtrusive, and may

draw subjects' attention to the fact that their behavior is being studied. The awareness of being studied is problematic, because self-consciousness can systematically bias facial behavior (Ekman, Friesen, & Ellsworth, 1982; Kleck et al., 1976). Also, facial EMG cannot detect activity in specific muscles; it is limited to discerning movement in muscle regions, due to electrical "cross-talk" between muscular groups. Although the application of certain filters may mitigate these effects (c.f., Fridlund, Price, & Fowler, 1982), there are still spatial resolution problems with facial EMG.

There are two other approaches to facial measurement that involve human observation of videotaped facial behavior. Videotaped facial behavior can be judged by a group of observers or systematically coded by trained coders. The former technique is known as the *judgment study* (Ekman, 1972). In judgment studies, observers who have no special training make judgments (using rating scales or choosing from a list of emotion words) about which emotions they think the person in the stimulus tape is feeling. The judges are naive to the eliciting circumstances in which the facial behavior occurred.

The other technique is the *components study* (Ekman, 1972). In components studies, records of facial behavior are scored for the presence of certain facial components (actions, configurations, or symmetries). There are different types of scoring systems that take a componential approach, and these may be either theoretically derived, anatomically derived, or both. Theoretically-based systems are necessarily selective in that they account only for certain, pre-designated behaviors. Anatomically-based systems may be either selective or comprehensive. The most thorough, and least biased approach for facial coding is to use a comprehensive, anatomically-based system. This latter approach has the advantage of more spatial resolution than facial EMG, thus allowing for information about more different kinds of emotional expressions. The primary drawback of comprehensive, anatomically-based coding, is that it is labor intensive *and* that it captures only those facial events that are visually distinguishable.

For the present research, I chose to use a components system for facial measurement: The Facial Action Coding System (FACS; Ekman & Friesen, 1978). FACS is a comprehensive, anatomically-based system of measurement, which provides specific information about muscular action that can be classified in terms of at least seven different emotions. I chose the components method over the judgment method for several reasons. First, if observers' judgments of emotion show no relationship to the variables of interest (e.g., coronary measures) then one would not know if there is truly no relationship between facial behavior and coronary change or if observers were merely inaccurate in their judgments. In such cases, the face may have moved in patterns that were systematically related to variables of interest but that the observers did not perceive. Second, if judges *did* impute emotion, one would not know whether it was accurate information, because there are very few other sources of information about emotion against which facial judgments could be validated. Facial movements measured by FACS have been shown both to accurately measure facial movement, as per the accuracy criteria of performed facial movement and needle facial EMG (Ekman, 1981; Ekman & Friesen, 1982). Further, FACS coded facial expressions of emotion have been shown to be accurate indices of spontaneous emotion, in that FACS coded behavior has related meaningfully to independent criteria of emotion in other research (e.g., Ekman et al., 1980; Rosenberg & Ekman, in press). Thus, comprehensive facial measurement is the most appropriate method for the present research, in which very little other information about emotion is available from which one could determine the accuracy of judgments.

Another benefit of a comprehensive system of facial measurement such as FACS over the judgment approach is that FACS can describe all facial behavior that is visually observable -- even those patterns of behavior that might not be perceived as emotions per se. Such patterns of behavior may still show meaningful relationships with dependent variables. For example, in recent research, FACS-coded upper face components of

anger expressions -- *not* full face anger expressions --distinguished between Type A and Type B males during the SI (Chesney et al., 1990). It is not known whether observers can perceive subtle, partial expressions such as these, or whether they would include them in their judgments if they were instructed only to judge emotions.

Using facial expressions in the study of coronary-prone behavior

There is one study in the literature (alluded to above) that has systematically measured facial expression to examine the emotional component of coronary-prone behavior. Chesney, Ekman and their colleagues found that Type A males showed significantly more facial expressions of disgust and upper face components of anger than did Type B males during the Structured Interview (Chesney, Ekman, Friesen, Black, & Hecker, 1990). This upper-face anger facial configuration -- which they called the "Glare" expression -- significantly more often than Type B males during the Structured Interview. The Glare expression involves lowering the eyebrows, raising the upper eyelid and/or tensing the lower eyelid, and gazing directly at the interviewer.

Chesney and her co-authors argued that the Glare expression could actually be evidence of a trait marker of hostility, because it contains core elements of the anger expression and it correlated with the SI speech component of Hostility. It is also possible that the Glare reflects a suppressed form of anger, as it consists of the upper face but not lower face components of the anger expression. Thus, facial expressions may reveal information about affective traits as well as more transient affective states. Affective dispositions may be marked by specific configurations of their own, as well as by the presence of certain types of emotion expressions. That is, hostility may be evident both in terms of a trait marker such as the Glare expression, in terms of the incidence of emotions that characterize the trait (e. g., anger, contempt, and disgust), or both.

Contrada, Hilton, and Glass (1991) found that Type B males showed more expressions of negative affect in response to emotional stimuli than did Type As, and that Bs also showed greater systolic blood pressure increases. These results are hard to interpret, though, because the expressions were evaluated in terms of broad categories of positive and negative emotion, based on untrained observers' judgments of facial expressions of emotion using semantic differential scales. It is necessary to look within these dimensions, however, because different negative emotions may have different physiological effects (Ekman, Levenson & Friesen, 1983).

No one has looked at facially expressed emotion and actual coronary outcome measures in cardiac patients. Blumenthal and Ekman have been awarded grants to do this in the context of a large study in which they are examining emotional behavior in coronary artery disease patients as well as the efficacy of behavioral interventions in reducing episodes of transient myocardial ischemia, a clinical manifestation of coronary artery disease (the rationale for the choice of this measure is described in more detail below). They provided me with the opportunity to examine a subset of their data: facial expressions and ischemia measurements obtained during the Structured Interview prior to intervention.

My goal in Study 1 of the present research was to use facial measurement to illuminate the relationship between specific emotions and incidence of ischemia. I examined the relationship between coronary artery disease patients' facial expressions of emotion shown during the Structured Interview and whether or not they evidence transient myocardial ischemia during a specific portion of the interview. The second part of my dissertation used the emotional data derived from Study 1 in order to address an important methodological question in health psychology: What does the Cook-Medley Hostility Scale measure? There has been much discussion and some research on the multi-dimensionality of the hostility construct, especially as measured by the Cook-Medley Scale (c. f., Barefoot, 1992; Barefoot, Dodge, Peterson, Dahlstrom, &

Williams, 1989; Blumenthal et al., 1987; Hardy & Smith, 1988; Smith, 1992; Smith et al., 1990). In Study 2, I explicate the affective component of hostility as measured by the Cook-Medley on the basis of coronary patients' facial expressions of emotion during the Structured Interview.

Transient myocardial ischemia: a state sensitive, clinically relevant outcome measure.

In Study 1, I sought to determine whether momentary emotion is related to cardiovascular change -- substantive changes that are clearly meaningful to coronary health. With respect to this goal, many of the cardiovascular or coronary measures used in previous research are not ideal. Most coronary endpoint measures are either too slow to respond to brief changes in affective state (e. g., measures of the incidence or degree of coronary artery disease and coronary heart disease) or if they are dynamic (e. g., cardiovascular reactivity measures) the changes observed may not be clinically significant. The present research required a clinically relevant cardiovascular outcome that is sensitive to temporal fluctuation in emotion. A measure that meets these requirements is the incidence of transient myocardial ischemia. Transient myocardial ischemia (TMI) is a coronary artery disease manifestation, in which blood flow to the heart muscle is temporarily restricted. TMI is prevalent among patients with coronary artery disease. Although TMI is most often asymptomatic or 'silent' to the patient, it has been shown to be predictive of mortality and frequency of threatening heart disease outcomes in coronary patients (DeWood & Rozanski, 1986; Gottleib, et al., 1986; Gottleib, et al., 1988).

Previous studies of stress in laboratory situations and in daily life have shown that ischemia is linked to psychological stressors (Barry, et al., 1988; Deanfield, et al., 1983, 1984, 1985; Krantz et al., 1993; Rozanski et al., 1988; Schang & Pepine, 1977). Rozanski et al. (1988) found that various stressors provoked ischemia in CAD patients. Mental arithmetic, the Stroop color-word task, and public speaking all provoked

ischemia, but the public speaking task provoked more frequent and stronger ischemic reactions, which were asymptomatic for most of the patients. A recent study of over 3,000 Finnish men revealed that Likert ratings of hostility predicted ischemic heart disease (IHD) incidence in men with a history of IHD and hypertension (Koskenvuo et al., 1988). Other research indicates that depression may play a role in whether patients with IHD experience anginal pain or not (Light et al., 1991). Type A coronary patients are more likely to exhibit ischemia during a treadmill exercise test than Type B patients (Siegel et al., 1989). The findings from the stress research indicate that negative emotions may provoke ischemic events. The possibility that TMI may be brought on by emotional events and the fact that is a clinically significant measure for coronary patients make it a desirable endpoint for the study of the role of emotion in coronary disease.

Relating facial measures to ischemic events

A key benefit of using facial behavior as the primary index of emotion in the present research, is that it offers the opportunity to specify the types of emotions that coincided with ischemic episodes. Although the cardiac measurement procedures did not yield precise temporal data on exactly when ischemia occurred during that two minute period, thereby making it impossible to link a single emotion with the ischemic event, the comparison of facial expressions of emotion with a clinically significant cardiac measure during a small window of time can still be very informative. Emotions are transient, often lasting only a few seconds (Ekman, 1984), thus several different emotions can occur during a two minute period. The relative frequencies of different types of emotions can provide information on the types of emotional behaviors that distinguish those patients who showed ischemia from those who did not. This design, while correlational, can inform the cardiovascular reactivity model (Figure 1) by helping to specify the types of emotions that occur with (and therefore may *cause*) clinically-significant coronary change.

Basic research on emotion has shown that facial expressions can be used both to distinguish among emotion-specific psychophysiological response patterns (Ekman et al., 1983; Levenson et al., 1990) and to isolate electro-encephalographic epochs for the comparison of physiological patterns during different affective states (c.f., Davidson, Ekman, Saron, Senulis, & Friesen, 1990). This research forms the foundation for the approach of examining facial expressions in relationship to ischemic events in the present research, especially the Ekman and Levenson studies, which have employed cardiovascular measures.

Evidence for the relationship between facial expressions of emotions and cardiovascular events

There is evidence from basic research on psychophysiology of emotion that about five different emotions have unique autonomic nervous system patterns relative to each other (Ekman et al., 1983; Levenson, Ekman, & Friesen, 1990). Of the three emotions I propose as characteristic of hostility [anger, contempt, and disgust, following Izard (1971, 1977)], only anger and disgust have been well-studied psychophysiologically. Studies in which several psychophysiological variables were measured while subjects held precise facial configurations for particular emotions on their faces for 10 seconds demonstrate that anger and disgust show different cardiovascular effects relative to each other, on a within-subject basis. Within subject heart rate acceleration from baseline is larger for anger than for disgust (Ekman et al., 1983; Levenson, Ekman, & Friesen, 1990).

On the basis of all the published Ekman and Levenson research, the only cardiovascular measure that discriminates between anger and disgust is heart rate change from baseline. There is evidence, however, that mental stress-induced ischemia occurs at relatively low heart rates (Deanfield et al., 1983; 1985). Further, Krantz et al. (1991) found that while mental stress-induced ischemia was associated with significant

increases in blood pressure, ischemia was not related to heart rate reactivity. Thus, the extant knowledge on cardiovascular differentiation among anger and disgust, which is based on heart rate, need not imply that only one of these emotions could create ischemia.

I am proposing that anger, disgust, and contempt should all relate positively to ischemia for the following four reasons. First, on the basis of studies relating the Cook-Medley Hostility Scale (Ho) to the NEO Personality Inventory (Costa & McCrae, 1985) and self-reports of emotions, Barefoot (1992, Barefoot et al., 1989) has suggested that the emotions that are relevant to Ho are anger, disgust, and contempt. The Cook-Medley has been shown to be positively related to mental stress-induced ischemia (Krantz et al., 1991). By logic it follows that anger, contempt, and disgust should relate all relate positively to ischemia. Second, this argument is not inconsistent with the published findings on cardiovascular differentiation between anger and disgust, because those differences are based on heart changes only. As mentioned above, heart rate changes may not relate to ischemic changes. Third, in hostile people, it is possible that anger, contempt, and disgust will all occur during the window of time studied, even if it *is* only anger that causes ischemia (causation cannot be verified in the present research, however, because it is correlational). Thus, if hostility is related to ischemia, so should be the incidence of these three emotions. Fourth, the results of pilot research on a small sample of coronary artery disease patients support the positive relationship between ischemia and anger, contempt, and disgust expression. Ekman and Blumenthal found that those patients who showed ischemia during the Structured Interview displayed significantly more anger expressions than those who did not show ischemia. There was also a non-significant trend ($p = .10$) for ischemics to show more disgust expressions. Ischemics showed nearly twice as many contempt expressions, but the variability in contempt expressivity was so high that the differences between the means was far from significant. Nevertheless, these results suggest that the presence of anger, contempt,

and/or disgust expressions may be more common in those who show an ischemic response during the Structured Interview, and they imply that all three of these emotions could account for variability in ischemic status.

STUDY 1: CLASSIFYING ISCHEMIC STATUS ON THE BASIS OF FACIAL EXPRESSIONS OF EMOTION

In Study 1, I used measures of CAD patients' facial expressions of emotion during the Type A Structured Interview as a basis for determining whether the patients showed transient myocardial ischemia during stress. Subjects underwent the Type A Structured Interview, and measurements of the patients' facial expressions of emotion shown during the interview were coded from videotape. I then used the patterns of emotional responding obtained from the facial measures to determine whether or not the patients evidenced TMI during a 2 min interval of the interview that immediately followed the Structured Interview question about anger.

Hypotheses

Hypothesis 1. 1. It is possible to determine whether coronary patients will evidence ischemia under stress on the basis of their facial expressions of emotions.

I base this hypothesis on the idea that emotions evoke ischemia, as has been indicated by ambulatory monitoring studies of reported emotion in daily life and ischemia (Hedges, Krantz, Contrada, & Rozanski, 1990). This hypothesis also draws from previous research in emotion that shows that the face reflects changes in spontaneous emotion (Ekman, Davidson, & Friesen, 1990; Ekman et al., 1980; Rosenberg & Ekman, in press).

Hypothesis 1. 2. The number of anger, contempt, disgust, and non-enjoyment smiling facial expressions shown during the critical period of the interview would each discriminate between ischemics and non-ischemics

Hypothesis 1.2 is based on the notion that anger, contempt, and disgust are the emotions that best specify the affective component of hostility, and further, that hostility has been predictive of cardiac outcomes (as per Barefoot's 1992 suggestion). Information about the number of different negative emotions is not available from the global ratings (which combines all negative emotions into one score), but specific coding of facial emotion offers such data.

The prediction on non-enjoyment smiling is based on the fact that in a pilot study I found that ischemics exhibited more non-enjoyment smiles than non-ischemics during the anger question period of the SI. Non-enjoyment smiles lack the involvement of *orbicularis oculi*, a muscle around the eyes, which is a critical component of the enjoyment expression. Non-enjoyment smiles are not associated with self-reports of enjoyment or with the central nervous systems changes of spontaneous enjoyment (Ekman et al., 1990; Ekman & Davidson, 1993). These smiles instead appear to be social conventions, and may function to put forth a positive façade in a stressful context. One speculative explanation for the non-enjoyment smile finding in my pilot research is that ischemics (who were also higher in anger and disgust expressions) may have been trying to suppress or repress their negative affect in a stressful context by putting on a smile.

There are several specific forms of the above hypothesis, listed below, each of which refers to the direction of the predicted difference between ischemics and non-ischemics on each expressions listed in Hypothesis 1.2.

Hypothesis 1.2.1: There should be more anger expressions in ischemics than in non-ischemics.

Hypothesis 1.2.1.a. There should be more Glare (upper face anger) expressions in ischemics than non-ischemics.

Hypothesis 1.2.2.: There should be more contempt expressions in ischemics than in non-ischemics.

Hypothesis 1.2.3. There should be more disgust expressions in ischemics than in non-ischemics.

Hypothesis 1.2.4.: There should be more non-enjoyment smiles in ischemics than in non-ischemics.

STUDY 2: USING FACIAL EXPRESSIONS OF EMOTION IN CORONARY ARTERY DISEASE PATIENTS TO EXPLICATE THE AFFECTIVE COMPONENT OF COOK-MEDLEY HOSTILITY SCORES

Study 2 examined how well behavioral and subjective measures of emotion can account for variance in inventory-measured hostility. The purpose of this strategy was to specify the affective component of hostility as measured by the Cook-Medley Hostility Scale. In particular, I was interested in which emotions account for variability in Cook-Medley Hostility Scores.

What is measured by the Cook-Medley? There has been some degree of controversy over the nature of the construct of hostility as measured by the Cook-Medley Hostility Scale (Cook & Medley, 1954), the most widely used hostility inventory in behavioral medicine. Although the Cook-Medley has good internal consistency and moderate convergent validity, some questions have been raised concerning its construct validity (Barefoot, 1992; Barefoot, et al., 1989; Smith, 1992; Smith & Frohm, 1985). This may be due to the fact that the Cook-Medley was not developed originally for the measurement of hostility, but rather as an index of student-teacher rapport (Barefoot, 1992; Contrada & Jussim, 1992; Smith et al., 1990). The fact that some large studies have failed to find significant relationships between Ho scores and coronary outcomes has contributed to this controversy (Hearn et al., 1989; Helmer et al., 1991; Maruta, 1993).

