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Effects of emotional imagery on cardiovascular and plasma catecholamine responses in Type A and Type B individuals

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EFFECTS OF EMOTIONAL IMAGERY ON CARDIOVASCULAR AND PLASMA CATECHOLAMINE RESPONSES IN TYPE A AND TYPE B INDIVIDUALS

by

WILLIAM F. HILTON, JR.

A dissertation
submitted to the Graduate Faculty in Psychology
in partial fulfillment of the requirements
for the degree of
Doctor of Philosophy,
The City University of New York.

1988

This manuscript has been read and accepted
for the Graduate Faculty in Psychology
in satisfaction of the dissertation requirement
for the degree of Doctor of Philosophy.

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ABSTRACT

EFFECTS OF EMOTIONAL IMAGERY ON CARDIOVASCULAR AND PLASMA CATECHOLAMINE RESPONSES IN TYPE A AND TYPE B INDIVIDUALS

by

WILLIAM F. HILTON, JR.

Adviser: Professor Susan Saegert

Most contemporary approaches investigating risk factors associated with coronary heart disease consider the interaction of psychological and physiological variables. This study examined the cardiovascular, plasma catecholamine, and behavioral responses of 42 Type A and B adult males to a psychological stressor (emctional imagery). Differences in A/B responses to emotional situations that promote stress could reveal other psychosocial or cognitive factors involved in the pathogenesis of CHD. Subjects were instructed to induce affective states by imaging past situations which had made them angry, fearful and distressed. After imaging, subjects described the emotional situations. The three emotional imagery tasks were counterbalanced with

subjects randomly assigned to control for sequence effects. Subject's expressions during the emotion task were video taped for subsequent ratings of facial affect by naive judges using semantic differential scales. It was expected that Type A's would express anger freely, but would suppress emotional reactions when relating fear or distress, engendering concomitant physiological dampening. Unexpectedly, Type B's exhibited greater increases in systolic blood pressure and plasma norepinephrine across all emotions. This finding was supported by behavicral measures showing Type B's rated higher on unpleasant affect. These results were discussed in terms of increased facial expressiveness linked to physiological arousal for Type B's and situational control and suppressed facial expression for Type A's.

ACKNOWLEDGEMENTS

The author would like to acknowledge and thank Laura Erlanger and Jean Landeau who were the primary Pattern A interviewer-auditors. I also want to thank Beverly Bannister, Carla Collins, Laura Erlanger, Anne-Marie James, and Susan Rosenthal who served as research assistants, and Carmele Colatorti, Theresa DeSalvio, Barbara Ann Ford, and Jeannette Michael, judges of non-verbal expressions.

Grateful acknowledgement is extended to Richard J. Contrada, who served as an auditor, for his kind advice and for being a supportive and helpful colleague; to Kathleen Kehoe, for conducting medical screenings, collecting blood samples and assisting in data tabulation; to Willam Isecke for providing electrical engineering assistance; to Barry Snow who served as an auditor, and for recruiting research assistants and facial affect judges; and to Ellen Elting, Lawrence F. Krakoff, and C. Raymond Lake for providing catecholamine assays. In addition I would like to thank J. Alan Herd of Harvard University Medical School and Lawrence R. Krakoff of the Mount Sinai School of Medicine for their advice throughout this study.

Acknowledgements and thanks are expressed to Samuel J. Messick for counsel pertaining to methodology; to Carolyn Springer of Columbia Teachers College and Robert Tobias of the NYC Board of Education for their timely statistical consultations; and to Prudence Opperman and Henry Soloman of the NYC Board of Education for their kind advice and encouragement.

My gratitude is extended to James M. Jones and the American Psychological Association, Minority Fellowship Program for their support, and especially to Dean K. Harrison of the CUNY Office of Expanded Educational Opportunity for his invaluable counsel and support throughout my graduate career. A debt of gratitude is also owed to Roger K. Williams, other professors and fellow students at Morgan State University, for endowing me with the spirit of inquiry, a quest for excellence, and unrelenting perseverance.

This study could not have come to fruition without the instruction, guidance, facilities and support of David C. Glass, for which I am grateful. I am also indebted to other members of my dissertation committee for their assistance with this work: To Susan Saegert who piqued my interest in stress research and interdisciplinary study, for her encouragement and judicious criticism; to Gary Winkel for his instruction and guidance in methodology and statistics; to Dalton Miller-Jones for much needed assistance in persever-

ance and the vicissitudes of academe; and to Suzanne Ouellette-Kobaza who graciously consented to participate in this endeavor on short notice, and for crystallizing critical issues.

Finally, I would like to acknowledge and thank all members of my family for their patient understanding and loving support, but especially to my parents, wife and son for their enduring help through this experience.

This research was supported, in part, by a grant from the National Heart, Lung and Blood Institute (NIH), HL-22514, and a National Research Service Award from the Department of Health and Human Services (NIH), 5 T32 MH15742-03.

CONTENTS

ABSTRACT iv

ACKNOWLEDGEMENTS vi

page

INTRODUCTION 1

A Conceptual Overview 1

REVIEW OF THE LITERATURE 4

A Synopsis of Coronary Heart Disease 4
Risk Factors for Coronary Disease 5
The Concept of Type A Behavior 6
Assessment of Type A Behavior 8
Type A Behavior and Clinical Coronary Heart Disease 11
Type A Behavior, Hostility and Coronary Disease 13
Type A Behavior, Reactivity and Coronary Disease 15
Imagery, reactivity and Type A behavior . . 16
Mechanisms Connecting Type A to CHD 17
Psychological stress and cardiovascular SAM concomitants 18
Psychological stress and catecholamines . . 19
Type A behavior and cardiovascular SAM concomitants 20
Plasma catecholamine measures and Type A behavior 21
Type A Behavior and the Suppression of Affect . . 27
Behavioral Index of Suppression 30
Suppression of facial expressiveness and physiological reactivity 30

STATEMENT OF THE PROBLEM AND RATIONALE FOR THE STUDY . . 33

STATEMENT OF HYPOTHESES 35

METHOD 36

Selection of Subjects 36
Demographics 38

Cardiovascular Screening 40
Behavior Pattern Classification 41
Measurement of Blood Pressure and Heart Rate . . . 42
Blood Collection and Measurement of Plasma Catecholamines 43
Measurement of Facial Affect 46
Design of The Study 48
Induction of Affective States by Emotional Imagery 49
Dependent Measures 50
Experimental Protocol 51

METHODS OF ANALYSIS 55

RESULTS 57

Physiological Baseline Measures 57
Cardiovascular Measures 59
Reactivity from Baseline 59
Resting Level Reactivity 61
Catecholamines 63
Behavioral Measures 64

DISCUSSION 70

Appendix page

A. BEHAVIOR PATTERN INTERVIEW 82

B. EXPERIMENTAL SCRIPT 86

C. POST-EXPERIMENTAL INTERVIEW AND DEBRIEFING 98

D. VIDEO FACIAL RATINGS 101

E. SUBJECTS CONSENT STATEMENT 103

F. FACTOR ANALYSIS OF VIDEO FACIAL RATINGS 105

BIBLIOGRAPHY 106

LIST OF TABLES

Table		page
1.	Subject's Educational Levels	40
2.	Mean Baseline Values for Each Experimental Group	59
3.	Systolic Blood Pressure: ANCOVA with Contrasts	60
4.	Systolic Blood Pressure: ANCOVA with Contrasts	62
5.	Norepinephrine: ANCOVA with Contrasts	64
6.	Video Facial Affect: MANOVA	66
7.	Mean Level of Involvement from Video Ratings	69

LIST OF FIGURES

Figure		page
1.	Counterbalanced Emotions.	48

INTRODUCTION

A Conceptual Overview

The characteristics of individuals which place them at risk for coronary heart disease (CHD) include psychosocial (psychological stress and the coronary-prone behavior pattern Type A) as well as biomedical factors (Glass, 1977a; Jenkins, 1971, 1976). However, attempts to identify most new cases of CHD by a combination of these risk factors has remained inconclusive (Jenkins, 1971). For example, two behavior patterns have been identified (Type A and Type B) which, at least statistically, show differential incidence of CHD, i.e., Type A's tend to have twice the frequency of CHD than Type B's (Rosenman, Friedman, Straus, Wurm, Kositchek, Hahn, & Werthessen, 1964; Rosenman, Brand, Jenkins, Friedman, Straus, & Wurm, 1975). Irrespective of the fact that Type A's are classified coronary prone, there is a relatively low incidence of premature CHD among Type A's. Additionally, classification as Type A is no guarantee for subsequently developing CHD, and conversely having Type B characteristics does not confer protection against CHD (Rosenman, et al., 1964, 1975). What is clear

is that all of the mechanisms underlying Type A's susceptibility to CHD and Type B's resistance are unknown, although a few explanations have been offered (Matthews, 1982; Rosenman, et al., 1975).