More than one researcher in behavioral medicine has published a paper with the title "What does the Cook-Medley Hostility Scale Measure?" (c.f., Contrada & Jussim, 1992; Smith et al., 1990), underscoring the confusion about this scale. A few studies have yielded results that help explicate Ho. In a large scale structural analysis of Ho scores in 470 healthy undergraduates, Contrada and Jussim (1992) reported data that failed to support the idea that the full Ho scale, or a subset of it, measures "a psychometrically sound personality trait" (p. 622). This is again attributed to fact the Cook and Medley did not have hostility measurement per se in mind when developing the scale, and even though Ho scores clearly measure something important to psychological and physical health "[w]hat is at issue is the nature and number of attributes reflected in Ho scores, and the adequacy with which they are measured" (Contrada & Jussim, 1992, p. 624).

Clearly Ho *does* measure something important, something that has predictive validity in terms of coronary disease, but what is it? We cannot begin to understand the mechanisms by which psychological dispositions such as hostility play a causal role in coronary heart disease until we can be more specific about the construct. One way to illuminate our knowledge of Ho is to examine the relationship between specific affective behaviors and Ho scores.

What are the emotional components of Cook-Medley Hostility?

My focus is on describing the emotional component of "hostility" as measured by the Cook-Medley scale, because of its widespread use in coronary health research. Whether or not the Cook-Medley is a good measure of hostility, per se, is another question. Some have argued that it is not, because the Cook-Medley is not a trait aggression/anger measure (Smith, 1992). Nevertheless, Ho scores have predicted coronary outcomes and fatalities. In order to understand the mechanisms by which psycho-behavioral factors measured by the Cook-Medley may be linked to coronary

outcomes, we need to study the psychological as well as the physiological aspects of the physiological reactivity model -- namely the emotional response to stress.

Learning about hostility from emotional behavior. There is a theoretical basis in the emotion literature for the idea that emotions can tell us about emotionally-relevant traits. Several emotion theorists have suggested that emotional traits are characterized by the frequency of and threshold for the occurrence of particular emotions: depression for sadness, hostility for anger, anxiousness for fear (Ekman, 1984; Lazarus, 1991; Ortony et al., 1988). Ekman's (1984) explanation of hostility makes this clear on an operational level, by proposing that hostility is marked by the frequency and ease of anger elicitation. There are at least three reasons why it is important to examine actual emotional behavior in relation to Cook-Medley scores in particular. First, theories of how hostility is related to cardiac events are based on the physiological reactivity model, in which repeated arousal eventually lead to heart disease. It would clarify our current understanding of the mechanisms by which certain dispositions may lead to coronary disease to know which emotions are actually evoked in Ho hostile people during stressful circumstances (and subsequently related to cardiac events). Second, for purely psychometric reasons, specifying the affective component of Ho on the basis of emotional behavior would be a very important contribution to the understanding of the construct. To date, research has only investigated how Ho scores relate to other self-report instruments (c.f., Contrada, Leventhal, & O'Leary, 1990 for a review) and to grossly defined hostile behaviors (Smith et al., 1990). Third, this would be one of the first empirical attempts to test the notion espoused by some emotion theorists (e. g., Ekman, 1984) that affective traits are marked by the frequency and nature of occurrence of particular emotions.

There is strong evidence that the Cook-Medley measures a cynical form of hostility, rather than overt aggression. Contrada, Leventhal, and O' Leary (1990) summarized research that has analyzed the conceptual structure of the Cook-Medley, which support

the "cynical hostility" interpretation of Cook-Medley Ho. Smith and Frohm (1985) examined the inter-relationships between Ho scores to the Buss-Durkee Hostility Inventory, the Machiavellian scale, the Framingham Type A Scale, and measures of hardiness, neuroticism, locus of control, life events, hassles, and social support. Costa et al. (1986) correlated Ho scores with other MMPI subscales. Blumenthal et al. (1987) described four dimensions of Ho: Anger and Hostility; Neuroticism; Ineffective Coping Style; and Social Maladjustment, which were derived on the basis of how coronary artery disease patient's Ho scores correlated with state and trait measures of anger and anxiety, subscales of the MMPI, the Hopkins Symptom Checklist (Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974), self-report of Type A behavior (using the TASRI), and measures of social support. Barefoot et al. (1989) conceptually grouped Ho items on the basis of their relationships with measures of neuroticism, extroversion, openness, conscientiousness, agreeableness, and hostility and came up with five dimensions for Ho: Hostile Attribution, a tendency to interpret the behavior of others as reflecting harmful intent; Cynicism, a negative view of people in which they are seen as unworthy, deceitful, and selfish; Hostile Affect, a tendency to experience anger, impatience, and loathing in interpersonal interactions; Aggressive Responding, a tendency to express anger and engage in aggressive behavior; Social Avoidance, a tendency to refrain form social interaction. Greenglass and Julkunen (1989) examined the relationship of Ho with a measure of cynicism. Principal components analysis yielded one dimension: "Cynical distrust, an inability to trust others and a view of people as dishonest and unreliable." Although the number of conceptual dimensions by which the Cook-Medley was described varied across the aforementioned studies, each of the above studies concluded that the Cook-Medley measures a cynical form of hostility, one that is characterized by distrust, resentment, and paranoia.

It may be this emphasis on cynicism that has led others (e.g., Barefoot, 1992) to propose that contempt and disgust are relevant to Ho. It is on the basis of the evidence

that the Cook-Medley measures a cynical form of hostility, as well as Izard's (1977) definition of hostility as an anger-contempt-disgust triad, that I propose that anger, contempt, and disgust are the emotions most relevant to Ho scores. Whether or not hostility as a general construct should be conceptualized in terms of contempt and disgust as well as anger is a different matter, one that cannot be resolved empirically in the context of this research.

Hypotheses

The hypotheses concern correlational and regression analyses of hostility scores in relation to facial expressions of specific emotions during the anger question period. Secondary analyses on the patterns of behavior that are likely to characterize people with different levels of Ho scores are presented in the Results chapter.

Hypothesis 2.1. High Ho scores will be associated with more frequent expression of anger, contempt, and disgust during the anger question period of the Structured Interview.

Hypothesis 2.1 is based on the suggestion by Barefoot (1992) and others (Smith, 1992) that Ho scores reflect a type of cynical hostility that may encompass disgust and contempt, as well as anger.

Hypothesis 2.2. Ho scores will be inversely related to the frequency of enjoyment smiles.

This hypothesis was based on the somewhat obvious idea that hostile people are not likely to exhibit evidence of enjoyment during the SI, especially in response to questions about their anger.

Hypothesis 2.3. High Ho scores will be related to more frequent GLARE expressions. This hypothesis draws on the finding from Chesney et al., (1990) that Type A males showed a specific configuration of tense eyelids and lowered brows,

which may indicate a suppressed anger response or a hostile disposition. This expression (called the GLARE expression) was not typical for Type B males.

Hypothesis 2.4. High Ho will be related to more frequent occurrence of non-enjoyment smiles. I draw this hypothesis from a pilot study in which I found that ischemics exhibited more non-enjoyment smiles than non-ischemics during the anger question period of the SI. Although I did not look at Ho scores in the pilot sample, I am making the inference that hostile people may engage in similar smiling behavior.

Hypothesis 2.5. The number of anger, contempt, disgust, and Glare expressions will each account for significant variance in Ho scores. The logic for this hypothesis is consistent with each of those listed above. The main point is mathematical as well as conceptual -- not only should each of these expressions relate to Ho scores in zero-order relationships, but when all of these expressions are considered together in a regression model, each emotion should account for a unique amount of variance in Ho.

Hypothesis 2.6. Contempt and disgust will account for approximately the same amount of variability in Ho scores (they proportion of variance contributed to the model will not differ significantly between the two), but each will account for significantly more variance than anger or Glare. This hypothesis draws directly from the cynical hostility notion, and suggests that since psychometric studies indicate that Ho scores reflect cynicism more than aggressiveness, so too should facial expressions. Implicit in this hypothesis is the notion that expressions of disgust and contempt are behavioral correlates of the psychometrically inferred "cynical hostility."

Chapter 2

Methods

Design

I will explain briefly the structure of the larger collaborative project, which is a joint effort between James Blumenthal and Paul Ekman, and then more specifically the design of my dissertation. Data collection occurred at Duke University, under the supervision of Dr. Blumenthal and his colleagues. Within Blumenthal and Ekman's study, 138 coronary patients who met the inclusion criteria (described below) underwent an extensive psychological and physiological assessment period (Phase I); one of three interventions geared at mediating TMI via psycho-behavioral, informational, or exercise techniques (Phase II); and a reassessment period (Phase III), in which the measurements identical to those taken in Phase I were obtained. Random assignment to treatment condition occurred prior to Phase I assessment. This dissertation makes use of the Phase I data only, because it focuses on psychological processes in the TMI population prior to the influence of intervention.

Subjects

The dissertation sample uses data from the first 100 subjects, because this was the full *N* of the ongoing project at the point in time when the dissertation analysis were set to begin. There were 94 men and six women. The ages of the subjects ranged from 38 - 75 years ($M = 58.76$, $SD = 8.00$). Mean age did not differ between the sexes.

Recruitment. Subjects were recruited in Durham, North Carolina from local newspapers, television and radio advertisements, direct mailings to local physicians, Kaiser Permanente, and Duke University Medical Center. Potential subjects had to have documented coronary disease (defined as prior myocardial infarction or greater than

75% occlusion in at least one coronary artery) and had to show evidence of TMI in a recent exercise stress test.

The Phase I assessment period

Phase I included three days of assessment: two days of ambulatory assessment (off anti-ischemic medications) followed by one day of in-laboratory assessment. The subjects were withdrawn from anti-ischemic medication the week preceding Phase I assessment. They were first fitted with a Holter monitoring device and instructed about the ambulatory assessment procedures. After two days of ambulatory assessment subjects returned to the laboratory, had the monitoring equipment removed, and then underwent mental and exercise stress testing (described below). Laboratory ischemia measurements were obtained during each of the mental stress tasks.

Ambulatory assessment. During the ambulatory assessment period, subjects wore a 3-channel Holter monitor device for ambulatory electrocardiographic measurement. This allowed for on-line detection of ischemic episodes, indicated by depression of the ST-segment of the electrocardiogram for at least one minute duration. Subjects also competed a diary during the two day ambulatory monitoring period. Between 6am - 12am subjects were prompted by an auditory signal at an average of three times per hour (excluding sleep) to complete brief diary reports on their activities and mood.

Laboratory assessment. The laboratory assessment portion of the Phase I assessment was conducted at the Duke Medical Center Nuclear Medicine Laboratory. After the Holter monitor was removed and subjects were debriefed about ambulatory assessment, subjects underwent several laboratory mental stress procedures: mental arithmetic, a personally relevant speech stressor, a speaking task involving reading innocuous material, a mirror trace task, and the Type A Structured Interview (Rosenman, 1978). Ischemia measurements were obtained during each of the mental stressors. Each task

(except the interview) lasted three minutes, with a one minute rest period between each task.

The interview was videotaped, to allow for later analysis of facial behavior. During the interview, cardiac wall motion and left ventricular ejection fraction measures were obtained (from which transient myocardial ischemia was inferred), as were measures of heart rate and blood pressure reactivity. Immediately following the interview, subjects provided subjective ratings of their affective states, as described below.

This dissertation draws on a specific subset of the Phase I assessment measures: the facial behavior from the Type A Structured Interview (completed during the laboratory assessment period), the Cook-Medley Hostility scores (which subjects completed on Day 1, prior to ambulatory measurement), and the post-interview subjective ratings of emotion.

Instruments

The Cook-Medley Hostility Scale. The Cook-Medley Hostility Scale (Cook & Medley, 1954) is a 50-item subscale derived from the MMPI. Representative items include: "Most people are honest chiefly through fear of being caught," "Most people inwardly dislike putting themselves out to help other people," and "I commonly wonder what hidden reason another person may have for doing something nice for me." Recent psychometric research indicates that the Cook-Medley measures a cynical form of hostility (c.f., Contrada et al. 1991 for a review), one that is characterized more by distrust and resentment than by aggression. The Cook-Medley was used in the present research because it has demonstrated power in predicting the extent of coronary artery disease (e. g., Williams et al., 1980). The first chapter contains an in-depth, critical discussion of the Cook-Medley as a measure of hostility.

Type A Structured Interview. The Type A Structured Interview consists of 26 questions about an individual's typical responses to a variety of situations (Rosenman, 1978).

These questions have the ability to elicit irritation, competitiveness, and impatience. Some questions, in particular, focus on the person's ways of dealing with anger-eliciting situations, while others are administered in an intentionally provocative manner. In this sample, the entire interview lasted between 10-20 min per subject, depending on how talkative he or she was. Two alternate forms of the Structured Interview were administered to this sample in counterbalanced order, so that during the Phase II assessment period subjects would undergo a different form of the interview from the one they did during Phase I. The first chapter of this dissertation includes an extensive discussion of the strengths and weakness of this procedure for measuring Type A behavior as well as a review of the research on how the Type A construct is related to heart disease and other cardiovascular outcomes.

Subjective ratings of emotion. Immediately following the Structured Interview, subjects completed ratings of the extent which they felt anxiety, frustration, and pain. Subjects placed a mark on a 100-mm visual analog scale to indicate to the extent to which they experienced each of these states.

Ischemia measurement. Two measures of ischemia were obtained over a two minute period immediately following the anger question of the Structured Interview: cardiac wall motion and left ventricular ejection fraction. These measures were obtained using an imaging technique called radionuclide ventriculography. Red-blood cells were first labeled, and then pictures were obtained of the movement of the left ventricle via Multi-Gated Coronary Angiography (MUGA). Cardiologists determined whether the patient showed ischemia or not from the MUGA images of wall motion.

For the wall motion measurement, the image of the left ventricle was segmented into four regions. The cardiologists rated the degree of wall motion disturbance in each region on a 1 - 7 severity scale (1 = normal, 7 = severe dyskinesis). Also, an additional index of ischemia was obtained via the left ventricular ejection fraction (LVEF), which provides a gross measure of the severity of heart disease. The lower the ejection

fraction, the greater area of the myocardium that has been affected by heart disease. Subjects were classified as ischemic either on the basis of wall motion change, LVEF change, or both, as per the following criteria: 1) Subjects had to show evidence of a wall motion *change* during the two minute portion of the Structured Interview, defined as at least a 1-point deviation in the rating on the 1 - 7 scale in any of the four regions of the left ventricle. Resting akinesis would not qualify a patient as stress-induced ischemic. For example, if person demonstrated resting akinesis but it did not change during stress, then he or she was not classified as ischemic for that period; 2) If a subject's ejection fraction dropped from baseline by five percentage points or more, then he or she was classified as ischemic for that period.

Facial data

Facial behavior from the portion of the interview during which subjects responded to questions about their anger -- the two minute period of ischemia measurement -- were scored using Ekman and Friesen's (1978) Facial Action Coding System (FACS). FACS dissects all observable facial movement into 44 visually distinguishable and anatomically separate units of muscular action (action units or AUs). Every observable facial event was coded in terms of the AUs that singly or in combination with other AUs, produced it.

Intercoder agreement. Intercoder reliability for FACS coding has been well established across several laboratories (Ekman & Friesen, 1976; Ekman, Friesen, & Simons, 1985; Fox & Davidson, 1988; Krause, Steimer, Sanger-Alt, & Wagner, 1989; Steiner, 1986). There were two coders for this study, the author and another trained coder from the Human Interaction Laboratory. Both coders were blind to ischemic status of the patient, and all coding was done with the audio track turned off. The coders' reliability levels had been established against a standard criterion (Ekman and Friesen's own scoring) prior to scoring. For the coding in this sample, interrater agreement was

calculated by obtaining a ratio of the number of agreements on the AUs for each event between the coders divided by the total number of agreements and disagreements. The mean agreement ratio between coders across a third of the sample was .80 (*SD* = .07).

Preparing FACS scores for data analysis

The FACS codes of all facial events (i. e., the AU-based descriptions of each expression) from the two minute period were run through a computer program called the *FACS emotion dictionary*, which determines whether each facial event included core facial movements that characterize certain facial expressions of emotion. The program's interpretations draw on a rich empirically and theoretically-derived database of expression data from our laboratory and others', and it has been used for the classification of spontaneous facial behavior in previous studies (e, g., Ekman et al., 1990; Rosenberg & Ekman, in press).

The emotion dictionary classifies facial events into several emotion and non-prediction categories. As the dictionary is very conservative and will only interpret an event as an emotion if it is an unambiguous instance of one, there are many events that receive no prediction. If an event includes almost everything required to be considered an instance of a particular emotion, the dictionary will give it a "?" interpretation. For example, a partial expression of anger may receive an "anger?" interpretation by the dictionary. It is then up to the investigator to decide whether or not this event should be interpreted a anger, based on his or her knowledge of the context in which the expression was exhibited. For events that are too impoverished to receive a "?" interpretation, the dictionary gives a "non-prediction" interpretation. The dictionary also flags any non-predictions that contain AUs that are potentially relevant to emotion, however, so that the investigator can consider whether or not these events can be interpreted as affect-relevant.

A listing of all unique events in the non-prediction category was generated for the present sample. It was then inspected by an expert in facial interpretation (Paul Ekman) and the author. Each non-predicted event was examined for the presence of emotion relevant AUs, and some were classified into emotion categories on the basis of being partial emotion expressions. This classification was done before any information about the relationship between facial variables and any other variables for analysis (e. g., ischemia or Cook-Medley scores) had been analyzed.

The above procedures yielded several categories of emotion-relevant behaviors that could be included for analysis. The number of times each subject showed expressions in each of these categories were the primary facial data for the analyses presented in the Results chapter.

Chapter 3

Results

The results are presented separately for each study. Within each study, they are organized by hypothesis or general research question. Direct tests of hypotheses are presented, unless severely skewed distributions prevented such tests, and exploratory analyses are also presented. For reasons of both necessity and statistical prudence, I adopted certain methodological strategies that are relevant to the results of both studies. Thus, I briefly discuss my use of the ischemia variable, subject sex, and statistical strategies (regarding potential confounds and reporting of *p* values) before presenting the formal findings relevant to my hypotheses.

Treatment of the ischemia variable

As described in Chapter 2, two cardiac measures were obtained that were relevant to the incidence of transient myocardial ischemia: left ventricular ejection fraction and cardiac wall motion abnormality. It is on the basis of changes in these two measures -- as described in Chapter 2 -- that a *dichotomous* ischemia variable was obtained (provided by Dr. Blumenthal). Thus, in all my analyses, ischemia is a *grouping* variable. The frequencies of emotions as coded from facial expressions are the *dependent* variables.

Special considerations regarding subject sex

Facial and ischemia data were available for 94 subjects: 88 men and 6 women. The low representation of women in the sample can be attributed to low self and physician referrals. There were not enough women in the sample to allow for the thorough analyses of sex differences. There is evidence for gender differences in the relationship between emotion and coronary disease (Evans & Moran, 1987; Helmers et al., 1993), but there were not enough women to treat them as a separate group. Thus, I omitted the

women from the formal inferential statistics. The female subgroup was examined alone on a descriptive basis, however, and a few comments on their behavior and physiology appear at the end of the section on Study 1 results.

The results for Study 2, however, contain both male and female subjects. The rationale for including the women in the Study 2 sample is two-fold. First, Study 2 involves no cardiovascular measures, so known sex differences on those variables does not necessarily suggest sex differences on the relationship between emotional behavior and a trait measure of hostility. Second, when I conducted all the analyses with and without the women included, the results came out the same, so their inclusion in the sample does not influence the interpretation of results.

Consideration of potential confounds

Before presenting the statistical comparisons of ischemic and non-ischemic groups on the variables relevant to my hypotheses, it is important to demonstrate that the groups did not differ on potential confounds. The primary variables I had available that could potentially influence the results of subsequent analyses were subject age and total expressivity. Previous studies have reported that the relationship between hostility and ischemia varies as a function of subject age (Helmers et al., 1993). In the present sample, ischemics and non-ischemics did *not* differ in age ($M_{ischemics}$ = 59.29, sd = 7.51; $M_{non\text{-}ischemics}$ = 57.56, sd = 8.20; $t\,(86)$ = 1.05, 2-tailed p = .30), nor did age correlate with any of the key emotion variables tested in subsequent analyses.

It is also important to rule out group differences in total expressivity, because one would want to be certain that group differences in specific emotions were not a function of one group merely being more expressive than the other. Ischemics and non-ischemics did not differ in the total number of facial expressions shown throughout the critical two minute period of the interview ($M_{ischemics}$ = 55.68, sd = 19.73; $M_{non\text{-}ischemics}$ = 53.57, sd = 19.39; $t\,(86)$ = .50, 2-tailed p = .61). Neither age nor total

expressivity were significantly correlated with any of the global or specific emotion variables tested in the following analyses. I took this approach to dealing with potential confounds, rather than conducting analyses of covariance (ANCOVA) for each hypothesis, because the non-normality of the distributions of most of the variables of interest prohibited the use of standard parametric ANCOVA tests.

Considerations regarding p-values

The p-values for all results are presented according to the following convention. If I had predicted a specific direction for a group difference or correlation, then a directional or *one-tailed* p-value is presented. If I had posited no specific hypothesis on the direction of a result or decided on a test post-hoc then a *two-tailed* p-value is presented.

Several of the analyses presented in this chapter are partially exploratory; that is, follow-up analyses guided by the results of direct hypotheses tests. As such, several significance tests are presented. For the sake of thoroughness, p-values are presented for each test. However, given the number of statistical tests, a Bonferroni correction was used to control experiment-wise error (Keppel, 1982). A p-value of .03 per test will be used as indicative of a clearly significant effect. Tests with p-values between .10 and .03 should be interpreted as indicative of an effect that requires further empirical verification, but exact p-values are reported for the reader's information.

STUDY 1

The first general hypothesis for Study 1 predicted that: It is possible to determine whether coronary patients will evidence ischemia under stress on the basis of their facial expressions of emotions (Hypothesis 1.1). There are several different ways of statistically examining how two groups can be distinguished from one another on the basis of facial expressions of emotion. I present three analytic approaches from which to address this question: 1) group comparisons; 2) non-parametric classifications; and

3) discriminant analyses. I drew on the results of each set of analyses in designing subsequent analyses, and in some cases tests were exploratory. If an emotion that I had predicted would distinguish between groups did not differ in group comparisons, then it was dropped from subsequent classification analyses in the interest of controlling overall risk of Type I error. While research is an opportunity to provide support for or disprove one's hypothesis, it is also a context in which to describe nature. In service of this latter objective, I present data on the patterns observed in this sample that were not specifically predicted or anticipated. Exploratory analyses are noted as such.

Group comparisons

Hypothesis 1.1 can be conceptualized in terms of group comparisons on each of the emotions predicted to "distinguish" between ischemics and non-ischemics. For example, one can evaluate whether ischemic and non-ischemic patients differed on the emotions that were predicted to be useful in classification. The analyses below first compare the ischemic and non-ischemic patients on global emotion variables, and then on the more specific emotion variables.

Global emotion variables

In the first chapter I put forth the argument that specific types of negative and positive emotions would differ between those patients who showed ischemia during the 2 min critical period and those who did not. The predicted difference on non-enjoyment smiles between ischemics and non-ischemics may lead to group differences on "global positive emotion," because there are only two types of expression that constitute the "global positive" category: enjoyment smiles and non-enjoyment smiles. There is much more variation within the negative category. As the hypotheses on negative emotions were at the specific emotion level only, no differences in global negative were predicted across groups.

The are two major purposes for looking at whether there were differences on global emotions. First, there is the issue of specificity. One of the major reasons for using a system such as FACS for facial coding is to detect specific types of emotions that might differ between groups. FACS allows for differentiation of emotion even *within* a valence; that is, within the general category of negative or positive emotion. A global coding technique would not be sensitive to these specific differences. Thus, the group comparisons on global positive and negative emotions are offered in support for the argument of specificity. Another reason for the group comparisons on positive and negative emotion is to demonstrate that specific emotion differences within valence are not merely a function of one group showing more of the global type of emotion than the other; for example, to show that group differences on anger are not merely a function of the fact that one group showed more negative emotion overall.

Overall emotion. For each subject, the total number of emotion expressions -- regardless of type -- were determined by summing across all emotion categories (including ambiguously valenced emotions (e. g., surprise), positive/negative blends, and non-specific emotion expressions). There were no differences between ischemics and non-ischemics on total number of emotion expressions shown (t (86) = .08, 2-tail p = .94). The descriptive statistics for this global variable, as well as those for the global positive and negative emotion variables, are given in Table 1.

Table 1. Descriptive statistics on global emotion variables.

		Ischemics (n = 41)	Non-Ischemics (n = 47)
Total Emotion	mean (SD)	33.02 (13.77)	32.77 (16.03)
	median	32	29
	modes	30	24
Total Negative Emotion	mean (SD)	21.17 (11.48)	24.04 (14.07)
	median	18	22
	mode	18	16
Total Positive Emotion	mean (SD)	11.56 (6.82)	8.49 (6.61)
	median	12	6
	modes	1, 7, 12, 15, 18	5

Global Negative and Positive Emotion. For each subject, global negative and global positive emotion scores were created. For the global negative score, the number of times each subject showed each type of negative emotional expression was summed across all categories of negative emotion expression. A similar procedure was used for the positive global scores on positive emotion categories. Surprise was not included in either of these variables, nor were any positive/negative blends (i. e., expressions that contained elements of both positive and negative emotion expressions). This procedure yielded two variables: total number of negative emotion expressions and total number of positive emotion expressions.

The distributions for these summary scores were fairly normal, so parametric procedures were used to compare means between groups. The mean total number of negative emotion expressions did not differ between ischemics and non-ischemics (t (86) = -1.04, 2-tail p = .30). Ischemics showed more positive expressions than non-ischemics (t (86) = 2.14, 2-tail p = .03), which would have been predicted on the basis of the non-enjoyment smile findings from my pilot study. One interesting descriptive finding (see Table 1) is that total negative emotion expressions were much more variable than total positive emotion expressions, for both ischemics and non-ischemics. This could result from the fact that more different types of emotion expressions go into the global negative category than into the global positive category.

The next set of analyses focus on the emotions about which I had made specific predictions. In Chapter 1, I predicted that the number of anger, contempt, disgust, and non-enjoyment smile expressions shown during the critical period of the interview would each discriminate between ischemics and non-ischemics (Hypothesis 1.2).

Analyses of specific negative emotions

Each of the negative emotions from Hypothesis 1.2 occurred infrequently. As their distributions were extremely positively skewed, two different *non-parametric* techniques were employed for statistical analysis of the differences between the ischemic and non-ischemic groups. First, I used Mann-Whitney U tests to compare the mean ranks of the frequencies of each expression between the groups. Second, I dichotomized the expression variables (in terms of whether they occurred or not) and compared the proportions of subjects in each group who showed the expression via a *z*-test of the differences between population proportions (Guilford, 1954; Shott, 1990). Once again, effects reported for p-values between .10 and .03 should be interpreted as indicative of an effect, but not formally supportive of a "significant" difference.

Table 2 presents the means, medians, and modes for anger, contempt, and disgust variables for ischemic and non-ischemic patients. The comparisons between groups are presented below, by emotion. In the group comparisons (unless otherwise noted) all variables are frequencies (number of times a certain type of facial expression occurred) *or* the proportion of subjects who showed each type of emotion expression.

Table 2. Average frequencies of selected emotions for ischemics and non-ischemics.

		Ischemics (n = 41)	Non-ischemics (n = 47)
Global Anger (composite)	mean (SD)	1.81 (1.99)	1.30 (1.92)
	median	1	1
	mode	1	0
Anger without AU10	mean (SD)	1.29 (1.94)	.70 (1.42)
	median	1	0
	mode	0	0
Anger with AU10	mean (SD)	.51 (1.08)	.60 (1.47)
	median	0	0
	mode	0	0

(Table 2., continued)

		Ischemics (n = 41)	Non-ischemics (n = 47)
Anger without AU23	mean (SD)	.95 (1.75)	.60 (1.41)
	median	0	0
	mode	0	0
Anger with AU23	mean (SD)	.85 (1.35)	.70 (1.28)
	median	0	0
	mode	0	0
Glare	mean (SD)	.54 (.84)	.57 (1.10)
	median	0	0
	mode	0	0
Global Contempt (composite)	mean (SD)	2.88 (4.10)	3.40 (4.26)
	median	1	2
	mode	0	0
Contempt with U10, but no U12 or U14, with speech	mean (SD)	1.07 (2.35)	.98 (1.99)
	median	0	0
	mode	0	0
Contempt with U10, no U12 or U14, without speech	mean (SD)	.17 (.49)	.19 (.68)
	median	0	0
	mode	0	0
Contempt with U12, no U10 or U14	mean (SD)	.15 (.48)	.23 (.760)
	median	0	0
	mode	0	0
Contempt with U14, no U10 or U12	mean (SD)	.98 (2.31)	1.11 (1.72)
	median	0	0
	mode	0	0
Global Disgust (composite)	mean (SD)	8.51 (8.20)	8.34 (7.93)
	median	6	7
	mode	0	0

(Table 2., continued)

		Ischemics (n = 41)	Non-ischemics (n = 47)
Disgust	mean (SD)	.37 (1.20)	.17 (.79)
AU9	median	0	0
(disgust	mode	0	0
with AU9, but no AU10)			

Anger

The FACS emotion dictionary interprets anger events into two categories: clear instances of anger (AN) and probable instances of anger (AN?). Events interpreted as AN are those combinations of action units that are unambiguously anger, regardless of context. They contain upper and lower face elements of anger: eyelids tightened, upper eyelids raised, brow lowered, lips tightened and/or pressed together. Events interpreted as AN? may be missing one or more of these possible actions, but contain some of the core actions. The frequency of AN and AN? showed the same pattern across ischemic and non-ischemic groups. Thus, these two variables were combined to form a composite anger variable (Global Anger). This composite was the primary anger variable used in the anger analyses, although some specific variations were also examined (to be discussed shortly).

Hypothesis 1.2.1 predicted that there would be more anger expressions in ischemics than in non-ischemics. Although the overall frequency of anger expression was low, ischemics showed more anger overall than non-ischemics (Mann Whitney U = 773.5, 1-tail $p < .05$). A total of 75.6% of the ischemics showed some form of anger (as per Total Anger) and 57.5% of the non-ischemics did, which is a significant difference (z = 1.93, 1-tailed p =.03). When anger expression was examined as a proportion of total negative emotion expression, the same results were obtained.

Next, I looked at specific types of anger expressions, which were characterized by certain FACS action units (AUs). While there are several related expressions that make up the anger family (Ekman, 1992), it is possible that various expressive forms within

the family may correspond to slightly different types of anger. Full face anger expressions can be distinguished on the basis of whether they involve the raising of the upper lip (AU10) in addition to the tightening and pressing of the lips together (AUs 23 and 24, respectively), and whether they are open mouthed or a closed mouthed. I examined all variations of anger that occurred frequently enough to be analyzed as separate categories. The following types of anger were compared between groups: anger with AU10, anger without AU10, anger with AU23, and anger without AU23.

Tests of emotion variants should be considered as specific cases of the general hypothesis test. Thus, I used 1-tailed p-values for tests of all of the anger variants, given that hypothesis 1.2.1 proposed that anger should occur more frequently in ischemics. Overall, ischemics showed more anger without AU10 than did non-ischemics (Mann-Whitney U = 910, 1-tail p = .046). This form of anger occurred in 53.7% of ischemics and 36.2% of non-ischemics (z = 1.67, 1-tail p =.045). Ischemics and non-ischemics did not differ on the number of anger expressions with AU10 (with AU10, Mann-Whitney U = 961, 1-tail p = .44), or on anger with AU23 (Mann-Whitney U = 888, 1-tail p = .15). Anger expression without AU23 did differ slightly between groups (Mann-Whitney U = 817.5, 1-tail p = .08), occurring in 43.9% of the ischemics but only in 27.7% of the non-ischemics (z = 1.60, 1-tailed p = .06). Thus, although Global Anger showed a more marked difference between ischemics and non-ischemics, two particular forms of anger distinguished the ischemic subjects from non-ischemics: those with no lip raise and those with no lip tightening.

The partial anger expression involving upper face components of anger as well as staring at the interviewer, which has been shown to occur more in Type A males than Type B males (c.f., Chesney et al., 1990) did *not* differ between groups (Mann-Whitney U = 916.5, 2-tailed p = .64). Thus, there is no support for Hypothesis 1.2.1.a. Although proportionally fewer ischemics showed Glare than non-ischemics (63.41% versus 70.21%), this difference was not significant (z = .69, 2-tailed p = .49)

Contempt

Hypothesis 1.2.2 predicted that contempt expressions would occur more often in ischemics than non-ischemics. However, the data indicated that there was a tendency for the opposite to occur. All contempt effects reported below were in the opposite direction of what had been predicted. Thus, none of the contempt results should be considered supportive of my hypothesis. In the interest of understanding the role of contempt expression in ischemia, however, I conducted exploratory analyses on group differences in contempt expression. Each of these exploratory tests uses 2-tailed p-values. Non-ischemics showed more Global Contempt than ischemics ($M_{non} = 3.40$, $sd_{non} = 4.26$; $M_{ischemics} = 2.88$, $sd_{ischemics} = 4.10$), but non-parametric comparisons indicate that this is not significant (Mann Whitney $U = 795$, 2-tail $p = .15$). Global Contempt was a composite of events interpreted by the FACS dictionary definitive instances of contempt (CO) and probable instances of contempt (CO?). When examined dichotomously, simply in terms of whether there was a difference between groups in the proportion of subjects who showed evidence of any type of contempt, non-ischemics showed Global Contempt more often than ischemics ($z = 2.10$, 2-tailed $p = .036$). While 65.85% of the ischemics showed Global Contempt, 85.10% of the non-ischemics did.