It may be that the causal mechanisms underlying cardiovascular disease are not evenly distributed throughout the Type A group, or that only certain components of the behavior pattern (i.e., hostility and anger) confer enhanced coronary risk (Dembroski, MacDougall, Shields, Petitto, & Lushene, 1978; Matthews, Glass, Rosenman, & Bortner, 1977; Williams, Barefoot, & Shekelle, 1985). Furthermore, it is also possible that Type A behaviors of any kind may lead to physiologic changes culminating in illness, but CHD occurs only in some A's because they lack the necessary psychological and/or physiological protective mechanisms (Vickers, Hervig, Rahe, & Rosenman, 1981). In addition, the increased cardiovascular and neuroendocrine reactivity of some persons to psychological stress may be determined by cognitive factors, such as intrapsychic defenses (i.e., suppression or denial) that may combine with behavioral characteristics (i.e., Type A or cynical hostility) in producing clinical outcomes (Glass, 1979; Houston, 1986; Leventhal, 1979: Personal communication; Smith & Frohm, 1985; Steptoe, 1981; Williams, et al., 1985).

Research indicates that under stressful and challenging conditions, Type A individuals show greater cardiovascular

and neuroendocrine reactivity than their Type B counterparts, while not differing in baseline levels of blood pressure, heart rate and plasma catechclamines (Dembroski, et al., 1978; Manuck, Craft, & Gold, 1978; Glass, Krakoff, Contrada, Hilton, Kehoe, Mannucci, Collins, Snow, & Elting, 1980a). Even though these studies present a compelling set of positive results, the fact remains that, similar to CHD studies, A/B effects are statistical. In any given study, there are Type A subjects who do not substantially elevate over their baseline levels while in experimental conditions where high reactivity is expected. It has been suggested that in situations which show A/B reversals, Type A persons may utilize certain cognitive mechanisms, such as suppression, which lower physiological reactivity (Pittner & Houston, 1980). Conversely, there are Type B's who have large elevations, contrary to theoretical expectation. There is some evidence to suggest that Type B's may be more reactive than their Type A counterparts under certain psychosocial conditions (Glass, 1977). Differences in A/B responses to emotional situations that promote psychological stress may shed light on the mechanisms underlying other psychosocial factors that contribute to the pathogenesis of CHD. To test the aforementioned observations, this research will investigate cognitively mediated affective factors that may influence responses of Type A and Type B persons to stressful situations.

REVIEW OF THE LITERATURE

A Synopsis of Coronary Heart Disease

Coronary heart disease (CHD) refers to a group of clinical disorders that are believed to result from morphological damage to the coronary arteries. This damage, a thickening of the arterial walls, is called atherosclerosis or coronary artery disease (CAD). The major clinical manifestations of this disease include myocardial infarction, angina pectoris and sudden cardiac death (Eliot, 1979).

Myocardial infarction (MI), commonly called heart attack, occurs when an inadequate blood supply causes necrosis or death in a part of the heart tissue. Angina pectoris, also the result of an inadequate supply of blood to the heart, produces paroxysmal attacks of chest pain. Although angina can be extremely painful, it may persist for years without significant damage to any heart tissue. This is not to say that angina pectoris is a benign disease. Affected persons may develop an acute MI, and they have been known to die suddenly. The presence of a thrombosis (clot formation) in one or more of the coronary arteries is sometimes associated with MI. However, whether coronary throm-

bosis plays a precipitating or a secondary role has been debated in recent years, since the pathophysiological processes underlying MI remain unclear. Several studies indicate that coronary thrombosis is rare in cases of sudden death due to cardiac arrhythmias. This has led some investigators to argue that sudden cardiac death and MI are two forms of myocardial necrosis with different pathophysiological mechanisms (Eliot, 1979).

Risk Factors for Coronary Disease

Various epidemiologic studies, such as the Heart Disease Epidemiology Study of the National Heart and Lung Institute at Framingham Massachusetts, suggest that persons prone to CAD and CHD can be identified by specific hereditary factors, metabolic alterations, and features of the individual's lifestyle (Brand, Rosenman, Sholtz, & Friedman, 1976; Herd, 1986; Kannel, McGee, & Gordon, 1976). The most widely accepted risk factors include: age; hypertension; systolic and diastolic blood pressure above 160/95 mm Hg; elevated levels of cholesterol (250-275 mg per 100 ml or greater of blood serum) and related lipoproteins; cigarette smoking (20 or more cigarettes per day); a family history of CHD; and the presence of diabetes mellitus. Even though the probability of developing heart disease is greater when more of the risk factors are present, the best combination of these

factors still fails to identify most new cases of CHD (Jenkins, 1971).

Early prospective studies of CHD risk factors largely ignored psychosocial factors in favor of biomedical variables. In recent years, however, considerable evidence has implicated reactivity to psychological and social factors with the etiology and pathogenesis of CHD (Glass, 1977a; Jenkins, 1971, 1976; Manuck & Krantz, 1986; Williams, et al., 1985). Two categories of psychological variables seem to be most promising as coronary risk factors, these being psychological stress and the Type A coronary-prone behavior pattern (Glass, 1977b; Rosenman & Friedman, 1974). The inclusion of psychosocial risk factors highlights the role that lifestyle may play in the development of CHD, and emphasizes dynamic processes involving the interaction of the individual with his/her environment (Krantz, Glass, Schaeffer, & Davia, 1982).

The Concept of Type A Behavior

The Type A coronary-prone behavior pattern may be defined as "an action-emotion complex that can be observed in any person who is aggressively involved in a chronic, incessant struggle to achieve more and more in less and less time, and, if required, to do so against the opposing efforts of other things or persons (Friedman & Rosenman,

1974)." This set of behaviors is characterized by competitive drive, impatience, time urgency, hostility and aggressiveness, and accelerated speech and motor movements. Type A behavior is conceptualized as the outcome of a person-situation interaction, in which the defining behaviors are observable when a susceptible individual is confronted by appropriately challenging and/or stressful environmental circumstances (Glass, 1977b; Rosenman, 1978; Rosenman & Friedman, 1974). A contrasting Type B behavior pattern is defined as the relative lack of Type A characteristics (Friedman & Rosenman, 1974). Type B persons are generally thought of as being relaxed and easy-going; they tend not to exhibit aggressive drive or time urgent characteristics.

Definitions of Type A and Type B behavior patterns represent the extremes of a hypothetical bipolar continuum. In clinical practice, the designation of a person as Type A, or Type B, depends upon assessment of the intensity and number of Pattern A characteristics (Glass, 1977b; Jenkins, 1975; Krantz, et al., 1982).

Psychological stress has been defined as an internal state of an individual when faced with threats to psychological or physical well-being (Lazarus, 1966). The salient features of stress are the individual's perception of potential harm, and the subsequent perception that his/her resources are inadequate to ccpe with the threat. Challenge is also defined in perceptual terms. The major dif-

ference is that the salient feature of a challenge is the potential for reward rather than for harm (Baum, Singer, & Baum, 1981; Lazarus, 1980).

The Type A concept does not refer to the stressful or challenging conditions that elicit Type A behavior, the individual responses themselves, or some hypothetical personality trait that could produce them. Rather, Pattern A refers to a persistent set of overt behaviors that occur in susceptible individuals given the appropriate eliciting conditions, namely, challenging and/or stressful situations.

Assessment of Type A Behavior

There are two principal techniques for assessing Type A behavior, these being a structured interview (SI) and a self-administered questionnaire. The first, the SI, was conceived and designed by Meyer Friedman and Ray H. Rosenman to elicit characteristics of Type A behavior (Rosenman, 1978; Rosenman, Friedman, Straus, Wurm, Kositchek, Hahn, & Werthessen, 1964). The SI focuses on the interplay between the individual being assessed and the interview situation. The person is asked approximately twenty-five questions pertaining to competitive drive, impatience and daily irritations. The interviewer deliberately phrases some of the questions to create a stressful and challenging atmosphere for the participant. Questions are asked in a brisk manner,

and the person is occasionally challenged by the interviewer by interrupting a response in midsentence. It is assumed that these conditions are optimal for eliciting Type A characteristics (Glass, 1977b; Rosenman, 1978; Rosenman, et al., 1964).

Assessments based on the interview emphasize the person's mannerisms and tone of response to questions. These features are considered to be more important than the content of the answers, although content is used as a determinant of behavior pattern classification. Several studies have shown that assessments using manner of response alone, are more highly correlated with global A/B assessment than just the content of a person's answers (Matthews, Krantz, Dembroski & MacDougall, 1982; Scherwitz, Berton & Leventhal, 1977; Schucker & Jacobs, 1977).