There are several different possible forms for the expression of contempt: unilateral AU10 (U10), unilateral AU12 (lip corner raiser, U12), unilateral AU14 (dimpler, U14), and all combinations of these actions. The contempt characterized by U10 was examined when it occurred in the presence of speech and in the absence of speech, because this action occurred frequently in both contexts. Table 2 contains the descriptive statistics on the specific forms of contempt that occurred with enough frequency to be statistically compared between groups. Only the U14 variant (occurring alone, with no U12 or U10 present) differed systematically between groups. Non-ischemics showed more U14 contempt expressions than ischemics (Mann-Whitney $U = 783.5$, 2-tail $p =$

.08). Only 26.83% of the ischemics showed this form of contempt, whereas 46.85% of the non-ischemics did (z = 1.99, 2-tailed p = .047). Incidentally, this form of contempt is the expression most consistently recognized as contempt in cross-cultural research on facial expression of emotion (Ekman & Friesen, 1986; Ekman & Heider, 1988).

The primary rationale for the argument that contempt would be related positively to the occurrence of ischemia was based on the notion that contempt was one of three emotions relevant to hostility (Izard, 1971), and subsequently, that hostility was related to ischemia, as indicated by previous research (Krantz et al., 1991). There are two points at which this argument can be tested by the data. First, there is the question of whether contempt is related to hostility. In Study 2, I present findings that indicate that this assumption is valid. The expression of contempt was positively related to Cook-Medley Hostility (Ho) scores. The second point of examination would be to determine whether Ho scores, in turn, related to ischemia.[5] In fact, Cook-Medley Hostility scores were not related to the occurrence of ischemia during the 2 minute critical period (r = .01, 2-tail p = .92). In the presentation of the results for Study 2 I report the correlations between anger, contempt, and disgust, and their relationship to Ho scores.

Disgust

Hypothesis 1.2.3 predicted that disgust expressions would occur more often in ischemics than non-ischemics. To test this hypothesis, I examined Global Disgust [FACS dictionary definitive disgust events (DI) and probable disgust events (DI?)], as well as specific variants of the disgust expression. Tests of disgust variants should be considered as specific tests of hypothesis 1.2.3, thus 1-tailed p-values are used in the general case as well as the specific case tests. There were no differences between

[5] The relationship between Ho scores and ischemia is not the focus of this dissertation, but inspection of this relationship is critical to the logic of my argument about contempt and ischemia. The presentation of this correlation is relevant only to the contempt argument.

groups on Global Disgust, either in terms of differences between mean ranks (Mann-Whitney U = 943.5, 1-tail p = .43) or in terms of the proportion of subjects in each group who showed disgust (87.80% of the ischemics showed this emotion, while 80.85% of non-ischemics did, z = .915, 1-tail p = .18).

Disgust expressions can be distinguished primarily on the basis of whether they feature the nose wrinkle only (AU9), the upper lip raise only (AU10), or combinations of both AUs. Each of these variations of disgust occurred with enough frequency to be examined separately in this sample. The only type of disgust that differed between the groups was that based on AU 9, when it occurred in the absence of AU10. This was true for both AU9 events with speech and those without, so the two variables were combined to create a total Disgust AU9 score. Ischemics showed more AU9-based disgust than non-ischemics (Mann-Whitney U = 863.5, 1-tail p = .06), which was consistent with my hypotheses. Given then difference on the Mann-Whitney tests of Disgust with AU9, it was expected that on a purely dichotomous basis, ischemics would be more likely to show this type of disgust than non-ischemics. While 17.07% of the ischemics showed this type of disgust expression, only 6.38% of the non-ischemics did. This is a borderline effect (z = 1.61, 1-tail p = .05). Descriptive statistics for this form of disgust as well as the Global disgust variable are listed in Table 2.

Smiling behaviors: enjoyment and non-enjoyment smiles.

Hypothesis 1.2.4 predicted that non-enjoyment smiles would occur more often in ischemics than non-ischemics, based on a finding from the pilot research for this dissertation that ischemics showed many more non-enjoyment smiles than non-ischemics. In light of the fact that these smiles were shown during the context of questions about anger, the occurrence of these non-enjoyment smiles finding was interpreted preliminarily as a behavior that may reflect the management of anger expression.

"Smiling" is a rubric for a heterogeneous group of behaviors, some of which have been linked to states of experienced enjoyment, others of which appear to serve as social conventions (Ekman & Davidson, 1993; Ekman & Friesen, 1982). Smiles (defined generally as contraction of the *zygomatic major* or FACS AU12) can be distinguished on several bases: the presence or absence of the contraction of *orbicularis oculi* (FACS AU6, or cheek raiser), on the basis of intensity (strength of contraction of *zygomatic major* , AU12, lip corner raiser, and/or AU6), the temporal variation in onset and offset of the various muscle components (AU12 or AU6), and whether or not these actions occur in the presence of other affect-relevant facial actions. Smiles with AU6 occur during spontaneous enjoyment (Ekman et al., 1990), while smiles without AU6 may occur for a variety of reasons: politeness, or to feign enjoyment. In this project, I classified smiles (AU12) on the following bases: 1) intensity, and 2) whether or not they included the contraction of AU6.

Separate variables were created for each category of smiles enjoyment smiles (also known as Duchenne smiles or D-smiles, c.f. Ekman et al., 1990) and non-enjoyment (or non D-smiles). Within each of these categories, intensity was also considered as a variable. Based on the FACS five point intensity scale, four intensity categories were examined, from lowest to highest: A, B, C, and combination of intensities D and E. This scheme yielded eight different smiling variables: enjoyment or non-enjoyment at each of the four intensity levels. Inspections of the means and distributions on these variables for each group suggested that across each level of intensity, the pattern of differences between ischemics and non-ischemics within enjoyment or non-enjoyment categories were maintained. Thus, the data were collapsed across the various intensity levels within each type of smile. This yielded two smile summary scores: the total number of enjoyment or Duchenne smiles (Total D-smiles) and total number of non-enjoyment or non-Duchenne smiles (Total Non D-smiles).

The smile summary variables Total D-smiles and Total Non D-smiles were fairly normally distributed, so standard parametric tests were employed for comparisons between the groups. The descriptive statistics for these variables are listed in Table 3. Ischemics showed more of both types of smiles than non-ischemics, (Total D-smiles, t (68.09) = 2.12, 1-tail p = .038; Total Non D-smiles, t (63.73) = 2.54, 1-tail p = .007), although the D-smile effect is borderline given the Bonferroni significance criterion of .03.

The fact that ischemics showed more non-enjoyment smiles was expected, but the enjoyment smile effect had not been anticipated. However, this findings alerted me to something I had observed anecdotally while viewing the videotapes. I had noticed that in version A of the interview, many subjects laughed slightly after the anger question. This was not the case in version B. In version A the anger question was "When you get angry or upset, do people around you know it?". In version B, the anger question was "Most people have pet peeves. What things aggravate you most?" This anecdotal observation led me to believe that the amount of enjoyment smiling might systematically vary as a function of interview version. An ANOVA on Total D-smiles showed that the greater enjoyment smiling by ischemics could be attributable to interview version, however (interview X ischemia interaction $F(1, 82) = 7.94, p = .006$). Only in version A of the interview did ischemics show more enjoyment smiles than non-ischemics (version A means: ischemics: = 6.56; non-ischemics = 3.07; version B, ischemics = 4.21, non-ischemics = 4.68). The differences between groups on non-enjoyment smiling (Total Non D-smiles), however, did *not* vary by interview version (Interaction $F(1, 82) = 2.42, p = .13$).[6]

[6] This prompted me to check interview version as a potential confound in all analyses. I retested all other analyses, and found that none varied as a function of interview version.

Table 3. Descriptive statistics on smiling variables

		Ischemics (n = 41)	Non-ischemics (n = 47)
Total D-smiles	mean (SD)	5.19 (3.94)	3.66 (2.62)
	median	5	4
	modes	1, 5, 7	4
Total Non D-smiles	mean (SD)	3.54 (3.33)	2.02 (2.01)
	median	3	2
	mode	0	0

Non-parametric classifications

Non-parametric classifications were conducted for each emotion dependent variable on which there were group differences, to determine whether any of these variables separated the groups in ways that were obscured by the nomothetic analyses. These classification schemes were developed on a post-hoc basis, guided by the results of the group comparisons. This was in the interest of controlling the experiment-wise error rate in the formal discriminant analyses (presented in the next section), so as to not conduct tests that seemed unprofitable.

The classification approach was to create a series of grouping or dichotomous variables that coded whether or not each subject was high or low (defined as above or below the median) on a particular type of expression, or, given the very low frequencies on most expression variables, whether the subject exhibited that expression or not. Expression variables that showed promise of discriminating between ischemics and non-ischemics on the basis of the nomothetic analyses were coded for the classification analyses. The classification analyses are organized by emotion. Single emotion classification schemes are presented first, followed by schemes that consider more than one type of emotion.

Subjects were classified according to whether they were at or above the median of: the composite anger variable, anger without AU10, non-enjoyment smiles, the joint

response pattern of anger and being above the median on non-enjoyment smiles, and the AU14 form of contempt. For example, subjects at or above the median on Total Anger were considered "high" on anger, while those below the median were "low" on anger. As the median was 1, being at or above the median was tantamount to a "present" score in an absent-present dichotomy.

Table 4 presents the classification tables for all of the variables that showed group differences in the nomothetic analyses. The anger tables are presented first. The chi-square tests are presented for the reader's interest, although none is significant using the .03 experiment-wise alpha level. Significance testing is not the point of these classification tables, however. Instead, the function of this classification strategy was to determine which emotions in this sample best classified ischemic from non-ischemic patients on a purely descriptive basis.

In each case, the tables indicate that more of the ischemics were high on each anger variable than non-ischemics. While this is an encouraging result, and one that indicates that the group differences described above were not due to a few extreme values pulling the means part, the classifications are far from perfect. Consider taking the perspective of a hit and miss analysis. In the anger tables presented in Table 4, the "hit" cells for the anger variables are the "low on anger variable, no on ischemia" and "high on anger variable, yes on ischemia." For each of the anger variables, there are more hits than misses, but there are still many misses. For composite anger, for example, three-fourths of the ischemics showed anger expressions (one-quarter misclassified) but the spilt on hits and misses for non-ischemics was 50 - 50. The variables that had the most "hits" in the classification scheme were Global Anger, Total Non D-smiles, and the combination of *both* Global Anger and Total Non D-smiles.

Table 4. Classification of ischemic status on the basis of anger variables, anger-smile patterns, and contempt.

		ISCHEMIA		
		no	yes	
Global Anger	low	20 (42.6%)	10 (24.4%)	
	high	27 (57.4%)	31 (75.6%)	
		47	41	hits = 60% misses = 40%

X^2 (df = 1), = 3.21, p = .07

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		ISCHEMIA		
		no	yes	
Anger without AU 10	low	30 (63.8%)	19 (46.3%)	
	high	17 (36.2%)	22 (53.7%)	
		47	41	hits = 59% misses = 41%

X^2 (df = 1), = 2.71, p = .10

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		ISCHEMIA		
		no	yes	
Total Non D-smiles	low	22 (46.8%)	13 (31.7%)	
	high	25 (53.2%)	28 (68.3%)	
		47	41	hits = 57% misses = 43%

X^2 (df = 1), = 2.08, p = .15

(Table 4, continued)

		ISCHEMIA: no	ISCHEMIA: yes	
Global anger and Total Non D-smiles	low	34 (72.3%)	21 (51.2%)	
	high	13 (27.7%)	20 (48.8%)	
		47	41	hits = 61% misses = 39%

X^2 (df = 1), = 4.17, p = .054

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		ISCHEMIA: no	ISCHEMIA: yes	
Contempt (with U14 only)	no	25 (53.2%)	30 (73.2%)	
	yes	22 (46.8%)	11 (26.8%)	
		47	41	hits = 41% misses = 59%

X^2 (df = 1), = 3.73, p = .053

The non-parametric classifications and the group comparisons suggest that non-enjoyment smiles and anger expressions as the best means for discriminating between coronary patients who show ischemia and those who do not, at least by inspection of the hit-and-miss analyses. The next section presents the parametric test of this assumption, by using these two expression variables to create a discriminant function for classifying patients according to ischemic status.

Discriminant analyses to classify ischemics and non-ischemics

In Chapter 1, I had proposed that one could use facial expressions of emotion to classify whether or not people showed ischemia (Hypothesis 1.1). I had specifically hypothesized that facial expressions of anger, contempt, disgust, and non-enjoyment smiles during the anger questioning period will distinguish ischemics from non-ischemics (Hypothesis 1.2), but the group comparisons and non-parametric classification analyses did not support substantive differences between ischemics and non-ischemics on contempt and disgust. Indeed, a formal parametric test of the idea that consideration of all of these emotions would distinguish between groups was not supported by a linear discriminant analysis either, given the modified alpha criterion of .03 (classification hit-rate = 62.5%, $X^2_{(4)} = 9.39$, $p = .05$).

In the group comparisons, only two variables differed as predicted between groups: anger expression and non-enjoyment smiling. The descriptive findings from the hit-and-miss analyses also suggest that the occurrence of both of these expressions best discriminated between ischemics and non-ischemics. Thus, the frequency of anger expressions (Global Anger) and non-enjoyment smiles (Total Non D-smiles) were entered along with the ischemia variable into a linear discriminant analysis, in order to determine if these two expression variables, considered in combination, would correctly classify whether or not patients showed ischemia.[7] The single function generated with anger and non-enjoyment smiles had a hit-rate of 69.3% That is, on the basis of these two types of facial expressions, it correctly discriminated between ischemics and non-ischemics 69.3% of the time. This hit-rate is substantial, and it is statistically significant ($X^2_{(2)} = 8.26$, $p < .02$).

The discriminant results mean that the linear combination of the anger and non-enjoyment smiles variables that maximally separates the groups is 69.3% accurate in

[7] In considering the discriminant results, it is important to keep in mind that the function should be and will be tested against an independent dataset. This is an ongoing study, and there are approximately 40 additional subjects on whom this function could be validated.

identifying which subjects were ischemic and which were not. The classification based on both of these expression variables did better than either variable alone: Total Non D-smiles alone: hit-rate = 61.4%, $X^2 = 6.41$, $p = .01$; Global Anger alone: hit-rate = 55.7%, $X^2 = 1.45$, $p = .23$). How does one interpret the discriminant function in terms of how many ischemics and non-ischemics actually show the expressions used in the discriminant function?

If one examines the subjects individually, in terms of how many subjects of each group actually showed anger at least once and were above the median on non-enjoyment smiles (see Figure 2), one finds that a quarter of non-ischemics showed this pattern but half of the ischemics do. The classification based on both types of expressions may have been 69.3% accurate, but the fact that about half of the ischemics did *not* show the pattern of anger and above the median non-enjoyment smiling raises questions as to why. What characterized the ischemics who did not show this pattern? There were no differences between groups on any other emotional expressions. So it is not as if this sub-group of ischemics was showing some other type of behavior more often, whereas the other sub-group of ischemics showed anger and non-enjoyment smiles.

There are at least three possible explanations for the lack of the anger/non-enjoyment smile facial pattern in some of the ischemics: 1) they were repressing or suppressing their anger expression; 2) their ischemia was not emotionally mediated; and/or 3) their ischemia was less intense than the ischemia of those who showed the facial response pattern. The dichotomous ischemia variable did not allow for coding of variations within the ischemic group, so it is impossible to evaluate the third explanation. It is possible to investigate the first and second explanations, however, by examining other physiological variables (as will be discussed later) and by consulting other measures of emotion.

In emotion research, when results based on measures of one response system are ambiguous, it is often helpful to consult another response measure, such as physiology

or self-report, to help clarify the picture. Fortunately, there was one other source of emotion information available from the interview. At the end of the interview, subjects reported on the amount of anxiety that they felt during the entire interview using a 100 mm visual analog scale on each term. As anxiety ratings were the only negative emotion ratings available, these can only be considered as an index of negative emotional arousal, not as an index of anxiety.[8] I then used these ratings to see if the ischemics who did not show the anger/non-enjoyment smile pattern reported experiencing as much negative emotion as those who showed the pattern. Learning this would shed light on the three possible explanations mentioned above.

I conducted a simple comparison between the anxiety means of those ischemics who showed the anger/non-enjoyment smiling pattern and those who did not. The ischemics who showed this facial pattern reported more anxiety (M = 25.76 mm, sd = 23.40) than ischemics who did not show the behavior pattern (M = 19.53 mm, sd = 20.12). This difference was a borderline effect, given the .03 criterion ($F_{(1,77)} = 3.15$, $p < .05$). Nonetheless, the tendency of an anxiety difference between groups, suggested further pursuit of the role of reported "anxiety" in discriminating between groups.

Thus, I separated the entire male sample into higher and lower anxious groups on the basis of a median split -- and then re-ran the classification scheme, in order to see if the facial variables would predict ischemic status better for subjects who reported more anxiety. For the lower anxious group, classification on basis of this behavior pattern was only 57% accurate, which is not significantly better than chance ($X^2_{(2)} = .46$, $p = .80$). For the higher anxious group the hit-rate was 82%. This hit-rate was significant at less than .001 ($X^2_{(2)} = 14.28$).

These findings suggest that facial expressions of emotion are better "predictors" of ischemia in patients who report experiencing more negative emotion during the interview. For the purposes of this dissertation, I did not have available measures (e. g.,

[8] Although ratings on frustration were collected as well, very few subjects completed the frustration scales. Thus, the anxiety scales were the only negative emotion scales available.

trait measures of anxiety and social desirability as well as heart rate and blood pressure reactivity data) to determine whether the differences among the "low anxious" and "high anxious" ischemics was attributable to repressive or truly "low anxious" sub-groups of ischemics. In the next chapter I discuss how future work with this dataset (in which trait measures relevant to repression and cardiovascular reactivity data may be available) might help explain the differences among ischemics in anger and non-enjoyment smiling.