Individuals who are Type A become impatient with the interview pace and periodic interruptions, and try to override the interviewer while answering questions. Typical of a Type A person is the frequent use of explosive vocal intonations, rapid and accelerated speech, intermittent sighing, and rhythmic motor behavior. Type B persons may exhibit some A characteristics, but rarely in an exaggerated form. Type B's tend to be relaxed, usually do not display chronic time urgency, and rarely exhibit accelerated and explosive speech stylistics (Glass, 1977b; Rosenman, 1978).

Behavior pattern assessments based on the SI are made on a four-point scale: Extreme, or fully developed A's (A1); incompletely developed A's (A2); relative absence of A characteristics (B); and an intermediate pattern comprised of characteristics of both A2 and B (X). Interviews are tape recorded for independent assessment by two trained raters. Various studies have shown agreement rates ranging from 75 to 84% (Caffrey, 1968; Jenkins, Rosenman & Friedman, 1965; Keith, Lown & Stare, 1965). Moreover, persons in one of these studies, Jenkins, et al., (1965) showed the same classification over a 12 to 20 month period. It would seem that the SI is a stable measure for behavior pattern classification.

The other commonly used technique for assessing Type A behavior is the Jenkins Activity Survey for Health Prediction (JAS). Developed as an objective alternative to the structured interview by C. David Jenkins, the JAS is a self-administered questionnaire which provides a continuous distribution of A-B scores (Glass, 1977b; Jenkins, Rosenman & Friedman, 1967; Jenkins, Zyzanski & Rosenman, 1971, 1979). JAS A-B scores usually show an agreement of about 70% with a dichotomous A/B assessment by the SI (Jenkins, et al., 1971; MacDougall, Dembroski & Musante, 1979). Since the JAS, a self-report measure, relies only on response content, it may measure different components of Pattern A than the SI (Krantz, et al., 1982). Comparative studies have shown that

the interview is, in fact, a stronger predictor of CHD than the JAS (Brand, Rosenman, Jenkins, Sholtz & Zyzanski, 1978; Jenkins, Rosenman & Zyzanski, 1974).

Type A Behavior and Clinical Coronary Heart Disease

The possible association between A-like behaviors (e.g. time-urgent, achievement-oriented, competitive and hostile) and clinical CHD had been suspected before the turn of the century (Krantz, et al., 1982; Osler, 1892). As noted earlier, the systematic description and definition of the Type A behavior pattern comes from the work of Friedman and Rosenman in 1959. Since that time, studies have demonstrated that Type A persons show a higher incidence and prevalence of CHD than Type B's (Friedman & Rosenman, 1959; Jenkins, 1971; Rosenman, et al., 1964). The strongest available evidence for this association comes from the prospective Western Collaborative Group Study (WCGS). The WCGS results indicate that of approximately 1500 Type A men who were free of CHD at intake, 12% (178) developed CHD during the 8.5 year follow-up period. Of approximately 1500 Type B's, 5% (79) cases also developed CHD. In short, Type A men experienced about twice the incidence of acute clinical events compared to Type B men (Rosenman, et al., 1964; Rosenmen, Brand, Jenkins, Friedman, Straus, & Wurm, 1975). This difference occurred independently of other risk

factors, including total serum cholesterol, elevated systolic blood pressure, and cigarette smoking.

Analysis of prospective data from the Framingham Heart Study, also found that Type A was related to the incidence of CHD even when standard risk factors were controlled (Haynes, Feinleib, & Kannel, 1980). It would appear, then, that Type A behavior is an independent risk factor associated with increased CHD (Weiss, Cooper, & Detre, 1981).

There also may be an association between Pattern A and coronary atherosclerosis. Several studies, using angiographic procedures, have examined the relationship between measures of Type A and the extent of atherosclerosis. Angiography is a diagnostic procedure that uses cardiac catheterization to determine the presence and severity of coronary artery disease (CAD) in living patients. At least three studies concur in reporting positive associations between severity of occlusion of the coronary arteries and measures of Type A behavior (Blumenthal, Williams, Kong, Schanberg, & Thompson, 1978; Frank, Heller, Kornfeld, & Sporn, 1978; Zyzanski, Jenkins, Ryan, Flessas, & Everist, 1976). In a study of men who, during a 17 month period, underwent repeated angiography, Krantz, Sanmarco, Selvester, and Matthews (1979) found a positive association between the progression of coronary atherosclerosis and the magnitude of Type A scores. In this study, extreme Type B subjects were unlikely to show significant increases in occlusions of the

coronary arteries. It should be noted, however, that recent studies by Dimsdale, Hackett, Hutter, Block, Catanzano, and White (1979) failed to confirm an association between Type A and the severity of atherosclerosis. These null results introduce a note of caution into conclusions that may be drawn from the angiographic research related to Type A behavior. However, additional psychosocial factors, i.e. anger and hostility, have recently been shown to contribute to the progression of CAD and possibly subsequent CHD (Houston, 1986; Williams, et al., 1985).

Type A Behavior, Hostility and Coronary Disease

Chronic hostility, an attitudinal set, and its frequently associated emotion, anger, have recently been related to an increased prevalence of CAD and CHD risk by three separate studies, two of which were prospective (Williams, et al., 1985). In an angiographic study which replicated findings of more severe CAD among Type A patients, Williams and associates also found a positive relationship between Type A behavior (as assessed by the SI) and hostility (as assessed by the Cook and Medly Ho scale of the MMPI) (Williams, Haney, Lee, Kong, Blumenthal, & Whalen, 1980). Further research not only reaffirmed the link between Type A, hostility and anger, but also suggested that some of the variance in CAD prevalence was due to increased

hostility levels among Type A patients (Williams, et al., 1985). Separate prospective studies of working-age men were able to confirm an independent association between hostility, as measured by the Ho scale, and increased CHD incidence over follow-up periods of 20-25 years. In both studies the test-retest correlation of Hc scores, obtained after a duration of one to several years, was remarkably high (at least .84 or above), indicating that the psychological characteristic assessed by the Ho scale is stable over time, as is Type A behavior. Even though hostility is a component of Type A behavior, only a certain form of hostility independently contributes to detrimental health effects (Williams, et al., 1985). Several investigations have described the psychosocial characteristic being measured by the Ho scale (Smith & Frohm, 1985; Williams, et al., 1985). A high Ho scorer is not likely to be overtly aggressive or assaultive, but instead harbors persistent cynical attitudes. Cynically hostile persons view others with distrust and resentment, believing people are generally undependable, mean or selfish; attitudes that often lend themselves to producing anger. To the extent that the Ho interpersonal style is operationally distinct from the Type A behavior pattern and consistent with findings regarding CAD, the Ho scale seems to assess an independent behavioral risk for health (Smith & Frohm, 1985; Williams, 1984; Williams, et al., 1985).

Type A Behavior, Reactivity and Coronary Disease

Excessive physiological reactivity to environmental influences is hypothesized to have detrimental effects on health (Matthews, 1986). Most of the available evidence linking reactivity and coronary disease is indirect, but suggestive (Manuck & Krantz, 1986; Steptoe & Ross, 1981). One prospective study reported a significant association between the magnitude of diastolic blood pressure responses to the cold pressor test and CHD development during a 23-year follow-up period. A more recent study employing the cold pressor test found exaggerated blood pressure reactions to be predictive of eventual hypertension, a CHD risk factor (Manuck & Krantz, 1986). However, additional studies have not found a prospective relationship between cold pressor reactions and subsequent hypertension.

Two mediating factors through which Type A behavior is believed to potentiate CHD are increased cardiovascular and neuroendocrine reactivity (Williams, 1975). A large number of laboratory studies indicate that Type A subjects, engaged in physical or cognitive tasks, are more reactive than their Type B counter parts (Glass, Lake, Contrada, Kehoe, & Erlanger, 1983). In fact, the enhanced physiological reactivity of Type A individuals, to environmental stressors and challenges, is so pronounced that hyperresponsiveness is considered characteristic of these persons (Glass, 1977b).

Imagery, reactivity and Type A behavior. The importance of reactivity as a separate factor effecting health was demonstrated by a study which reported A-B differences in cardiovascular responses, using Type A relevant imagery as the task (Baker, Hastings, & Hart, 1984). In this instance the environmental influence was mainly cognitive, and apart from the laboratory setting, did not rely on external stress or challenge. The potential of the imagery technique is to pattern complex situations which have been assumed to elicit Type A behavior. An important assumption in previous research with imagery is the notion that physiological responses to imaged stimuli are similar to those occurring in the real situation (Lang, 1979; Lang, Kozak, Miller, Levin, & McLean, 1980).

In the Barker, et al. (1984) study, twenty adult male cardiac patients, who had been classified Type A or Type B by the JAS were asked to image a series of Type A relevant and Type A neutral situations. Subjects were instructed to experience these situations as if they were really happening to themselves. Type A situations described scenes containing elements of time urgency, competitiveness, and loss of control. Neutral scenes did not contain any Type A relevant elements. Subjects rated each imaged scene for vividness and emotional arousal. After the imagery task, subjects were given the structured interview, thereby exposing them to an actual Type A relevant situation. A variety of phys-

iological measures were taken throughout the imagery task and the SI.

No differences were found between Type A's and Type B's on any baseline measures. The results showed that Type A subjects were more physiologically reactive to Type A scenes compared to neutral scenes. Compared to Type B's, Type A's were more reactive throughout the imagery task and the SI. However, both Type A's and Type B's rated Type A scenes as more emotionally arousing than neutral scenes. In relation to Type A behavior, the Barker, et al. (1984) study clearly showed that situational specificity, even though encountered through imagery, can influence reactive outcomes.

Research on imagery suggests that an imaging task would be an appropriate condition for examining A/B differences. Further, it may be important, for understanding processes that lead to increased physiological reactivity, to allow subjects to chose their own event to image. Under such conditions there may be situations where Type B's are more reactive than Type A's. These issues will be explored in a later section.

Mechanisms Connecting Type A to CHD

The mechanisms through which psychological factors, such as stress and Type A behavior, potentiate the pathogenesis of CHD and atherosclerosis are probably mediated by the sym-

pathetic-adrenomedullary (SAM) system (Herd, 1978, 1986; McGeer & McGeer, 1980). The physiological concomitants of increased SAM activity have the potential to facilitate cardiovascular diseases. SAM concomitants include increased blood pressure (BP), heart rate (HR), and circulating catecholamines such as epinephrine (E) and norepinephrine (NE) (Mason, 1972; Schneiderman & Pickering, 1986).

Psychological stress and cardiovascular SAM concomitants. The prototypic relationship between behavior and cardiovascular function is characterized by the classic fight-or-flight response (Frankenhaeuser, 1971; Mason, 1972). The cardiovascular SAM concomitants that comprise fight-or-flight behavior are increased cardiac output (heart rate and blood pressure), decreased peripheral resistance and the shunting of blood away from skin and viscera to the skeletal muscles (vasodilation). Typically, this syndrome prepares the body for physical activity in the presence of potential harm. Similar physiological responses have been observed in humans, where no threat of physical danger was present, using mental arithmetic as the stressor (Abramson & Ferris, 1941; Brod, Fencl, Heji, & Jirka, 1959; Krantz, Manuck, & Wing, 1986). Although the response pattern may appear similar to fight-or-flight activity in animals, the relationship between psychological stress and concomitant physiological responses in humans is more complicated. Two

distinct cardiovascular response patterns are discernable in humans (Williams, 1986). The relationship between psychological stress and cardiovascular function is usually discussed in terms of the person's sensory behavior in respect to environmental factors.

In the presence of stressful psychological stimuli, such as solving a mental arithmetic problem, or experiencing emotions such as anger or fear, sensory rejection behavior (shutting out distracting environmental influences) has been shown to be associated with elevated heart rate and blood pressure, together with skeletal muscle vasodilation (Lacey, Kagan, Lacey, & Moss, 1963). However, when a person is trying to detect events in the environment, sensory intake behavior is effected and heart rate decrements occur along with skeletal muscle vasoconstriction (Lacey, et al., 1963; Obrist, Lawler, & Gaebelein, 1973; Williams, Bittker, Buchsbaum, & Wynne, 1975). The heart rate slows whenever an individual intends to take in sensory information, even if the information is unpleasant (Craig & Wood, 1971). Excellent summaries of these relationships are presented by both Glass (1977b) and Williams (1975, 1986).

Psychological stress and catecholamines. Two SAM hormones known to be responsive to the impact of psychological stressors and implicated in the pathogenesis of CHD are the catecholamines epinephrine and norepinephrine (Eliot, 1979;

Frankenhaeuser, 1971; Haft, 1974; Mason, 1972). Basel rates of catecholamine excretions are generally low. Ordinarily, during daily activities catecholamine rates rise to about twice the resting levels. However, excretion rates of 3 to 5 times the basel rates are not uncommon under moderately stressful conditions (Frankenhaeuser, 1971). Several studies have found increased levels of catechclamines to be associated with components of psychological stress, such as anger, fear and aggressive competition (Ax, 1953; Elmadjian, Hope, & Larson, 1958; Mason, 1972). Of particular interest is the fact that psychological stress can increase catecholamine levels, which in turn influence cardiovascular functions. In addition to inducing acute hemodynamic effects (increases in BP and HR), catechclamines can potentiate lipid mobilization and atherogenesis, processes related to the development of coronary disease (Eliot, 1979; Haft, 1974; Herd, 1978, 1986; Ross & Glomset, 1976).

Type A behavior and cardiovascular SAM concomitants. Several studies report that, while not differing in baseline levels, Type A subjects do, indeed, respond with greater elevations in blood pressure and heart rate than Type B's, when both types of subjects are confronted with stressful or challenging experimental situations (Dembroski, MacDougall, & Shields, 1977; Manuck, Craft, & Gold, 1978; Van Egeren, 1979). Dembroski, et al. (1977) found that Type A's,

assessed with the SI, had greater elevations of systolic blood pressure (SBP) and heart rate (HR) than Type B's while working on a choice reaction time task, under high challenging instructions. A partial replication of this study using a cognitive task under high incentive conditions found that Type A's, assessed by the JAS, were also higher on SBP (but not HR) than similarly assessed Type B's (Manuck, et al., 1978). Further research has established that a positive relationship exists between cardiovascular effects and Type A behavior (Baker, et al., 1984; Blumenthal, Lane, Williams, McKee, Haney, & White, 1983; Corse, Manuck, Cantwell, Giordani, & Matthews, 1982; Dembroski, MacDougall, Herd, & Shields, 1979; Dembroski, MacDougall, Shields, Petitto, & Lushene, 1978). However, principal support for this conclusion comes primarily from studies employing the SI as the method of assessing Pattern A behavior (Wright, Contrada, & Glass, 1984).

Plasma catecholamine measures and Type A behavior. The first study to examine the relationship between measures of plasma catecholamines and Type A behavior was initiated by Friedman and his colleagues (Friedman, et al., 1975). This study involved thirty middle-aged men, classified to be Type A or B by the SI, and divided into A/B pairs in order to engage in competition. The men competed against each other by trying to solve mental puzzles which were virtually

insoluble. Subjects were bled by repeated venipuncture. During the puzzle task the contestants were subjected to music from two radios which were turned to different 'rock' stations. In addition to the radio intrusion, an experimenter, located in the room, announced the number of minutes remaining in the contest. One week later, all subjects returned to the laboratory to have a baseline blood sample drawn.

There were no significant differences for baseline values of norepinephrine between A's and B's. The levels of norepinephrine during the contest were averaged, and found to be significantly higher than baseline levels for Type A's, but not for Type B's. The mean increase of NE during the contest was also greater in Type A subjects, remaining significantly higher after the task was over. There were no corresponding effects for plasma epinephrine. These results indicate elevated sympathetic-adrenal-medullary responses in Type A persons when presented with challenging and stressful situations.

There are several problems with the design and analysis of this experiment. The use of repeated venipuncture, which is, by itself, a stressor can affect behavior-catecholamine relationships. There is also no control group against which to evaluate the competition manipulation, and there is a confounding of competition with other stressors such as instructions designed to promote feelings of time urgency

and impatience. In addition, the analysis of experimental plasma occurred within 72 hours of its collection, while baseline samples were drawn one week after the experimental samples. This procedure engenders the analyzing of baseline and experimental samples in separate catecholamine assay runs, which probably increased the variability of E and NE values raising questions about the reliability of the findings.

Other, more carefully controlled experiments, provide evidence of an association between plasma catecholamines and Type A behavior. In the first of a series of studies, Glass and his colleagues assessed the effects of competition with a benign opponent against competition with a harassing and hostile opponent (Glass, Krakoff, Contrada, Hilton, Kehoe, Mannucci, Collins, Snow, & Elting, 1980a). This study used 44 paid male volunteers (22 Type A and 22 Type B), 35 to 50 years old and free of the major risk factors for CHD. Subjects were classified A or B on the basis of the SI, and randomly assigned to one of the two experimental conditions (Harass - No Harass). The experiment was described as a "tournament," in which a subject played 9 games of a computerized video game similar to tennis, called Pong. A member of the research staff pretended to be another subject, ostensibly the opponent. During the games, the opponent either expressed verbal hostility toward the subject, through a series of predetermined comments at designated

times (Harass condition), or he was benign (No Harass condition).