Comments on the women as a group

All of the 6 women in the sample were ischemic, but only 1 of them showed anger expressions. It is interesting that the women were all above the median on expression of non-enjoyment smiles. The data on the full sample certainly indicate that non-enjoyment smiling is related to ischemic events, and the data on the men indicate that anger and non-enjoyment smiling pattern does a better job of discriminating between ischemics and non-ischemics than anger expression alone. Although this is too small an *n* to generalize, the fact that all 6 of the women were ischemic and only 1 of them showed anger would suggest that they are even more likely than men to use social smiles in an anger context, a potential "coping strategy" that appears to have deleterious coronary consequences. The interpretation of the anger/non-enjoyment smile behavior pattern as a coping strategy, the possible differences in this behavior pattern between the sexes, and further research that could substantiate this argument will be discussed in detail in Chapter 4.

STUDY 2

Study 2 focused on the extent to which expressive data shown during the anger portion of the interview showed a meaningful relationship with Cook-Medley hostility scores. The results for Study 2 (which are based on the full sample, including the 6

women) are presented first in terms of regression analyses and analysis of variance relevant to the hypotheses, and then typological analyses are presented, which draw on the findings from the parametric tests.

The first four hypotheses for Study 2 describe the relationship between Cook-Medley hostility scores (Ho scores) and the incidence facial expressions of specific emotions during the anger question period in terms of simple correlational relationships. The second two hypotheses use more than one source of expression data to account for variability in Ho scores via a multiple regression model. In the sections below I report the results using the Global forms of anger, contempt, and disgust. I also conducted analyses using more specific variants of these expressions (as per results in Study 1 on specific forms of anger, contempt, and disgust), but the results were redundant with those on the global variables. Given this, and the fact that the global variables had more range and higher frequencies across subjects, only the findings on the global variables are reported.

The simple correlational hypotheses are repeated below:

Hypothesis 2.1. High Ho scores will be associated with more frequent expression of anger, contempt, and disgust during the anger question period of the Structured Interview.

Hypothesis 2.2. Ho scores will be inversely related to the frequency of enjoyment smiles.

Hypothesis 2.3. High Ho scores will be related to more frequent GLARE expressions.

Hypothesis 2.4. High Ho will be related to more frequent non-enjoyment smiling.

All of the above hypotheses can be evaluated in terms of zero-order correlations, whereas the unshared variances in Ho scores accounted for by each of these expressions

will be presented in the multiple regression analyses. The Pearson correlations between each of the expression variables and Ho scores are listed in Table 5.

Table 5. Zero-order correlations between selected expression variables and Cook-Medley Hostility Scores

Expression variable	*r*	*p* (1-tailed)
anger (Global Anger)	-.09	.20
contempt (Global Contempt)	.24	.009
disgust (Global Disgust)	.19	.035
Glare	-.10	.17
non-enjoyment smiles (Total Non D-smiles)	.03	.40
enjoyment smiles (Total D-smiles)	-.01	.47

As is evident from Table 5, only contempt showed a significant relationship with Ho scores, and disgust a borderline effect. Both of these correlations were in the predicted direction. These correlations provide preliminary, partial support for Hypothesis 2.1. The fact that anger did not relate to Ho scores is inconsistent with Hypothesis 2.1, however. The zero-order correlations provided no support for Hypotheses 2.2 through 2.4.

Also relevant to the correlational hypotheses and to Izard's (1971) framework that anger, contempt, and disgust are the triad of emotions relevant to hostility that serves as the basis for Hypothesis 2.1, are the correlations among these three emotions. Contempt was not significantly correlated with anger or disgust ($r_{anger,\ contempt} = .10$, 1-tailed $p = .18$; $r_{contempt,\ disgust} = -.004$, 1-tailed $p = .49$), but anger and disgust were slightly correlated ($r_{anger,\ disgust} = .19$, 1-tailed $p = .04$). Thus, these emotion expression appear to provide unique information; there is no overlap between contempt and any of the other two emotions, and there is only minimal shared variance between anger and disgust. This could account for why they each relate to Ho scores differently.

Although the simple correlational analyses between the specific emotion variables and Ho scores shed light on the hypotheses and indicate which emotions are worth considering as relevant to Ho, zero-order correlations can be misleading. Shared variance between any two variables can make it appear that both variables are related to Ho scores, when in fact only one variable may be (Cohen & Cohen, 1983). Take, for example, the finding that contempt and disgust are both related to Ho scores. This could result from any of the following possibilities: 1) each variable shares unique variance with Ho scores that cannot be attributed to shared variance between the two variables; 2) contempt and disgust are related to each other, but only contempt has a unique relationship with Ho score; or 3) contempt and disgust are related to each other but only disgust has a unique relationship with Ho scores. The lack of a significant zero-order correlation between contempt and disgust make the second and third explanations unlikely. However, one can best choose among these alternative explanations by examining semi-partial correlation coefficients via a multiple regression approach. These relationships are explored in the regression section below, in the context of the discussion of my original regression hypotheses.

Regression hypotheses

In Chapter 1, I had originally proposed the following hypotheses for the regression model:

Hypothesis 2.5. The number of anger, contempt, disgust, and Glare expressions will each account for significant variance in Ho scores.

Hypothesis 2.6. Contempt and disgust will account for approximately the same amount of variability in Ho scores (the proportion of variance contributed to the model will not differ significantly between the two), but each will account for significantly more variance than anger or Glare.

The results of the zero-order correlations suggest that it may not be profitable to examine all of the expression variables proposed in hypotheses 2.5 and 2.6. Indeed, a multiple regression model including all of the emotions in hypotheses 2.5 and 2.6, did not account for significant variability in Ho scores ($F_{(4, 89)} = .57, p = .68$). Thus, I pursued more exploratory analyses, in which I employed a model that included those emotions with most promise of accounting for unique variability in Ho scores according to the zero-order correlations: contempt and disgust. Although anger showed no relationship with Ho scores in zero-order correlations, it was also included in the model, because of its conceptual relevance to the construct of "hostility". Also, it is possible that anger's relationship with Ho scores was suppressed by shared variance with contempt and disgust (cf., Cohen & Cohen, 1983, pp. 94 - 95 for a discussion of suppression effects).

I conducted a multiple regression analysis in which the anger, contempt, and disgust composite variables were forced into the model. The full model including anger, contempt, and disgust accounted for a significant portion of variance in Ho scores ($R^2 = .09, F_{(3, 89)} = 3.01, p = .03$). By examining the squared semi-partial correlation coefficients from the regression model, we can determine how much variance in Ho scores could be uniquely attributable to each variable. Table 6 presents these squared semi-partial coefficients (sr^2s), and the significance tests for each variable (which indicate whether this portion of variance in Ho was significant or not). Only contempt expressions accounted for a significant amount of variability in Ho scores, accounting for most of the explained variance from the entire regression model.

Table 6. Contributions to variability in Ho scores ($R^2 = 9\%$)

	sr^2	p
anger	.01	.40
contempt	.06	.01
disgust	.02	.17

Thus, although I hypothesized that anger, contempt, and disgust would all account for unique variability in Ho scores and that contempt and disgust would account for similar amounts of variability in Ho scores, it appears that only contempt expressions account for variability in Ho scores. This is consistent with psychometric studies, which have shown that the Cook-Medley measures a cynical form of hostility (Barefoot, 1992; Smith, 1992).

The regression analyses tell us that *across subjects* of all the expression variables, only contempt expressions account for a significant amount of variability in Ho scores. What they do not tell us, however, is whether individual subjects who score high on Ho consistently show more contempt than subjects who score low on hostility. In order to answer those questions, more specific analyses need to be conducted.

I split the sample into thirds on the basis of Ho scores, to form low, medium, and high hostility groups. The low hostility group had Ho scores of less than 15, the middle hostility group had scores greater than or equal to 15 and less than 23, while the high hostile group had scores equal to or greater than 23. I then determined the proportion of subjects in each group who showed more than one expression of contempt during the critical period. Figure 3 depicts the results.

One can see that there are progressively more subjects who show more than one contempt expression as we moves from low to high hostility subjects, as would be predicted from the regression analyses. Of the low hostile subjects, only about 30% showed more than one contempt expression, of the mid-level hostility group half showed more than one expression of contempt, but of the high hostility group 63% showed more than one contempt expression. Pairwise comparisons between each set of proportions showed that the low hostility group showed significantly less than the middle and high hostility groups ($z_{low\ vs.\ mid.} = 1.65$, 1-tail $p < .05$, $z_{low\ vs.\ high} = 2.65$, 1-tail $p = .004$), but the proportion of subjects in the middle hostility group who

showed more than one contempt expression did *not* differ significantly from the high hostility group ($z = 1.07$, 1-tail $p = .14$).

Chapter 4

Discussion

The primary question underlying the research in Study 1 is a causal one: which emotions or emotional reactions to stress can lead to deleterious coronary events such as ischemia? In Chapter 1 I presented this question in terms of the cardiovascular reactivity model, and conceived of emotional reactions to stressors as either mediating variables between dispositional factors and cardiovascular outcomes or independent influences on cardiovascular outcomes (as per Figure 1). The findings from Study 1 provide correlational evidence that certain facial expressions of emotion -- be they dispositionally provoked or not -- are linked with transient myocardial ischemia in the context of the Structured Interview. As predicted, anger expression and non-enjoyment smiling were more common in ischemics than non-ischemics. Counter to prediction, however, contempt and disgust expression were not. Whether the anger and non-enjoyment smiling behavior that characterized the ischemic group were a cause or effect of ischemia remains to be seen, as does the extent to which these behaviors may be indicative of an enduring affective trait. On this latter point, it is relevant that the Cook-Medley Hostility Scale (Cook & Medley, 1954) showed no relationship with ischemia in the present context. While this could suggest that the anger effects from Study 1 operated at the state level rather than the trait level, this is as yet unclear, because the findings from Study 2 indicate that the Cook-Medley may not be a good index of trait anger. Only the expression of contempt accounted for unique variability in Cook-Medley scores, contrary to my hypothesis that this would be the case for three emotions -- anger, contempt, and disgust.

In the pages that follow, I present a critical analysis of the findings from Study 1 and Study 2 in terms of their implications for both health psychology and basic research on emotion. Each study is analyzed separately, and in that context considerations for future

research are discussed. These sections are followed by an integration of the two studies at a more global level of analysis.

Discussion of Study 1

Implications for health psychology

Study 1 showed that in the emotional context of the Structured Interview, facial expressions of particular emotions can be useful in determining when ischemia occurs in coronary artery disease patients. A pattern of facial responding characterized by anger expression and non-enjoyment smiling was more common in patients who showed ischemia than in those who did not. The relationship between anger expression and ischemia is consistent with previous theory and research that documents a relationship between hostility (an anger-related trait or disposition) and coronary artery disease (e.g., Williams et al., 1980).

The non-enjoyment smiling finding is more complicated. Previous research has shown that these smiles are characterized by neither the self-report of enjoyment nor the physiological patterns of true enjoyment (Ekman et al., 1990; Ekman & Davidson, 1993). There are several possible functions for these smiles. They may be attempts to be polite or attempts to mislead (Ekman & Friesen, 1982). Non-enjoyment smiles might also be considered forms of *facial management* , which either modulate an expression of another feeling or falsify the message sent (Ekman & Friesen, 1975). This is an interesting interpretation, given that the non-enjoyment smiles were interspersed with expressions of anger for many of the ischemic subjects. Another intriguing possibility is that the non-enjoyment smiles were attempts to falsely put forth or simulate the experience of happiness.

The possibility that the anger and non-enjoyment smiling behavior pattern is a potential manifestation of both anger expression and attempts to modulate the appearance

of anger should be studied further, in more anger-specific contexts, because it is consistent with some previous findings in the literature. In studies of coded vocal and speech behavior from audio tapes of the Structure Interview, researchers have described two types of anger-related behaviors that are independently associated with the degree of atherosclerosis: 1) people's tendencies to become irritated when provoked, and 2) "anger-in," or the inability or unwillingness to express anger (Dembroski & MacDougall, 1983; Dembroski et al., 1985; MacDougall et al., 1985). On the basis of the present research, however, it is impossible to say whether the non-enjoyment smiles were facial manifestations of an "anger-in" tendency or whether they simply reflected attempts to be polite in the interview context.

The presence of the non-enjoyment smiling and anger expressions in ischemics is also reminiscent of the behavior described for the cancer-prone "Type C" person, who suppresses anger expression and/or puts a positive face forward under stress (for review see Contrada, Leventhal, & O'Leary, 1990). It is worth noting that researchers have reported that the relationship between Type C behavior and cancer risk may be especially problematic for women (Hagnell, 1966). Of the six women in this study, all were ischemic, all showed high proportions of non-enjoyment smiling, but only one showed anger. Could this be a type of illness related behavior similar to that described in Type C studies? It would be helpful to study a larger sample of female cardiac patients, to determine if non-enjoyment smiling in the absence of anger is consistently linked with ischemia in women.

Study 1 also plays as an important methodological role in cardiovascular health psychology. Although one previous study has used comprehensive facial coding to uncover patterns of emotional behavior that distinguish Type A from Type B males (Chesney et al., 1990), the present research is the first to comprehensively measure facial expression and relate expressive changes to clinically significant coronary events. FACS coded facial expressions of emotion provided a picture of emotional responding

that was not available from other measures in this study. If I had relied solely on Cook-Medley scores, I would have found no evidence of emotion-related differences in the incidence of ischemia. As already stated, the scales for subjective ratings of emotion were impoverished, in addition to being obtained after the interview rather than during the interview. Speech content from the interview could be consulted in future work with this data set, to see if emotional information obtained from verbal sources during the interview corresponds with emotional information from facial expressions.

Why did contempt and disgust fail to discriminate between groups? Of the four types of expressions predicted to distinguish between ischemics and non-ischemics (anger, contempt, disgust, and non-enjoyment smiling), contempt and disgust did *not* show the predicted effects. In fact, while I had hypothesized that contempt would be more frequent in ischemics than non-ischemics, there were borderline effects in the *opposite* direction. Although there was a non-significant trend for one variant of disgust to occur more frequently in ischemics than non-ischemics [all disgust expressions with AU9 (nose wrinkle) only], there were no consistent statistically significant differences between ischemics and non-ischemics on disgust expression.

I had proposed that contempt and disgust would vary with anger in discriminating between groups based on Izard's (1971, 1977) notion that these three emotions are core to hostility. Given previous research (Krantz et al., 1991) that had shown that ischemia occurred more often in high-hostile patients (Cook-Medley measured), I reasoned that the three supposedly "hostility-relevant" emotions would occur more in ischemics. The findings of Study 2 showed, however, that contempt was the only "hostility-relevant" emotion in this triad. Further, Cook-Medley hostility did not relate to ischemia in this study.

In Chapter 1 I explained that previous results from emotion research indicate that anger is associated with heart rate increases relative to baseline, while disgust (and presumably contempt, due to its similarity with disgust) is associated with heart rate

decreases from baseline (Levenson et al., 1990). I argued, however, that it was possible for contempt and disgust to relate positively to ischemia for two reasons. First, as putative correlates of hostility, these emotions might occur in people who are prone to anger. Given that ischemia was measured from a two minute sampling period, it is possible that more than one type of emotion occurred during this interval even if only anger could created the physiological conditions to *cause* ischemia. Second, I cited previous research that has shown that ischemia is not necessarily related to large heart rate increases (Deanfield et al., 1983, 1985; Krantz et al., 1991) so that the apparently opponent physiological effects of anger and disgust might not pertain to ischemia. My finding that anger was the only negative emotion significantly related to ischemia (and that contempt *tended* to behave in the opposite direction) suggests that the physiological differences among anger and disgust reported in previous research might have contributed to the ischemia effects observed. This interpretation requires further studies of heart rate, blood pressure, and other hemodynamic changes that precede ischemia in coronary artery patients.

The "anxiety" effect: Explaining the absence of the anger/non-enjoyment pattern in "low anxious" ischemics. The joint pattern of anger and non-enjoyment smiling had a stronger relationship with ischemia in patients who reported experiencing higher anxiety during the interview than those who did not. Further analyses revealed that the subset of ischemics who did not show this behavior pattern were low on reported anxiety in post-interview subjective ratings. It is important to keep in mind, however, that there was no specificity in subjective measures of negative emotion. For all practical purposes, *anxiety* was the only negative emotion rated. While it is safe to assume that this rating reflected some type of negative emotion, to call it anxiety rather than anger or sadness or fear would be too liberal an interpretation of these data. Thus, I will use the term "anxiety" in quotation marks only.

As mentioned in the previous chapter, there are at least three possible explanations for the "anxiety" effect. One is methodological. The ratings might simply indicate that this group of subjects was not aroused emotionally by the context of the interview questions for which facial data were available. That does not mean that the subjects who showed ischemia but no emotional behavior were not under mental stress, but perhaps that they did not consciously perceive this stress or they chose not to report it.