An indwelling venous catheter was used to collect blood for plasma E and NE levels, thus eliminating repeated venipuncture. The baseline blood sample was taken at the end of a 25-minute relaxation period, prior to the "tournament."

There were no differences in mean baseline values for E and NE either between A's and B's, or between the Harass and No Harass conditions. Increments of both catecholamines in experimental conditions over baseline values were statistically significant for both A's and B's. When submitted to analysis of variance, the change-score data for epinephrine revealed a Harass - No Harass main effect, as well as an interaction between E and the A/B classification. Plasma E increased more for Harassed A's than any of the other experimental groups. Systolic blood pressure elevations over base, as well as HR elevations, showed effects that paralleled the E data. The data for plasma NE, while in the same direction, were not statistically significant.

Several additional studies have demonstrated A/B differences in catecholamine responses. One experiment from Glass' laboratory used an individualized version of Pong. All other aspects of this study were virtually identical to the experiment reported above (Glass, et al., 1980a). The findings indicated an A/B main effect demonstrating Type A's tendency toward higher E levels than Type B's when both are

performing challenging tasks. Another experiment in this series, using task-overload to induce stress and challenge, showed higher catecholamine elevations for Type A's than for Type B's (Glass, Krakoff, Finkelman, Snow, Contrada, Kehoe, Mannucci, Isecke, Collins, Hilton, & Elting, 1980b). Research conducted by Williams et al. (1982), using a serial mental arithmetic task, also reported significant catecholaminergic effects, with Type A's exhibiting greater increases than Type B's (Williams, Lane, Kuhn, Melosh, White, & Schanberg, 1982).

Obrist (1976) has suggested that effortful coping such as attempts to control aversive stimulation can be expected to facilitate sympathetic activity and consequent cardiovascular changes. Experiments have shown that active coping is associated with increased levels of catecholamines such as norepinephrine (Frankenhaeuser & Rissler, 1970; Elmadjian, Hope, & Larson, 1958). In testing this line of thought, further work by Contrada, Glass and their colleagues report differential catecholamine responses in a situation designed to induce uncontrollable threat (Contrada, Glass, Krakoff, Krantz, Kehoe, Isecke, Collins, & Elting, 1982). Under conditions where subjects had the opportunity to control aversive events, Type A's, as predicted, exhibited greater NE levels than B's. Unlike previcus studies from Glass' laboratory which did not vary threatened loss of control, under the circumstances of this study an A-B difference in

NE reactivity would be expected. However, B's unexpectedly exceeded A's in epinephrine elevations under the same conditions.

Even though experimental results, in general, show that A's experience greater catecholamine elevations than B's, in stressful and/or challenging circumstances, one must note that these A/B effects are statistical (Glass, et al., 1983). The fact remains that there are Type A subjects, in any given study, who do not elevate over baseline levels in experimental conditions where increased reactivity is expected. Similarly, there are Type B's who produce elevations under stressful psychosocial conditions. Although these cases present an enigma seeking explanation, they are not so numerous as to impair the internal validity of the investigations where they occur. There is some evidence to suggest that in situations which encourage competitive striving and the attainment of concrete goals, Type B's may perform as compulsively as prototypic A's (Burnam, Pennebaker, & Glass, 1973). Comparative studies already cited have shown that Type B behavior is not a guarantee from contracting CHD (Rosenman, et al., 1964; Rosenman, et al., 1975). Thus far relationships have been shown to exist between psychological stress and cardiovascular SAM concomitants including catecholamines. Type A behavior, in the presence of stress or challenge, is also linked to increased cardiovascular and catechclamine responses, pro-

cesses related to the development of CHD. Perhaps, explanations for reverse A/B results may be found in the combined effect of other psychological behaviors and physiologic factors (Houston, 1986; Matthews, 1986; Williams, et al., 1982). This notion shall be examined in the next section.

Type A Behavior and the Suppression of Affect

It has been proposed that Pattern A behavior is, in part, a coping style intended to overcome potentially uncontrollable environmental stressors (Glass, 1977b). Observations of Type A individuals describe them as persons who usually present themselves as being under control and capable of handling any crisis. Although prone to express overt bravado, Type A's struggle to avoid expression of emotional affect, in an effort to control themselves and the situation (Jenkins, 1975).

An illustration of this point can be found in a study by Carver, Coleman, and Glass (1976). Physical fatigue was induced by a treadmill test. Measurements of subjective fatigue (self-ratings) were obtained throughout the test, as were recordings of the subject's aerobic capacity as an indication of effort exerted on the treadmill. As hypothesized, Type A's suppressed, or at least denied subjective fatigue while exerting greater efforts than Type B's on the treadmill. The authors concluded that suppression may be

understood in terms of the hard-driving characteristic of Pattern A. Thus, concealment of fatigue may have instrumental value for A's because it aids their struggle in the attainment of achievement-related goals. "The acknowledgement of fatigue, on the other hand, might interfere with successful task mastery - a situation which A's could not tolerate easily (Glass, 1977b, p. 48)." Further support for this conclusion comes from a study showing that Type A's use more suppression than Type B's in order to cope with laboratory manipulations of stress (Pittner & Houston, 1980). This study used electric shock and loss of self-esteem as stressors. When threatened with shock, Type A's were not physiologically more reactive than Type B's, on measurements of blood pressure. However, BP readings were higher for Type A's in contrast to B's when threatened with loss of self-esteem. The differences revealed by the Type A response strategy presented in this study is consistent with a situation-specific interpretation of the coronary-prone behavior pattern (Glass, 1982). Situational-specificity offers an explanation of reverse A/B effects, i.e. where A's are less physiologically reactive, under several conditions: When responding is not instrumental to Type A's goals; when the situation is not relevant to Type A characteristics; or when Type B's are motivated to respond greater than Type A's. While not equating physical stressors with affective states, it is reasonable to extrapolate the occurrence

of similar response patterns when Type A's are confronted with psychologically stressful environments (Glass, 1977b; Jenkins, 1975). To the extent that Type A's are effective in reducing emotional affect, they may be able to suppress concomitant physiological arousal. This idea is elaborated in the next section.

The preceding studies indicate it is possible that when confronted with stressful situations, such as those giving rise to fear or distress, Type A's will attempt to cope with the stressor by concealing (i.e., suppressing) affective behavior and possibly dampening physiological reactivity. This view of Type A behavior is consistent with Glass' (1977, 1982) notion that Type A's are people who attempt to ward off threating situations by intense self-protective coping responses.

Anger is perhaps one of the few emotions Type A's may not attempt to conceal, since it is not viewed by these individuals as a threat to their self-perceptions of being in control (Jenkins, 1975). When confronted by a hostile opponent, in a task oriented situation, Type A's were more physiologically reactive than Type B's (Glass, et al., 1980a). It is highly likely that Type A's experiencing anger will not attempt to suppress either the affect or its concomitant physiological reactions.

Behavioral Index of Suppression

Affective states may be assessed through the use of one or more of the following behaviors: Self-reports, physiological reactivity, and facial expressions (Zuckerman, Klorman, Larrance, & Spiegel, 1981). The relationships among these affective responses is central to many theories of emotion (Notarius, Wemple, Ingraham, Burns, & Kollar, 1982).

Suppression of facial expressiveness and physiological reactivity. One of the most prevalent ideas concerning emotional reactions is the facial feedback hypothesis. Based essentially on an arousal model, this hypothesis states that there is a positive relationship between facial expressions and emotional reactions, suggesting that one leads to the other. In an affect-laden situation, persons who are more facially expressive would be expected to exhibit greater physiological activity and stronger subjective reports than persons who show a less intense facial display. Conversely, lower levels of facial expressiveness are accompanied by lower levels of autonomic activity. Support for these hypotheses comes from several studies.

Additional evidence suggests that physiological measurements together with facial ratings are useful in the assessment of certain response mechanisms, such as suppression of affect.

Lanzetta, Cartwright-Smith, and Kleck (1976) administered electrical shock to subjects while requiring them to either exaggerate or suppress their facial responses. The investigators included a baseline period of spontaneous reactions to the shocks. It was found that the baseline results for skin conductance and self-reports of pain were similar to those of the exaggeration phase. In addition the results indicated that when subjects attempted to suppress facial expressions as opposed tc exaggerating their reactions, they showed less electrodermal arousal and rated the shocks as being less painful.

In an experiment by Zuckerman, et al. (1981), randomly assigned subjects were asked to respond to videotaped scenes, which served as stimuli, with either exaggerated expressions, spontaneous behavior or by exhibiting suppression. Heart rate, finger blood volume, skin conductance and respiration rate were monitored. Six one minute scenes, two pleasant, two unpleasant and two neutral were used to elicit the facial and autonomic responses. Subject's facial expressions were videotaped for subsequent rating by naive judges. The results showed that higher levels of autonomic activity were accompanied by higher levels of facial expressiveness and subjective reports of emotional experience. The notion that facial expression affects both autonomic and subjective responses appears to be ccnfirmed, since correlations between autonomic responses and self-reports were

lower than correlations of either measure with facial expressions.

The preceding section presented evidence showing Type A's attempt to suppress emotional affect, excluding anger. In concealing affective behavior, A's possibly decrease their physiological reactivity. Other research shows that suppressed emotion, represented by facial expressiveness, is coupled with decreased physiological responsivity. This evidence demonstrates that ratings of the facial components of emotion, when combined with physiological measures, may be used as a behavioral index of psychophysiological suppression.

STATEMENT OF THE PROBLEM AND RATIONALE FOR THE STUDY

Research reviewed earlier indicated that under stressful and challenging conditions, Type A individuals show greater cardiovascular and neuroendocrine reactivity than their Type B counterparts, while not differing in baseline levels of blood pressure, heart rate and plasma catecholamines. These A/B differences emerge under conditions that are relevant to Type A characteristics, which include competitive, time-dependent, and situations where subjects lack control over environmental factors. Conditions which are not Type A relevant may not produce similar results. However, it has been observed that some Type A subjects do not elevate substantially over baseline levels, in experimental conditions where such response would be expected. As noted earlier, Type A persons present themselves as being under control and capable of handling any crisis. In order to cope with laboratory manipulations of stress Type A's, under some conditions, appear to deny subjective states which might interfere with task performance, and conjointly are less physiologically reactive than Type B's.

It might be expected, that under certain emotional situations Type A's would attempt to conceal affective reactions

to maintain their sense of control, for example, when relating stressful incidents in their lives that gave rise to fear or distress. While describing the affect-laden nature of the situation, they may show few outward signs of emotion. To the extent that they are effective in concealing such affect, we might expect a concomitant dampening of physiological arousal. This latter expectation derives from research showing that lower levels of facial expressiveness are accompanied by lower levels of physiological activity and subjective reports of affective experience. Whereas low reactivity is expected from Type A's during fear or distress, opposite reactions are expected during incidents involving anger. As noted earlier, Type A's are partially defined by a propensity for expressing anger and aggression. They do not attempt to conceal such affect, since it is not viewed by such individuals as a threat to their self-perceptions of being in control.

The foregoing expectations were tested in a laboratory experiment in which A and B subjects (classified by a structured interview) were asked to think of a situation that made them (angry, fearful, distressed) in the past.

STATEMENT OF HYPOTHESES

1. It was hypothesized that when relating incidents involving fear and distress, Type B's would be more physiologically reactive than Type A's.
2. It was expected that Type A's would attempt to conceal their affect, reflected by suppressed facial expressiveness, to a greater extent than Type B's when relating situations that engender fear and distress.
3. It was hypothesized that Type A's would be more physiologically reactive than Type B's when relating incidents involving anger.
4. It was also expected that when relating incidents involving anger, Type A's would be more facially expressive, than Type B's, thereby freely expressing their affect.

METHOD

Selection of Subjects

The participants in this study were paid male volunteers from the New York City Transit Authority (TA) and the New York City Police Department (PBA). Although most were patrolmen, they represented a variety of transit and police occupations, including detectives, computer personnel, bus drivers, motormen, and administrative personnel. Participants were recruited by letter, through their respective unions. This letter informed them that heart disease is a major cause of death in this country. Also, that many research projects are being undertaken to learn more about the causes, treatment, and prevention of cardiovascular illness. This particular study being concerned with life-style, biological, and chemical factors causing heart disease. Potential participants were told in the letter that the study is endorsed, in the case of the PBA, by the union, and for the TA by management, all information collected will be kept confidential. Each participant could request that his personal physician receive a copy of his medical tests.

To qualify, participants must have been men between the ages of 25 and 55, and without a history of heart disease, high blood pressure or diabetes. The recruitment letter outlined all procedures volunteers were subject to, including blood sampling. They were then asked to phone in and schedule an appointment for medical screening. During this initial phone contact a research assistant conducted a preliminary screening concerning questions of age, presence of cardiac related diseases, diabetes, smoking behavior and current medications.

All volunteers were given a medical screening and classified for behavior patterns A or B, several weeks (8) to months (12) prior to the experiment. This delay, between screening and experiment, was needed to allow sufficient time for a thorough evaluation of the medical screening and behavior pattern classification. Participants were paid at the rate of $5.00 per hour for the time they spent in the study. The medical screening and behavior pattern classification occured on the same day, usually taking 1.5 hours. The subsequent experimental session was scheduled separately, and took about 3 hours to complete. Volunteers were selected to be experimental subjects on a first come first served basis, according to their behavior pattern classification and medical screening. The experimenter was blind to this procedure. Subjects were excluded from the study if they had CHD, abnormal ECGs, diabetes, blood pressures of

145/95 mmHg or higher, elevated serum cholesterol (i.e., more than 275mg/100ml), or reported smoking more than one pack of cigarettes per day.[1]

The sample of 42 cases in the study (21 Type A's and 21 Type B's), consisted of 7 TA men (1 A and 6 B's), and 35 PBA (20 A's and 15 B's). The specific behavior pattern classification of the sample contained 9 A1's, 12 A2's, 4 X's and 17 B's.

Demographics

The sample ranged in age from 31 to 60 years, with the mean being 39.4 yrs. (SD = 5.9). The average age of the A's was 39.0 yrs. (SD = 6.0), and of the B's 39.7 yrs. (SD = 6.0). The average height and weight of the Type A's was 70.9 in. (SD = 2.0) and 191.0 pounds (SD = 22.5). The Type B's had a mean height and weight of 70.9 in. (SD = 2.1) and 191.8 pounds (SD = 24.4).

Seventeen A's and 17 B's were nonsmokers. Three Type A's and 4 Type B's smoked less than a pack of cigarettes per day. Only 1 Type A smoked more than a pack of

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[1] The sample contained one Type A with a cholesterol level of 276 mg/dl. Four A's and 4 B's reported smoking a pack of cigarettes per day or less. None of these persons had smoked for 4 hours prior to the experimental session. Moreover, no subjects in the sample had taken any medication or drugs, drank coffee, tea, cocoa, soft drinks containing caffeine, or alcohol related beverages 4 hours prior to the experiment.

cigarettes a day.

Sixteen Type A's and 16 Type B's are White. Two A's and 5 B's are Black. The three Hispanics in the study are Type A.

The average screening blood pressure (BP) of the sample was 117.8/78.3 (SD = 11.0/7.8), N = 42. The Type A's had a mean BP of 120.2/78.5 (SD = 9.9/8.4), n = 21. The mean BP of the Type B's was 115.5/78.1 (SD = 11.8/7.5), n = 21. The mean screening BP for African-Americans was 116.4/80.3 (SD = 10.7/5.7), n = 7; Black Type A's, 124.5/81.5 (SD = 4.9/4.9), n = 2; Black Type B's, 113.2/79.8 (SD = 11.0/6.5), n = 5. The Hispanic-Americans, all Type A's, had a mean BP of 113.7/80.7 (SD = 5.5/1.2), n = 3.

Of the subjects who reported a family history of hypertension (either father, mother or both have abnormally high blood pressure - HBP), 6 are Type A and 5 are Type B, N = 42. A further breakdown of this group reveals that 7 are White, 2 Black and 2 Hispanic. A much higher incidence for a family history of CHD was reported (either father, mother or both, have or had MI, Angina or generalized heart problems singularly or in combination with HBP); 9 Type A's and 12 B's, N = 42 of these 17 are White, 3 are Black and 1 Hispanic. Only 7 subjects, of the 21 who reported a family history of CHD, also had a family history of HBP, 4 A's and 3 B's. The relatively high incidence of family histories for CHD among the subjects (50%), may reflect a self concern that could have prompted them to volunteer for the study.

The educational levels of both Type A's and Type B's were approximately equivalent, (see Table 1).