A second possible explanation for the "anxiety" effect is that those ischemics who reported low anxiety but did not display the anger/non-enjoyment smile behavior may have been repressing or suppressing specific emotions. Repression has been defined operationally in terms of low reported anxiety, low expressivity, high social desirability, and high cardiovascular reactivity (Weinberger, 1990; Weinberger, Schwartz, & Davidson, 1979). While the "low anxious" ischemics were not less expressive overall, the repression interpretation would be consistent with the fact that neither their subjective reports nor their expressive behavior indicated that these patients felt anger. If there is repression operative in this group, it could be specific to the emotion of anger. Clearly there is a need for much more empirical work on this question. For the subjects in the present research, there are heart rate and blood pressure reactivity data available from the remainder of the interview, as well as trait ratings on anxiety and social desirability. Although these data were not available for use in this dissertation, including such variables in subsequent analyses with this dataset should shed light on the repression question. If the ischemics who did not show the anger/non-enjoyment smiling pattern were low on trait measures of anger, high on social desirability, and high on cardiovascular reactivity throughout the rest of the interview, then this would support repression interpretation. If the repression argument withstands further empirical scrutiny, then it should be studied directly in future studies, in men as well as women.

A third possible explanation for the "anxiety" effect is that the difference in emotional behavior and reported experience between these groups of ischemics is attributable to

differences in the degree of ischemia. There is evidence that emotional variables are more predictive of ischemia in more severely ischemic groups (Krantz et al., 1991). Perhaps the ischemia of those subjects who did *not* show the anger/non-enjoyment smiling pattern was less intense than the ischemia of those who did show the facial response pattern. Unfortunately, the dichotomous ischemia variable used in the present research did not allow for examination of intensity variations within the ischemic group.

Implications for emotion research

I have emphasized the benefits of applying the tools and concepts of emotion research to health psychology, but the health psychological context can inform emotion research as well. Study 1 allowed for an ecologically valid test of two important areas of debate in the emotion literature: 1) the relationship between facial expression and physiological change, and 2) the extent to which there is specificity in emotional responding.

1) The relationship between facial expression and physiological change. Independent of its contributions to health psychology, Study 1 is basic research on emotion, as an investigation of the relationship between facial expression and physiological change during an emotionally-evocative situation. Although the sample population of coronary artery disease patients limits the generality of my findings, this limitation is far outweighed by the benefit of being able to look at how facially manifest emotion corresponded with changes in a direct measure of cardiac *function*, one that has not been studied before in emotion research.

Most emotion theorists agree that emotions are complex phenomena, characterized by subjective, physiological, and expressive components, but there are numerous views on the nature of the relationship among these response measures. Some evolutionary or biological theorists purport that the three systems are tightly linked during an emotional response (e.g., Darwin, 1872/1965; Ekman, 1977; Izard, 1971; Tomkins, 1962), as do

facial feedback theorists (e.g., Laird, 1974). Among social constructionists, there are those who argue that these systems need not be related at all (Harré, 1986; Shweder, 1993). To them emotions are first and foremost socially-derived *concepts*. Some researchers who study individual differences in expressive style have argued that facial and physiological changes in emotion are inversely related (e.g., Jones, 1950).

In general, the findings of Study 1 are consistent with theories of positive congruence between response systems in emotion, which supports either an evolutionary or facial-feedback point of view. In particular, the anger findings are consistent with previous research on the autonomic effects of anger (Ekman et al., 1983; Levenson et al., 1990), in that anger expression was associated with the increase in cardiovascular activity from baseline (i.e., the presence of ischemia). The non-enjoyment smile findings are consistent with previous research in emotion that reports that these smiles generate patterns of central nervous systems activation that are more akin to those of negative emotion than positive emotion (Ekman & Davidson, 1993).

2) Specificity in emotional responding -- categorical versus dimensional approaches. The question of whether emotions should be described in terms of specific categories (e.g., anger, fear, or happiness) or on the basis of simple dimensions (e.g., positive and negative emotion) is a source of major debate in the emotion literature (e.g., Diener & Emmons, 1984; Ekman, 1977; 1992; Lazarus, 1991; Watson & Tellegen, 1985). The findings from this research underscore the value of looking at emotions on a categorical level. A dimensional approach would have provided a very different picture of these data. Overall negative emotion alone did not differ between groups -- it was only one particular type of negative emotion that mattered. Although positive emotion did differ between groups, a dimensional approach would not have revealed that this effect was attributable primarily to a particular type of smile, one that does not imply the experience of positive emotion.

Discussion of Study 2

Implications for health psychology

Examination of the facial expressions of emotion shown in the anger questioning period of the Structured Interview revealed that of all the emotions hypothesized to be relevant to hostility (i.e., anger, contempt, and disgust), only contempt expression accounted for a unique and significant amount of variability in Cook-Medley Ho scores. The most salient implication of these findings for health psychology concerns the conceptualization of hostility as measured by the Cook-Medley. There has been considerable debate in the health psychological literature over what this instrument measures, and most attempts to explain Cook-Medley Ho scores have been made on the basis of factor analytic questionnaire studies. Recent empirical and theoretical articles indicate that the Cook-Medley measures a "cynical" form of hostility, one that is characterized by resentment, paranoia, and distrust (Blumenthal et al., 1989; Costa et al., 1986; Greenglass & Julkunen, 1989; Smith & Frohm, 1985). While cynicism and contempt are semantically similar terms, the "cynical distrust" notion derives from the fact that certain Cook-Medley items measure paranoia, which may or may not be related to contempt. The fact that the variance in Ho scores accounted for by contempt amounted to only 7%, suggests that this scale primarily measures emotions, attitudes, or thoughts that were not evoked or measured in the present study context.

In Chapter 1 I distinguished between hostility and anger on the basis of one being an affective trait or disposition and the other being an affective state, respectively. The emphasis on the distinction among different levels of affect (e.g., anger versus hostility) in Study 2 makes clear that health psychologists should reconsider their bias toward a trait or disposition approach to the question of the role of affect in coronary disease or other illnesses. My findings suggest that research at the level of affective state can inform theories of the role of affect/behavior in heart disease as well. Furthermore, the state level of analysis should be advocated -- whether in service of trait explanation via

the study of aggregated state episodes or for purposes of investigating state effects alone -- simply because there is more direct measurement available at that level of analysis. Over the past 20 years, emotion researchers have developed a sophisticated body of tools for measuring changes in affective state, including extensive behavioral, physiological, and paper and pencil techniques. The assessment of traits or dispositions relies largely on pencil and paper questionnaires.

Additionally, the findings from Study 2 contribute to our understanding of the components of hostility. In Chapter 1 I explained that the construct of hostility had been described as having "experiential" and "expressive" components (Dembroski et al., 1989). While I criticized this distinction on the basis of how it misrepresents what can be measured by a self-report instruments or even audio-tape coding schemes, it is nonetheless likely that hostility as a construct can be described in terms of more than one specific dimension or aspect. It certainly has been described this way in factor analytic studies (e.g., Blumenthal et al., 1987). My findings contribute to our understanding of the expressive and affective characteristics of hostility as measured by the Cook-Medley by demonstrating that -- counter to prediction -- contempt was the *only* emotion related to Ho scores. Even though hostility as a construct might also include other negative emotions such as contempt and disgust (as per Izard, 1977), most theorists agree that hostility is primarily *anger* predisposition (Ekman, 1984; Speilberger et al., 1985). My data indicate that the Cook-Medley is not an index of anger predisposition. This is important, given the tendency in the health psychological literature to treat "Ho" as synonymous with the hostility construct in general, as well as a tendency to treat anger as synonymous with hostility.[9]

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[9] For an example of this conceptual problem see the recent article by Siegman et al., (1992), in which the authors resort to combining the terms anger and hostility into *anger-hostility* in reference what is measured by several scales and/or interview assessment techniques in the study of coronary health. This term reflects that the ambiguity in affect measurement via inventories and gross behavioral coding schemes, and epitomizes the confusion created in the literature by the failure of researchers to consider the various levels at which any emotion can be studied (from brief state to enduring trait).

Implications for emotion research

In one sense, this study is precedent-setting research on the relationship between emotions and more enduring affective traits. Many emotion theorists have written on the distinctions among various levels of affect along the state-trait continuum (Ekman, 1977; Lazarus, 1991; Ortony et al., 1988). Yet what is fundamentally acceptable to theorists from vastly different orientations within psychology (e. g., from Ekman to Ortony) is that affective traits influence the occurrence of particular emotions. More to the point, they *enable* the incidence of like emotions: depression facilitates sadness, hostility facilitates anger, and optimism facilitates happiness.

While the findings of Study 2 are relevant to the study of the anger-hostility relationship, the nature of the design of Study 2 limits the generalizability of the results in three ways. First, hostility was measured only in terms of the Cook-Medley, which may or may not be a good measure of hostility as a general construct, even though it is used widely as such. Second, my findings are relevant only to the question of which facial expressions of emotion -- exhibited in the context of questions about anger -- are relevant to Ho scores. While one would expect that other measures of emotion might correspond similarly to Ho, the face was the only emotion measure used in this study. Third, the expression data that were used to account for variability in Ho were derived from only a single context. One would prefer to assess multiple contexts in the study of the state-trait relationship, in the sense that a trait implies stability in behavior (Epstein, 1980).

With these limitations in mind, there are nonetheless several interesting points raised by my findings. The fact that anger expression did not *uniquely* account for a significant amount of variability in Ho scores suggests that the Cook-Medley does not measure an anger-expressive form of hostility. The relationship of Ho scores with the frequency of contempt expression and the typological findings that high-Ho patients showed more contempt than low-Ho patients certainly indicate that the Cook-Medley

measures an affective disposition that relates to contempt expression in some way. Whether the observed contempt expression is the behavioral manifestation of what factor analytic, psychometric studies of the Cook-Medley call "cynical hostility" or "cynical distrust" remains to be seen, but this is a reasonable interpretation. Perhaps the paranoid dimension would not be evident via facial expression, but through speech content. This possibility could be examined in further work with this data set and others.

It may be that the Cook-Medley is more a measure of contempt[10], while hostility as a general construct is marked by anger, contempt, and disgust expression, as per Izard's (1977) proposal of the anger-contempt-disgust triad for hostility. This can only be determined on the basis of future research that measures emotion expression and employs more than one trait measure of hostility. The Buss-Durkee Hostility Inventory (Buss & Durkee, 1957), which has been linked with gross behavioral signs of aggression (c.f., Dembroski et al., 1985), might be useful for this purpose.

Integration and Conclusions

The findings from the present research support the notion derived from previous theory and research that anger is relevant to coronary disease. The emphasis on direct emotion measurement, however, speaks to a role for anger at the "emotion" level, not at the "trait" level. The lack of a relationship between anger and hostility in Study 2, however, is probably more indicative of a failure of Ho scores to capture the dispositional correlate of anger rather than it is of the possibility that anger expression in an "anger context" is not representative of an underlying affective disposition. The

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[10] There has been debate in the emotion literature over whether the emotion contempt is a consistently recognized from facial expression of emotion. Expressions of contempt similar to those shown spontaneously by subjects in this study have been linked with the word "contempt" in forced-choice studies. Rosenberg and Ekman (1994) have shown that while people cannot provide a label for this expression in a free response task, it nevertheless has clear signal value. Observers consistently recognized these putative "contempt" expressions as indicating a certain type of social situation that many emotion theorists would call a contemptuous situation.

Cook-Medley may simply not be a good index of what most emotion theorists would call *hostility*, but rather its relationship with the expression of contempt in Study 2 indicate that it may well measure another type of disposition.

Even though emotion theorists (e.g., Ekman, 1984) have posited that hostility is marked by the ease and frequency of occurrence of anger, this does not mean that this *single* context was a good test of this notion. The Structured Interview context was not especially provocative. Ultimately, one needs to look at anger exhibited across a variety of situations -- or at least across similar situations on repeated instances -- and relate the data obtained about anger across these contexts with trait measures of hostility.

There are two possible explanations for the fact that the Cook-Medley scores were related to neither anger expression nor ischemia. First, the type of hostility measured by the Cook-Medley might reflect the predisposition towards contempt more than the predisposition towards anger. Clearly, this is what my data indicate, but it should be replicated in other, more provocative contexts. Second, it could well be that a trait measure such as the Cook-Medley would better relate to more a more stable index of ischemia. In this study I looked at only a single instance of a *transient* cardiac event. One would expect that Ho scores would be related to aggregated ischemic episodes (an issue Blumenthal will be studying with this sample).

If the Cook-Medley is related to neither anger expressivity across a variety of contexts nor to ischemia, then it would be safe to conclude that if the psychological factors that lead to ischemia are in some sense a function of a type of hostility, then it is a *type* of hostility that is not measured by the Cook-Medley. I would not expect this to be the case, however, because there have been too many positive findings from prospective and cross-sectional research using this tool to discount its predictive utility. Findings that Cook-Medley scores predict heart disease mortality (Shekelle et al., 1983) and the degree of atherosclerosis (Williams et al., 1980) suggest that Ho scores should relate to aggregated occurrences of ischemia.

As another tack, one could abandon inventory measurement of affective traits, and simply define an affective trait or disposition operationally -- in terms of consistent responding across repeated instances of similar contexts. If one could obtain evidence that anger expression across several situations is consistently linked with ischemia, then this would be compelling evidence that at both the dispositional and state level anger has deleterious coronary effects.

On the basis of the findings one can say with confidence that anger expression is linked with ischemia. Whether this "anger" response is a mediator of a disposition-to-ischemia linkage or whether it simply indicates that anger can cause ischemia is unclear. The lack of a trait measure related to transient ischemia and the lack of evidence that the inventory I used even measures "trait anger" in this single context makes this question hard to address.

While the fact that anger expression was more common in ischemics indicates that this emotion *may* actually alter cardiac function, the non-enjoyment smiles -- as expressions that do not reflect felt positive emotion -- may well be signs of personality characteristics or behavioral styles that are more common in patients who show ischemia under stress than those who do not. Why are coronary patients who show non-enjoyment smiles under stress more likely to show ischemia? Do they do this intentionally to feign happiness? Is this behavior an effort to compensate for anger expression? These are all provocative questions raised by the current research, which can only be answered on the basis of future studies.

The questions raised by the "anxiety" effect speak to the importance of measuring potential moderating variables, ensuring that the emotion situation is adequately intense to thoroughly test the idea that certain emotions can lead to cardiac change, and taking adequately refined measurements of subjective experience. Consideration of such methodological improvements in future work should help us determine whether there are two sub-groups of ischemic coronary patients: anger expressive and anger-repressive.

My plans for future work include studying the extent to which the relationships observed in the present research occur in more intense emotional contexts and when emotion is measured from multiple response systems. Most importantly, by inducing emotion in an experimental context, I can more directly assess whether anger, in particular, can *cause* ischemic events. Such a step is crucial, but not final. Longitudinal studies that determine whether emotions can lead to each of the cardiovascular outcomes in the reactivity model -- or other feasible intermediary mechanisms -- is the only research strategy that can support a definite causal role for emotion in heart disease.

Chapter 5

References

Baker, L. J., Hastings, J. E., & Hart, J. D. (1984). Enhanced psychophysiological responses of Type A coronary patients during Type A-relevant imagery. *Journal of Behavioral Medicine, 7*, 287-306.

Barefoot, J. C. (1992). Developments in the measurement of hostility. In H. S. Friedman (Ed.), *Hostility, coping, and health* (pp. 13-31). Washington, D. C.: American Psychological Association.

Barefoot, J. C., Dahlstrom, W. G., & Williams, R. B. (1983). Hostility, CHD incidence, and total mortality: A 25-year follow-up study of 255 physicians. *Psychosomatic Medicine, 45*, 59-63.

Barefoot, J. C., Dodge, K. A., Peterson, B. L., Dahlstrom, W. G., & Williams, R. B., Jr. (1989). The Cook-Medley hostility scale: Item content and ability to predict survival. *Psychosomatic Medicine, 51*, 46-57.

Barefoot, J. C., Haney, T. L., Simpson, S., Blumenthal, J. A., & Williams, R. B. (1990). Depression and the assessment of Type A behavior in a clinical population. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 2*, 483-485.

Barry, J., Selwyn, A. P., Nabel, E. G., Rocco, M. B., Mead, K., Campbell, S., & Rebecca, G. (1988). Frequency of ST-segment depression produced by mental stress in stable angina pectoris from coronary artery disease. *American Journal of Cardiology, 61*, 989-993.

Blumenthal, J. A., Barefoot, J. C., Burg, M. M., & Williams, R. B. (1987). Psychological correlates of hostility among patients undergoing coronary angiography. *British Journal of Medical Psychology, 60*, 349-355.

Blumenthal, J. A., Herman, S., O' Toole, L. C., Haney, T. L., Williams, R. B., & Barefoot, J. C. (1985). Development of a brief self-report measure of the Type A

(coronary-prone) Behavior Pattern. *Journal of Psychosomatic Research, 29*, 265-274.

Blumenthal, J. A., & Kamarck, T. (1987). Assessment of the Type A Behavior Pattern. In J. A. Blumenthal &. D. C. McKee (Eds.), *Applications in behavioral medicine and health psychology: A clinician's sourcebook* . Sarasota, FL: Professional Resource Exchange, Inc.

Blumenthal, J. A., O'Toole, L. C., & Haney, T. (1984). Behavioral assessment of the Type A Behavior Pattern. *Psychosomatic Medicine, 46*, 415-423.

Blumenthal, J. A., Williams, R. B., Kong, Y., Schanberg, S. M., & Thompson, L. W. (1978). Type A Behavior Pattern and coronary atherosclerosis. *Circulation, 58*, 634-639.

Booth-Kewley, S., & Friedman, H. S. (1987). Psychological predictors of heart disease: A quantitative review. *Psychological Bulletin, 101*, 343-362.

Bortner, R. W. (1969). A short rating scale as a potential measure of pattern A behavior. *Journal of Chronic Diseases, 22*, 87-91.

Buss, A. H., & Durkee, A. (1957). An inventory for assessing different kinds of hostility. *Journal of Consulting Psychology, 42*, 155-162.

Chesney, M. A., Ekman, P., Friesen, W. V., Black, G. W., & Hecker, M. (1990). Type A behavior pattern: Facial behavior and speech components. *Psychosomatic Medicine, 53*, 307-319.

Chesney, M. A., Hecker, M., & Black, G. W. (1989). Coronary-prone components of Type A behavior in the WCGS: A new methodology. In B. K. Houston &. C. R. Synder (Eds.), *Type A Behavior pattern: Research, theory, and intervention* (pp. 168-188). New York: Wiley.

Chesney, M. A., & Rosenman, R. H. (Eds.). (1985). *Anger and hostility in cardiovascular and behavioral disorders*. Washington, D.C.: Hemisphere Publishing.

Christensen, A. J., & Smith, T. W. (1993). Cynical hostility and cardiovascular reactivity during self-disclosure. *Psychosomatic Medicine, 55*, 193-202.

Cohen, J. (1977). *Statistical power analysis for the behavioral sciences* (Revised edition). New York: Academic Press.

Cohen, J., & Cohen, P. (1983). *Applied multiple regression/correlation analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum.

Contrada, R. J., Hilton, W. F., & Glass, D. C. (1991). Effects of emotional imagery on physiological and facial responses in Type A and Type B individuals. *Journal of Psychosomatic Research, 35*, 391-397.

Contrada, R. J., & Jussim, L. (1992). What does the Cook-Medley hostility scale measure? In search of an adequate measurement model. *Journal of Applied Social Psychology, 22*, 615-627.

Contrada, R. J., Leventhal, H., & O' Leary, A. (1990). Personality and health. In L. A. Pervin (Ed.), *Handbook of personality: Theory and research* (pp. 638-669). New York: Wiley.

Cook, W. W., & Medley, D. M. (1954). Proposed hostility and pharisaic-virtue scales for the MMPI. *The Journal of Applied Psychology, 38*, 414-418.

Costa, P. T., & McCrae, R. R. (1985). *The NEO personality inventory manual.* Odessa, FL: Psychological Assessment Resources.

Costa, P. T., Zonderman, A. B., McCrae, R. R., & Williams, R. B. (1986). Cynicism, paranoid alienation in the Cook-Medley Ho scale. *Psychosomatic Medicine, 48*, 283-285.

Darwin, C. (1872/1965). *The expression of the emotions in man and animals.* Chicago: University of Chicago Press.

Davidson, R. J., Ekman, P., Saron, C. D., Senulis, J. A., & Friesen, W. V. (1990). Approach-withdrawal and cerebral asymmetry: Emotional expression and brain physiology I. , *58*, 330-341.

Deanfield, J. E., Maseri, A., Selwyn, A. P., Ribeiro, P., Chierchia, S., Krikler, S., & Morgan, M. (1983). Myocardial ischemia during daily life in patients with stable angina: Its relation to symptoms and heart rate changes. *Lancet*, *2*, 753-758.

Deanfield, J. E., Shea, M. J., Kensett, M.., Wilson, R. A, de Landsheere, C. M., & Selwyn, A. P. (1984). Silent myocardial ischemia due to mental stress. *Lancet*, *2*, 1001-1004.

Deanfield, J. E., Shea, M. J., & Selwyn, A. P. (1985). Clinical evaluation of transient myocardial ischemia during daily life. *American Journal of Medicine*, *79 (Supplement 3A)*, 18-24.

Dembroski, T. M., & Costa, P. T., Jr. (1987). Coronary-prone behavior: Components of the Type A patterns and hostility. *Journal of Personality*, *55*, 211-235.

Dembroski, T. M., MacDougall, J. M., Costa, P. T., Jr., & Grandits, G. A. (1989). Components of hostility as predictors of sudden death and myocardial infarction in the Multiple Risk Factor Intervention Trial. *Psychosomatic Medicine*, *51*, 514-522.

Dembroski, T. M., MacDougall, J. M., & Lushene, R. (1979). Interpersonal interaction and cardiovascular responses in Type A subjects and coronary patients. *Journal of Human Stress*, *5*, 28-36.

Dembroski, T. M., MacDougall, J. M., Shields, J. L., Petitto, J., & Lushene, R. (1978). Components of Type A coronary-prone behavior pattern and cardiovascular responses to psychomotor performance challenge. *Journal of Behavioral Medicine*, *1*, 159-176.

Dembroski, T. M., MacDougall, J. M., Williams, R. B., Jr., Haney, T. L., & Blumenthal, J. A. (1985). Components of Type A, hostility, and anger-in: relationship to angiographic findings. *Psychosomatic Medicine*, *47*, 219-233.

Derogatis, L., Lipman, R. S., Rickels, K., Uhlenhuth, E. H., & Covi, L. (1974). The Hopkins symptom checklist (HSCL): A measure of primary symptom dimensions.

In P. Pichot (Ed.), *Psychological measurements in psychopharmacology: Modern problems in pharmacopsychiatry* . Basel: Karger.

DeWood, M. A., & Rozanski, A. (1986). Long-term prognosis of patients with and without silent ischemia. *Circulation, 94,* 59.

Diener, E., & Emmons, R. A. (1984). The independence of positive and negative affect. *Journal of Personality and Social Psychology, 47,* 1105-1117.

Dimsdale, J. E., Hackett, T. P., Black, P. C., & Hutter, A. M. (1978). Type A personality and extent of coronary atherosclerosis. *American Journal of Cardiology, 42,* 583-586.

Dimsdale, J. E., Hackett, T. P., & Hutter, A. M. (1979). Type A behavior and angiographic findings. *Journal of Psychosomatic Research, 23,* 273-276.

Edwards, J. R., Baglioni, J., & Cooper, C. L. (1990). The psychometric properties of the Bortner Type A scale. *British Journal of Medical Psychology, 81,* 315-333.

Ekman, P. (1977). Biological and cultural contributions to body and facial movement. In J. Blacking (Ed.), *Anthropology of the body* (pp. 53-84). London: Academic Press.

Ekman, P. (1984). Expression and the nature of emotion. In K. R. Scherer &. P. Ekman (Ed.), *Approaches to emotion* (pp. 319-343). Hillsdale: Lawrence Erlbaum.

Ekman, P. (1985). *Telling lies: Clues to deceit in the marketplace, marriage, and politics.* New York: Norton.

Ekman, P. (1992). An argument for basic emotions. *Cognition and Emotion, 6,* 161-168.

Ekman, P., & Davidson, R. J. (1993). Voluntary smiling changes regional brain activity. *Psychological Science, 4,* 342-345.

Ekman, P., Davidson, R. J., & Friesen, W. V. (1990). The Duchenne smile: Emotional expression and brain physiology II. *Journal of Personality and Social Psychology, 58*, 342-353.

Ekman, P., & Friesen, W. V. (1969). Nonverbal leakage and clues to deception. *Psychiatry, 32*, 88-105.

Ekman, P., & Friesen, W. V. (1975). *Unmasking the face: A guide to recognizing emotions from facial expressions.* Englewood Cliffs, NJ: Prentice-Hall.

Ekman, P., & Friesen, W. V. (1976). Measuring facial movement. *Journal of Environmental Psychology and Nonverbal Behavior, 1*, 56-75.

Ekman, P., & Friesen, W. V. (1978). *The facial action coding system.* Palo Alto: Consulting Psychologists Press.

Ekman, P., & Friesen, W. V. (1982). Felt, false, and miserable smiles. *Journal of Nonverbal Behavior, 6*, 238-252.

Ekman, P., Friesen, W. V., & Ancoli, S. (1980). Facial signs of emotional experience. *Journal of Personality and Social Psychology, 39*, 1125-1134.

Ekman, P., Friesen, W. V., & Ellsworth, P. (Eds.). (1972). *Emotion in the human face: Guidelines for research and an integration of findings.* New York: Pergamon.

Ekman, P., Friesen, W. V., & Ellsworth, P. (1982). Methodological decisions. In P. Ekman (Ed.), *Emotion in the human face* (pp. 22-38). Cambridge: Cambridge University Press.

Ekman, P., & Heider, K. (1988). The universality of a contempt expression: A replication. *Motivation and Emotion, 12*, 303-308.

Ekman, P., Levenson, R. W., & Friesen, W. V. (1983). Autonomic nervous systems activity distinguishes between emotions. *Science, 221*, 1208-1210.

Epstein, S. (1980). The stability of behavior: II. Implications for psychological research. *American Psychologist, 35*, 790-806.

Evans, P. D., & Moran, P. (1987). The Framingham Type A scale, vigilant coping, and heart-rate reactivity. *Journal of Behavioral Medicine, 10*, 311-321.

Feuerstein, M., Labbe, E. E., & Kuczmierczyk, A. R. (1986). *Health psychology: A psychological perspective.* New York: Plenum.

Fox, N. A., & Davidson, R. J. (1988). Patterns of brain electrical activity during facial signs of emotion in 10-month old infants. *Developmental Psychology, 24*, 230-236.

Frank, K. A., Heller, S. S., Kornfeld, D. S., Sporn, A. A., & Weiss, M. B. (1978). Type A behavior pattern and coronary angiographic findings. *Journal of the American Medical Association, 240*, 761-763.

Fridlund, A. J., Ekman, P., & Oster, H. (1987). Facial expressions of emotion: Review of the literature, 1970-1983. In A. W. Siegman &. S. Feldstein (Eds.), *Nonverbal communication* (pp. 143-224). Hillsdale, New Jersey: Lawrence Erlbaum.

Fridlund, A. J., Price, A. W., & Fowler, S. C. (1982). Low-noise, optically isolated electromyographic preamplifier. *Psychophysiology, 19*, 701-705.

Friedman, H. S., & Booth-Kewley, S. (1987). Personality, Type A behavior, and coronary heart disease: The role of emotional expression. *Journal of Personality and Social Psychology, 53*, 783-792.

Friedman, M., Byers, S. D., Diamant, J., & Rosenman, R. H. (1975). Plasma catecholamine response of coronary-prone subjects (Type A) to a specific challenge. *Metabolism, 24*, 205-210.

Friedman, M., & Rosenman, R. H. (1959). Association of specific overt behavior pattern with blood and cardiovascular findings: Blood cholesterol level, blood clotting time, incidence of arcus senilis and clinical coronary artery disease. *Journal of the American Medical Association, 169*, 1286-1296.

Friedman, M., & Rosenman, R. H. (1974). *Type A behavior and your heart.* New York: Fawcett.

Goldband, S. (1980). Stimulus specificity of physiological response to stress and the Type A coronary-prone behavior pattern. *Journal of Personality and Social Psychology, 39*, 670-679.

Gottleib, S., Gottleib, S. H., & Achuff, S. (1988). Silent ischemia on Holter monitoring predicts mortality in high-risk post-infarction patients. *Journal of the American Medical Association, 259*, 1030-1035.

Gottleib, S. O., Weisfeldt, M. L., Ouyang, P., Mellits, D., & Gersteinblith, G. (1986). Silent ischemia as a marker for early unfavorable outcomes in patients with stable angina. *New England Journal of Medicine, 314*, 1214-1219.

Greenglass, E. R., & Julkunen, J. (1989). Construct validity and sex differences in Cook-Medley hostility. *Personality and Individual Differences, 10*, 209-218.

Group, F.-B. C. (1982). Ischemic heart disease and psychological patterns: Prevalence and incidence studies in Belgium and France. *Advances in Cardiology, 29*, 25-31.

Guilford, J. P. (1954). *Psychometric Methods (2nd ed.)*. New York: McGraw-Hill.

Hagnell, O. (1966). The premorbid personality of persons who develop cancer in a total population investigated in 1947 and 1957. *Annals of the New York Academy of Sciences, 125*, 846-855.

Hardy, J. D., & Smith, T. W. (1988). Cynical hostility and vulnerability to disease: Social support, life stress, and physiological response to conflict. *Health Psychology, 7*, 447-459.

Harré, R. (1986). An outline of the social constructionist viewpoint. In R. Harré (Ed.), *The social construction of emotion* . Oxford, England: Basil Blackwell.

Haynes, S. G., Levine, S., Scotch, N., Feinleib, M., & Kannel, W. B. (1978). The relationship of psychosocial factors to coronary heart disease in the Framingham study. I. Methods and risk factors. *American Journal of Epidemiology, 107*, 362-383.

Hearn, M. D., Murray, D. M., & Luepker, R. V. (1989). Hostility, coronary heart disease, and total mortality: A 33-year follow-up study of university students. *Journal of Behavioral Medicine, 12*, 105-121.

Hecker, M., Chesney, M. A., Black, G. W., & Frautschi, N. (1988). Coronary-prone behaviors in the Western Collaborative Group Study. *Psychosomatic Medicine, 50*, 153-164.

Hedges, S. M., Krantz, D. S., Contrada, R. J., & Rozanski, A. R. (1990). Development of a diary for use with ambulatory monitoring of mood, activities, and physiological function. *Journal of Psychopathology and Behavioral Assessment, 12*, 203-217.

Helmer, D. C., Ragland, D. R., & Syme, S. L. (1991). Hostility and coronary artery disease. *American Journal of Epidemiology, 133*, 112-122.

Helmers, K., Krantz, D. S., Howell, R. H., Klein, J., Bairey, C. N., & Rozanski, A. (1993). Hostility and myocardial ischemia in coronary artery disease patients: Evaluation by gender and ischemic index. *Psychosomatic Medicine, 55*, 29-36.

Ivancevich, J. M., & Matteson, M. T. (1988). Type A behavior and the healthy individual. *British Journal of Medical Psychology, 61*, 37-56.

Izard, C. E. (1971). *The face of emotion.* New York: Appleton-Century-Crofts.

Izard, C. E. (1977). *Human emotions.* New York: Plenum.

Jenkins, C. D., Rosenman, R. H., & Friedman, M. (1967). Development of an objective psychological test for the determination of the coronary prone behavior pattern in employed men. *Journal of Chronic Diseases, 20*, 371-379.

Jenkins, C. D., Zyzanski, S. J., & Rosenman, R. H. (1979). *The Jenkins Activity Survey for health prediction.* New York: The Psychological Corp.

Joesoef, M. R., Wetterhall, S. F., DeStefano, F., Stroup, N. E., & Fronek, A. (1989). The association of peripheral arterial disease with hostility in a young, healthy veteran population. *Psychosomatic Medicine, 51*, 285-289.

Jones, H. E. (1950). The study of patterns of emotional expression. In M. Reymert (Ed.), *Feelings and emotions*. New York: McGraw-Hill.

Kamarck, T. W., Manuck, S. B., & Jennings, J. R. (1990). Social support reduces cardiovascular reactivity to psychological challenge: A laboratory model. *Psychosomatic Medicine, 52*, 42-58.

Keppel, G. (1982). *Design & Analysis: A researcher's handbook* (2nd ed.). Englewood-Cliffs, NJ: Prentice-Hall.

Kleck, R. E., Vaughan, R. C., Cartwright-Smith, J., Vaughan, K. B., Colby, C. Z., & Lanzetta, J. T. (1976). Effects of being observed on expressive, subjective, and physiological responses to painful stimuli. *Journal of Personality and Social Psychology, 34*, 1211-1218.

Koskenvuo, M., Kaprio, J., Rose, R. J., Kesaniemi, A., Sarna, S., Heikkila, K., & Langinvainio, H. (1988). Hostility as a risk factor for mortality and ischemic heart disease in men. *Psychosomatic Medicine, 50*, 330-340.

Krantz, D. S., Arabian, J. M., Davia, J. E., & Parker, J. S. (1982). Type A behavior and coronary artery bypass surgery: Intraoperative blood pressure and perioperative complications. *Psychosomatic Medicine, 44*, 273-284.

Krantz, D. S., & Durel, L. A. (1983). Psychobiological substrates of the Type A behavior pattern. *Health Psychology, 2*, 393-411.

Krantz, D. S., Gabbay, F. H., Hedges, S. M., Leach, S. G., Gottdiener, J. S., & Rozanski, A. (1993). Mental and physical triggers of silent myocardial ischemia: Ambulatory studies using self-monitoring diary methodology. *Annals of Behavioral Medicine, 15*, 33-40.

Krantz, D. S., Helmers, K., F., Bairey, N., Nebel, L. E., Hedges, S. M., & Rozanski, A. (1991). Cardiovascular reactivity and mental stress-induced myocardial ischemia in patients with coronary artery disease. *Psychosomatic Medicine, 53*, 1-12.

Krantz, D. S., & Manuck, S. B. (1984). Acute psychophysiological reactivity and risk of cardiovascular disease: A review and methodological critique. *Psychological Bulletin, 96*, 435-464.

Krantz, D. S., Schaeffer, M. A., Davia, J. E., Dembroski, T. M., MacDougall, J. M., & Shaffer, R. T. (1981). Extent of coronary atherosclerosis, Type A behavior, and cardiovascular response to social interaction. *Psychophysiology, 18*, 654-664.

Krause, R., Steimer, E., Sanger-Alt, C., & Wagner, G. (1989). Facial expression of schizophrenic patients and their interaction partners. *Psychiatry, 52*, 1-12.