TABLE 1

Subject's Educational Levels

	Some High School	High School Graduate		
Type A	1	6		
Type B	1	7		

	Some College	College Graduate	Post Graduate
Type A	6	6	2
Type B	5	7	1

Cardiovascular Screening

Upon arrival at the C.U.N.Y. Graduate Center, volunteers were greeted and escorted to the medical screening room. Ms. Kathleen Kehoe, the medical technician (MT) conducted the screenings, which included an electrocardiogram (ECG), urine test for diabetes, blood pressure, height and weight measurements, and personal and familial medical histories. Analysis of the blood samples taken during the screening for

serum cholesterol level was conducted by Laboratory Procedures, Inc., (King of Prussia, Pennsylvania). Electrocardiographic evaluations were conducted by Dr. Lawrence R. Krakoff and his colleagues at the Mount Sinai School of Medicine. Dr. Krakoff also acted as medical supervisor of the research and oversaw the blood collection protocol. ECG abnormalities were reported to the volunteers' personal physicians, not directly to the individuals involved. These steps were taken to enable their physicians to check the results, repeat tests if necessary, and explain the findings to their patients.

Behavior Pattern Classification

The behavior pattern classifications followed criteria established by Friedman and Rosenman (Rosenman, 1978; Rosenman, et al., 1964). At the end of the cardiovascular screening, subjects were taken to a separate room where Type A or Type B behavior was determined by a structured interview (see Appendix A). All interviews were recorded on audio cassettes. Interviewers completed their behavior pattern assessment immediately after the subject left the room. Interview tapes were subsequently reviewed by an auditor who made an independent behavior pattern assessment.[2] Cases

[2] Laura Erlanger and Jean Landeau were the primary Pattern A interviewer-auditors. Richard Contrada and Barry Snow also served as auditors. Mses. Erlanger and Landeau were

classified as Type X (n = 4) were included in the Type B category. The degree of agreement between interviewer and the auditors was 91% for the cases reported in the final sample. Disagreements were resolved by a third auditor.

Both the medical technician and experimenter were unaware of the subject's final behavior pattern classification.

Measurement of Blood Pressure and Heart Rate

In the experimental setting, measurements of systolic and diastolic blood pressure (DBP) were obtained automatically with a Roche Arteriosonde 1216 (Hoffmann - La Roche, Inc., Cranbury, New Jersey). The transducer and compressing cuff were placed on the subject's non-preferred arm (the arm that was not used for blood sampling). The transducer ultrasonically detects arterial wall motion. Arteriosonde measurements correlate highly with blood pressure taken by intra-arterial catheterization (Tahir & Adriani, 1973).

At the beginning of the experimental session, manual blood pressure readings were taken to assure that the Arteriosonde cuff was accurately placed over the brachial artery. The cuff was connected to a monitor, located in an adjacent observation room, and attached by a cable extended through a sealed conduit in the wall. The Arteriosonde monitor, which

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trained by Theodore M. Dembroski of Eckerd College. Drs. Contrada and Snow were trained by Ray H. Rosenman of Mount Zion Hospital and Medical Center.

registers SBP and DBP readings on a mercury column, contains a special indicator which signifies when a displayed reading might be in error. Under these circumstances the measurement was usually repeated, or if it could not be repeated in time, then the suspect reading was deleted. Less than 1% (.5) of all experimental readings had this problem.

Heart rate, measured as digital pulsation, was monitored by a photocell plethysmograph attached to the index finger of the subject's preferred hand. A digital monitor, in the observation room, displayed heart rate (HR) in beats per minute (bpm). The plethysmograph and monitor were also connected by a cable through the wall. This apparatus was constructed by Mr. William Isecke, the staff engineer for the laboratory.

Visual observation of the subject and video tape recording of facial expressions were made using closed-circuit TV.

Blood Collection and Measurement of Plasma Catecholamines

Plasma catecholaminergic reactivity is the measure of choice for short term laboratory interventions (Mason, 1972). Major changes in the release and reuptake of epinephrine and norepinephrine (catechclamines) can occur in very short periods of time. Studies of catecholamine reuptake report half-times of about 1.2 mins. for E, and 2.5 mins. for NE (LaBrosse, Mann & Kety, 1961; Silverberg, Shah,

Haymand & Cryer, 1978). Because of the short time spans involved in the neural reuptake and episodic changes of E and NE, it is essential that blood samples are collected within about 60 sec. after an experimental intervention is completed. Blood collection procedures were timed with a stopwatch to insure adherence to this protocol.

Blood samples were collected at designated times during the experimental session, by drawing off blood from an Angiocath Teflon catheter placed in the forearm vein of the subject's preferred arm. This technique, using an indwelling venous catheter, allowed the drawing of serial samples without repeated venipuncture. Initial venipuncture followed by a 30-minute "resting period" permitted sufficient acclimation to alleviate stressor effects that were introduced by the venipuncture (Glass, et al., 1980a; Glass & Contrada, 1984; Lake, Ziegler & Kopin, 1976). Baseline values for plasma epinephrine and norepinephrine were obtained from blood samples drawn at the end of a 30-minute period following venipuncture.

The samples were prepared for plasma E and NE assay by the following procedures. Blood specimens (10 ml) were drawn into plastic syringes, then immediately transferred to chilled glass tubes containing anticoagulants EGTA and glutathione to preserve plasma epinephrine and norepinephrine . The tubes were placed in a refrigerated Triac centrifuge and spun at 3500 rpm for about 10 min. The plasma obtained was

pipetted into glass vials (in triplicate) and frozen at approximately -80°C until transport to the assay laboratories at the Mount Sinai Medical School, New York City, (26 cases), and the Uniformed Services University School of Medicine, Bethesda, Maryland, (16 cases), several months later.

Catecholamine measurement followed a standardized procedure previously reported in the literature (Glass, et al., 1980a, 1980b, 1983). Plasma E and NE was measured by radioassay employing catechol O-methyl transferase, in the presence of ^{3}H-methyl S-adenosyl-methionine (Vlachakis, Ribeiro, & Krakoff, 1978). The reaction products, normetanephrine and metanephrine, were separated by thin layer chromatography and converted to vanillin by periodate oxidation for quantification by liquid scintillation spectrometry (Glass, et al., 1980a). Values of plasma E and NE are expressed as picograms per milliliter (pg/ml). The inter-assay coefficients of variation were 18% and 15% for E and NE, respectively. Intra-assay variation of duplicates (triplicates when necessary) was 10% for E and 9% for NE.

The radioenzymatic technique used at the Uniformed Services University differed slightly from the procedure employed at Mount Sinai (Durrett & Ziegler, 1980). The sensitivity of the assay was 27.2 pg/ml for NE and 13.6 pg/ml for E. The inter-assay variability was 11% for both NE and E.

A number of potential confounding factors must be controlled when measuring catecholamines. These include substances containing caffeine or alcohol, tobacco (nicotine stimulates catecholamine release), medications related to sympathetic-adrenal medullary activity, such as antihistamines, antihypertensives, anticoagulants, steroids and tranquilizers (Frankenhaeuser, 1975; Glass & Contrada, 1984; Lake, 1979; Mason, 1972). When scheduled for the experiment, subjects were instructed not to use antihistamines, caffeine or tranquilizers for at least four hours prior to their session.

Measurement of Facial Affect

Three affective states, anger, fear and distress, were induced through the use of an emotional imagery technique (Scherwitz, Berton, & Leventhal, 1978). Subjects were told to image a situation that led to a specific affect and think about it for a few minutes. Afterwards, they were instructed to talk about the situation they imagined and the feelings they associated with it.

An assessment of subject's affective reactions was conducted using facial expressiveness as the behavioral index. The measurement of facial expressiveness was accomplished through the use of the semantic differential scales (see Appendix D), following procedures similar to those developed

by Hastorf, Osgood and Ono in 1966 (Orr & Lanzetta, 1980; Osgood, 1966). These seven-point scales consist of expressive dimensions of affect (e.g., "excitable-calm), and were rated by four naive judges. To assess the degree to which subjects were actively engaged in the emotion task, a level of involvement scale was also used (see Appendix D).

During the emotion producing phases of the experiment, subjects's facial expressions were recorded on videotape. Subject's speech was deleted from the recordings. One minute segments, corresponding to the interval of highest SBP activity (to ensure S's involvement), were labeled and rerecorded for the two phases, imaging and relating, of each emotion. The labels, which appeared on the videotape for 30 seconds, contain the subject's identification number, and (in code) the emotion (anger, fear or distress), and phase (imaging or relating) of each segment. Using random assignment, to control sequence and order effects in subsequent rating procedures, these video segments were rerecorded and combined onto 30 minute tapes. The edited videotapes of subject's facial expressions were presented to judges, who then rated the expressive affects they saw according to the semantic differential scale. An attempt was made to have judges rate facial expressions directly for anger, fear and distress. However, the raters were unable to make reliable assessments from the video segments, so only scales validated by previous research were used.