Laird, J. D. (1974). Self attribution of emotion: The effects of expressive behavior on the quality of emotional experience. *Journal of Personality and Social Psychology, 29*, 475-486.

Lake, B. W., Suarez, E. C., Schneiderman, N., & Tocci, N. (1985). The Type A behavior pattern, physical fitness, and psychophysiological reactivity. *Health Psychology, 4*, 169-187.

Lazarus, R. S. (1991). *Emotion and adaptation.* New York: Oxford University Press.

Leon, G. R., Finn, S. E., Murray, D., & Bailey, J. M. (1988). The inability to predict cardiovascular disease from hostility scores or MMPI items related to Type A behavior. *Journal of Consulting and Clinical Psychology, 56*, 597-600.

Levenson, R. W., Ekman, P., & Friesen, W. V. (1990). Voluntary facial action generates emotion-specific autonomic nervous system activity. *Psychophysiology, 27*, 363-384.

Leventhal, H. (1970). Findings and theory in the study of fear communications. *Advances in Experimental Social Psychology, 5*, 119-186.

Light, K. C., Herbst, M. C., Bragdon, E. E., Hinderliter, A. L., Koch, G. G., Davis, M. R., & Sheps, D. S. (1991). Depression and Type A behavior pattern in patients with coronary artery disease: Relationships to painful versus silent ischemia and ß-endorphin responses during exercise. *Psychosomatic Medicine, 53*, 669-683.

Lundberg, U., Hedman, M., Melin, B., & Frankenhaeuser, M. (1989). Type A behavior in healthy males and females as related to physiological reactivity and blood lipids. *Psychosomatic Medicine, 51*, 113-122.

MacDougall, J. M., Dembroski, T. M., Dimsdale, J. E., & Hackett, T. P. (1985). Components of Type A, hostility, and anger-in: Further relationships to angiographic findings. *Health Psychology, 4*, 137-152.

Manuck, S. B., & Garland, F. N. (1979). Coronary-prone behavior pattern, task incentive, and cardiovascular response. *Psychophysiology, 16*, 139-142.

Maruta, T., Hamburgen, M. E., Jennings, C. A., Offord, K. P., Colligan, R. C., Frye, R. L., & Malinchoc, M. (1993). Keeping hostility in perspective: Coronary heart disease and the hostility scale on the Minnesota Multiphasic Personality Inventory. *Mayo Clinic Proceedings, 68*, 109-114.

Matthews, K. A. (1982). Psychological perspectives on the Type A behavior pattern. *Psychological Bulletin, 91*, 293-323.

Matthews, K. A., Glass, D. C., Rosenman, R. H., & Bortner, R. W. (1977). Competitive drive, Pattern A, and coronary heart disease: A further analysis of some data from the Western Collaborative Group Study. *Journal of Chronic Diseases, 30*, 489-498.

Matthews, K. A., & Haynes, S. G. (1986). Type A behavior pattern and coronary disease risk. *American Journal of Epidemiology, 123*, 923-960.

McCranie, E. W., Watkins, L. O., Brandsma, J. M., & Sisson, B. D. (1986). Hostility, coronary heart disease (CHD) incidence and total mortality: lack of association in a 25-year follow-up study of 478 physicians. *Journal of Behavioral Medicine, 9*, 119-125.

Ortony, A., Clore, G. L., & Collins, A. (1988). *The cognitive structure of emotions.* Cambridge, England: Cambridge University Press.

Powell, L. H., Friedman, M., Thoresen, C. E., Gill, J. J., & Ulmer, D. K. (1984). Can the Type A behavior pattern be altered after myocardial infarction? A second year report from the recurrent prevention project. *Psychosomatic Medicine, 46*, 293-313.

Ragland, D. R., & Brand, R. J. (1988). Coronary heart disease mortality in the Western Collaborative Group Study. *American Journal of Epidemiology, 127*, 462-475.

Rosenberg, E. L., Ekman, P. (1994). Conceptual and methodological issues in the judgment of facial expressions of emotion. Unpublished manuscript.

Rosenberg, E. L., & Ekman, P. (in press). Coherence between expressive and experiential systems in emotion. *Cognition & Emotion, 8.*

Rosenman, R. H. (1978). The interview method of assessment of the coronary-prone behavior pattern. In S. M. Weiss, T. M. Dembroski, J. L. Shields, S. G. Haynes, & M. Feinleib (Eds.), *Coronary-prone behavior* (pp. 55-70). New York: Springer-Verlag.

Rosenman, R. H., Brand, R. J., Jenkins, D., Friedman, M., Straus, R., & Wurm, M. (1975). Coronary heart disease in the Western Collaborative Group Study: Final follow-up experience of 8 1/2 years. *Journal of the American Medical Association, 233*, 872-877.

Rosenman, R. H., Friedman, M., Straus, R., Wurm, M., Kositchek, R., Hahn, W., & Werthessen, N. T. (1964). A predictive study of coronary artery disease. *Journal of the American Medical Association, 189*, 113-124.

Rozanski, A., Bairey, C. N., Krantz, D. S., Friedman, J., Resser, K. J., Morell, M., Hilton-Chalfen, S., Hestrin, L., Bietendorf, J., & Berman, D. S. (1988). Mental stress and the induction of silent myocardial ischemia in patients with coronary artery disease. *The New England Journal of Medicine, 318*, 1005-1012.

Schang, S. J., & Pepine, C. J. (1977). Transient asymptomatic ST-segment depression during daily activity. *American Journal of Cardiology, 53*, 396-402.

Scherer, K. R. (1981). Vocal indicators of stress. In J. Darby (Ed.), *Speech evaluation in psychiatry*, (pp. 171-187). New York: Grune & Stratton.

Scherer, K. R. (1989). Vocal correlates of emotional arousal and affective disturbance. In H. Wagner &. A. Manstead (Eds.), *Handbook of Social Psychophysiology*, New York: John Wiley & Sons.

Scherwitz, L. (1988). Interview behaviors in the Western Collaborative Group Study and the Multiple Risk Factor Intervention Trial structured interviews. In B. K. Houston &. C. R. Snyder (Eds.), *Type A Behavior Pattern: Research, theory, and intervention* (pp. 32-50). New York: Wiley.

Scherwitz, L., Berton, K., & Leventhal, H. (1977). Type A assessment and interaction in the behavior pattern interview. *Psychosomatic Medicine, 39*, 229-240.

Scherwitz, L., Graham, L. E. II, Grandits, G., & Billings, J. (1987). Speech characteristics and behavior-type assessment in the Multiple Risk Factor Intervention Trial (MRFIT) structured interviews. *Journal of Behavioral Medicine, 10*, 173-195.

Scherwitz, L., McKelvain, R., Laman, C., Patterson, J., Dutton, L., Yusim, S., Lester, J., Kraft, I., Rochelle, D., & Leachman, R. (1983). Type A behavior, self-involvement, and coronary atherosclerosis. *Psychosomatic Medicine, 45*, 47-57.

Schucker, B., & Jacobs, D. R. (1977). Assessment of behavior pattern A by voice characteristics. *Psychosomatic Medicine, 39*, 219-228.

Schwartz, G. E., Weinberger, D. A., & Singer, J. A. (1981). Cardiovascular differentiation of happiness, sadness, anger, and fear following imagery and exercise, *Psychosomatic Medicine , 43*, 343-364.

Shekelle, R. B., Gale, M., & Norusis, M. (1985). Type A score (Jenkins Activity Survey) and risk of recurrent coronary heart disease in the Aspirin Myocardial Infarction Study. *American Journal of Cardiology, 56*, 221-225.

Shekelle, R. B., Gale, M., Ostfeld, A. M., & Paul, O. (1983). Hostility, risk of coronary heart disease and mortality. *Psychosomatic Medicine, 45*, 109.

Shekelle, R. B., Hulley, S. B., Neston, J. D., Billings, J. H., Borboni, N. O., Gerace, T. A., Jacobs, D. R., Lasser, N. L., Mittlemark, M. B., & Stamler, J. (1985). The MRFIT behavior pattern study: Type A behavior and incidence of coronary heart disease. *American Journal of Epidemiology, 122*, 559-570.

Shott, S. (1990). *Statistics for health professionals.* Philadelphia: W. B. Saunders.

Shweder, R. A. (1993). The cultural psychology of the emotions. In J. Haviland &. M. Lewis (Eds.), *The handbook of emotions.* New York: Guilford.

Siegel, W. C., Mark, D. B., Hlatky, M. A., Harrell, F. E., Pryor, D. B., Barefoot, J. C., & Williams, R. B. (1989). Clinical correlates and prognostic significance of Type A behavior and silent myocardial ischemia on the treadmill. *American Journal of Cardiology, 64*, 1280-1283.

Siegman, A. W., Anderson, R., Herbst, J., Boyle, S., & Wilkinson, J. (1992). Dimensions of anger-hostility and cardiovascular reactivity in provoked and angered men. *Journal of Behavioral Medicine, 15*, 257-272.

Siegman, A. W., Dembroski, T. M., & Ringel, N. (1987). Components of hostility and the severity of coronary artery disease. *Psychosomatic Medicine, 49*, 127-135.

Smith, T. W. (1992). Hostility and health: Current status of a psychosomatic hypothesis. *Health Psychology, 11*, 139-150.

Smith, W., & Frohm, K. D. (1985). What's so unhealthy about hostility? Construct validity and psychosocial correlates of the Cook and Medley Ho scale. *Health Psychology, 4*, 503-520.

Smith, T. W., Sanders, J. D. & Alexander, J. F. (1990). What does the Cook and Medley hostility scale measures? Affect, behavior, and attributions in the marital context. *Journal of Personality and Social Psychology, 58*, 699-708.

Spielberger, C. D., Jacobs, G., Russell, S., & Crane, R. (1983). Assessment of anger: The state-trait anger scale. In J. N. Butcher &. C. D. Spielberger (Eds.), *Advances in personality assessment.* Hillsdale, N. J.: Lawrence Erlbaum.

Spielberger, C. D., Johnson, E. H., Russell, S. F., Crane, R. J., Jacobs, G. A., & Worden, T. J. (1985). The experience and expression of anger: Construction and validation of an anger expression scale. In M. A. Chesney &. R. H. Rosenman (Eds.), *Anger and hostility in cardiovascular and behavioral disorders.* Washington, D. C.: Hemisphere Publishing.

Steiner, F. (1986). Differentiating smiles. In E. Baenninger-Huber &. F. Steiner (Eds.), *FACS in psychotherapy research* (pp. 139-148). Zurich: Department of Clinical Psychology, Universitat Zurich.

Suarez, E. C., & Williams, R. B. (1989). Situational determinants of cardiovascular and emotional reactivity in high and low hostile men. *Psychosomatic Medicine, 51,* 404-418.

Thoresen, C. E., & Powell, L. H. (1992). Type A Behavior Pattern: New perspectives on theory, assessment, and intervention. *Journal of Consulting and Clinical Psychology, 60,* 595-604.

Tomkins, S. S. (1962). *Affect, imagery, consciousness.* New York: Springer.

Ward, M. M., Chesney, M. A., Swan, G. E., Black, G. W., Parker, S. D., & Rosenman, R. H. (1986). Cardiovascular responses in Type A and Type B men to a series of stressors. *Journal of Behavioral Medicine, 9,* 43-49.

Watson, D., & Tellegen, A. (1985). Toward a consensual structure of mood. *Psychological Bulletin, 98,* 219-235.

Weinberger, D. A. (1990). The construct validity of the repressive coping style. In J. L. Singer (Ed.), *Repression and dissociation: Implications for personality, psychopathology, and health* (pp. 337-386). Chicago: University of Chicago Press.

Weinberger, D. A., Schwartz, G. E., & Davidson, R. J. (1979). Low-anxious, high-anxious, and repressive coping styles: Psychometric patterns and behavioral and physiological responses to stress. *Journal of Abnormal Psychology, 88,* 369-380.

Williams, R. B., Jr., Barefoot, J. C., Haney, T. L., Harrell, F. E., Jr., Blumenthal, J. A., Pryor, D. B., & Peterson, D. B. (1988). Type A behavior and angiographically documented coronary atherosclerosis in a sample of 2,289 patients. *Psychosomatic Medicine, 50,* 139-152.

Williams, R. B., Barefoot, J. C., & Shekelle, R. B. (1985). The health consequences of hostility. In M. A. Chesney &. R. H. Rosenman (Eds.), *Anger and hostility in cardiovascular and behavioral disorders.* Washington, D. C.: Hemisphere Publishing.

Williams, R. B., Jr., Haney, L. T., Lee, K. L., Kong, Y., Blumenthal, J., & Whalen, R. (1980). Type A behavior, hostility, and coronary atherosclerosis. *Psychosomatic Medicine, 42,* 539-549.

Williams, R. B., Lane, J. D., Kuhn, C. M., Melosh, W., White, A. D., & Schanberg, S. M. (1982). Type A behavior and elevated physiological and neuroendocrine responses to cognitive tasks. *Science, 218,* 483-485.

Williams, R. B., Suarez, E. C., Kuhn, C. M., Zimmerman, E. A., & Schanberg, S. M. (1991). Biobehavioral basis of coronary-prone behavior in middle-aged men. Part I: Evidence for chronic SNS activation in Type As. *Psychosomatic Medicine, 53,* 517-527.

Zyzanski, S. J., Jenkins, C. D., Ryan, T. J., Flessas, A., & Everist, M. (1976). Psychological correlates of coronary angiography findings. *Archives of Internal Medicine, 136,* 1234-1237.

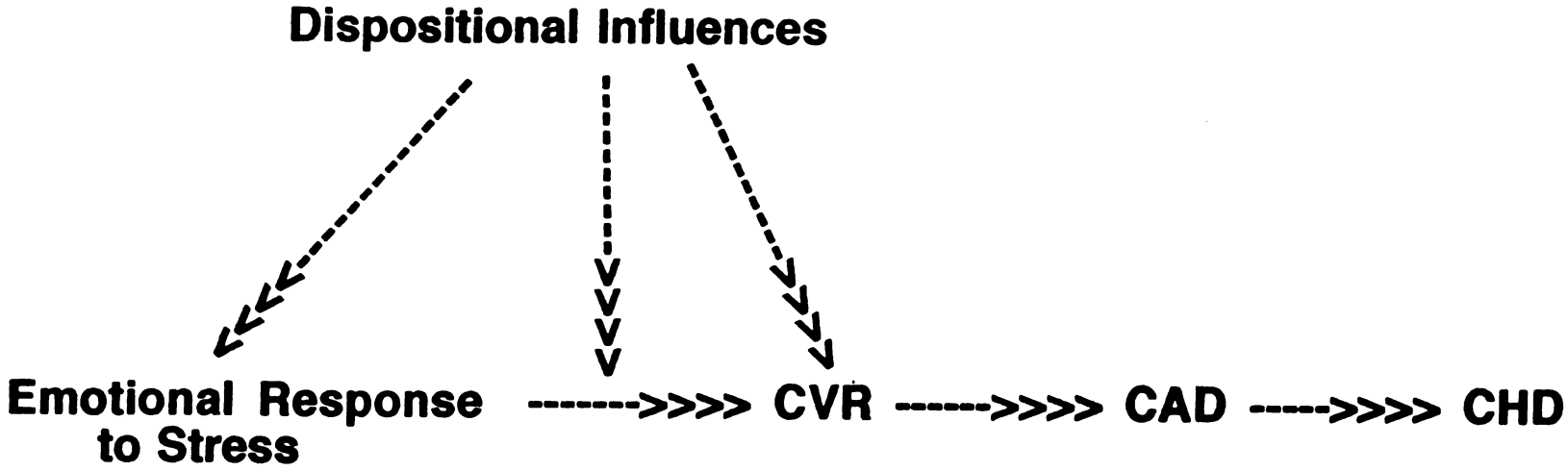

Figure 1. Psychophysiological reactivity model

Figure 2. Percentage of subjects showing both anger and non-enjoyment smiles

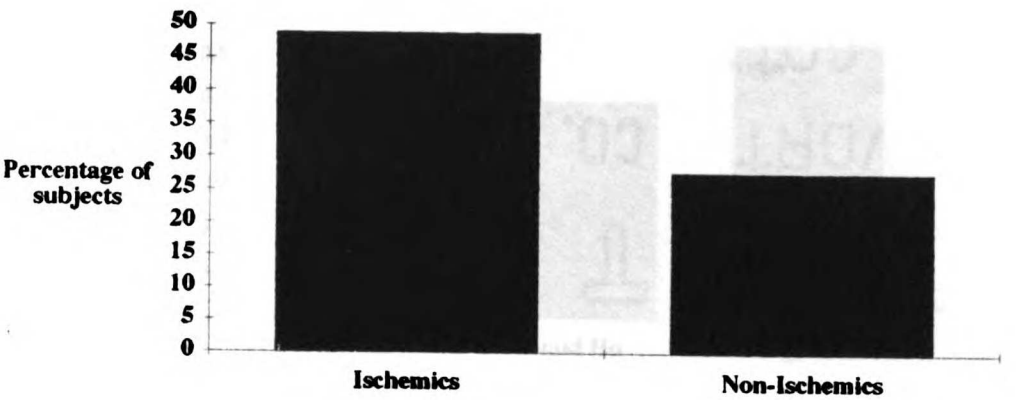

Figure 3. Proportion of subjects showing more than 1 contempt expression for low, middle, and high Ho groups.