Design of The Study

The study consisted of a 2 x 3 repeated-measures, factorial design: A - B (between factor) by Anger - Fear - Distress (within factor). Type A and Type B men were asked to image and relate past incidents which made them feel angry, fearful and distressed. Each emotion-provoking episode was divided into three phases: Imaging (3 min.), relating (3 min.) and resting (5 min.). Subjects participated in the study individually and were recorded on videotape during the imaging and relating phases of the experiment.

The order in which emotions were presented to the subjects was counterbalanced in a Latin Square design (Lindquist, 1953, p. 258; Winer, 1971, p. 685), with 7 A's and 7 B's randomly assigned to each of the three orders, to control for possible sequence effects (see Figure 1). Subjects completed a practice emotion (fatigue), before engaging in the counterbalanced trials, so they would be familiar with the imagery technique.

I	Anger	Fear	Distress
II	Fear	Distress	Anger
III	Distress	Anger	Fear

Figure 1: Counterbalanced Emotions.

Induction of Affective States by Emotional Imagery

The emotional imagery technique as used in this study was similar to that used in previous psychophysiological research (Carroll, Marzillier, & Merian, 1982; Lang, 1979; Lang, et al., 1980; Singer, 1974). Recent work by Baker and her colleagues (1984) demonstrates that imagery techniques can be used to effectively study Type A behavior and physiological reactivity (Baker, et al., 1984). Conforming to much of the work in this area, subjects were "trained" in the production of imagery immediately before the experimental trials took place. An innocuous affect situation (fatigue) was used at the outset of the experiment to help the subject overcome any inhibitions he might have had about engaging in the imagery task. In order to increase their confidence in performing the task, I personally explained the importance of actually feeling whatever emotion they were asked to experience. It was emphasized that they should try to choose a real situation that led to the requested affect. Subjects were told before the task began that they would be asked to describe the situation that created the affect and the intensity of their feelings. Three affects -- anger, fear and distress -- were used in this study. The specific instructions used for the generation (imaging phase) and expression (relating phase) of emotions were similar to those successfully used in previous Pattern

A research by Scherwitz, Berton, & Leventhal (1978), (see Appendix P).

Dependent Measures

Repeated systolic and diastolic blood pressure, and heart rate measurements were taken every minute during the three phases of each emotion. In addition, blood samples, to be assayed for plasma catecholamines, were drawn after the imaging and relating phases of each emotion. Baseline measurements, prior to the experimental task, consisted of the mean of the last two cardiovascular measures and a blood sample, taken at the end of a 30-minute resting period following venipuncture.

Blood pressure and heart rate reactivity were measured by subtracting appropriate baseline levels from those obtained during imaging and relating phases of the task. Similarly, plasma epinephrine and norepinephrine values derived from samples drawn during the three emotions, were taken as deviations from baseline levels. These plasma catecholamine and cardiovascular change-scores constitute the physiologic dependent measures.

Behavioral dependent measures were used to understand the relationship between physiological responsiveness and affective suppression. An analysis was conducted using the video-tapes of each subject during the imaging and relating

phases of the study. Four female undergraduate students, recruited from Hunter College, served as naive judges. Various studies examining nonverbal behavior and emotion have reported sex differences in accuracy of perception of facial emotion of other persons (e.g. Buck, Savin, Miller, & Caul, 1972). The consensus is that females are more accurate than males in perceiving emotion in others (Schwartz, Brown, & Ahern, 1980). Prospective judges were screened for having had no prior experience serving in psychological research. They were paid $5.00 an hour for their participation. These judges made their ratings, along several expressive dimensions (e.g., "pleasantness-unpleasantness"), without knowledge of the circumstances under which the expressions were obtained (Hastoff, Csgood, & Ono, 1966; Osgood, 1966). The only instructions they received are those contained in the video rating form, Appendix D.

Experimental Protocol

Participants were greeted by the experimenter (E) upon their arrival at the research facility for the experiment proper (see full experimental script in Appendix B). The experimenter escorted the subject to the experimental room and seated him in a reclining lounge chair. The subject was asked to take a semi-supine position in the chair, while the medical technician (MT) took manual blood pressure readings.

Only one person, either E or MT, was in the room with the subject at any time. Following the manual blood pressure readings, the experimenter returned and outlined the general purposes of the study.

The subject was told that researchers are interested in the role that a person's life style, including everyday behavior, plays in the development of heart disease. It was further explained that in order to study the behavior - heart disease relationship under controlled conditions, each participant will work on standard laboratory tasks that "capture the essence of various skills and activities that are important in the daily routines of most people." The experimenter next explained that physiologic indices will be monitored as the subject works on the tasks. The blood sampling procedure was explained at this point, and the subject was asked to indicate his willingness to participate by signing a university-approved informed-consent agreement (see Appendix E).

After the consent form was signed, the MT proceeded with venipuncture (VP) and insertion of the catheter. The Arteriosonde cuff was placed on the arm opposite the catheter. To record HR and BP simultaneously, the photocell plethysmograph was attached to a finger on the same limb that contained the catheter. The subject was instructed to relax, try not to move his arms, and to maintain a semi-reclining position in the lounge chair. Quiet music was played and MT

left the room. The Arteriosonde and photocell plethysmograph were activated five minutes after VP, to acclimate the subject to the cuff's inflation, and to check that the instruments were operating properly. Ten minutes after VP, a 30-min. resting period began, during which time SBP, DBP, and HR measurements were recorded every 2 min. Recordings continued until SBP and DBP values remained relatively constant across 2 successive samples (± 5mmHg). As noted earlier, the average of these 2 readings constituted the SBP and DBP baselines. The same procedure was used for the HR baseline. The MT re-entered the experimental room after baseline recordings were completed and drew the first (baseline) blood sample.

The experimenter returned and gave general instructions for the study. He informed the subject that the medical technician will come in periodically during the session to take blood samples and that the pressure cuff will inflate automatically throughout the session. The subject was told that his description of images will be recorded on tape, but kept in strict confidence. The experimenter continues by stressing the importance of actually feeling whatever emotion the subject was being asked to experience. Subjects were instructed to engage in the task fully, to spend the entire 3 min. allotted to <u>think</u> (image) or <u>talk</u> (relate) about an emotional episode.

The specific instructions regarding the generation (imaging) and expression (relating) of emotional states were pre-recorded and delivered over headphones (see Appendix B).

Following the relating phase of each emotion, the subject was given a 5 min. rest at the end of which, the experimenter began the taped instructions for the next emotion. Throughout the experimental task, SBP, DBP and HR, were measured every minute. Blood samples were drawn after the imaging and relating phases of each experimental emotion (anger, fear and distress).

The experimental session was concluded with a 15-min. rest period, following which there was an interview and debriefing concerning the subjects's reactions to the study (see Appendix C).

METHODS OF ANALYSIS

Mean baseline values of cardiovascular measures and catecholamines, were submitted to a series of t-tests, to ascertain A - B differences. As noted earlier, the cardiovascular and catecholamine data, collected during the experimental trials, were expressed as changes from their respective baselines. Change-scores for the relating phases of each emotion were computed separately.

The principal method of analysis of the change-scores was a 2 x 3 repeated-measures analysis of covariance, with the appropriate baseline values constituting the covariate.

The ratings of facial expressiveness were factor analyzed to express them in psychologically relevant dimensions. Initially, Osgood's (1962) rating instrument was explicitly designed to assess affects being expressed by the face. The procedure proposed here replicates the traditional approach in this area (Hastorf, et al., 1966). The resulting factors (see Appendix F) were submitted to both a MANOVA, and separate correlation analyses using residual values of the physiological dependent measures. These analyses were used to assess expectations regarding the tendency of Type A's to inhibit emotional affect, thereby dampening

physiologic activity. As above, these analyses were computed separately for the relating phases of each emotion.

The computation of ANCOVAs, ANOVAs, and MANOVA were conducted using the BMDP computer program accessed through SAS, all other analyses were computed with SAS (Dixon, Brown, Engelman, Frane, Hill, Jennrich, & Toporek, 1985; Helwig & Council, 1979).

RESULTS

Physiological Baseline Measures

Mean baseline values for the five physiological variables are presented in Table 2. Separate t-tests were conducted for each physiological measure to reveal any A/B differences. None of the alpha levels showed an association between behavior pattern classification and baseline levels of the five physiological dependent variables (p's >.45 except HR where p =.09).

Upon inspection, baseline values for systolic blood pressure may seem unusually low when compared to resting levels in other studies which range from 116 to 120 mmHg, however this is not the case (Glass, et al., 1980a; Scherwitz, et al., 1978). While it is true that baseline SBPs, for Type A's and Type B's, were 5 or more mmHg below those previously taken, during the cardiovascular screening, several factors may account for this decrease. First, there is usually a large degree of variability associated with the cardiovascular and catecholamine measures used in experimental research, and because of this there need not be a direct correspondence between screening and baseline measurements

(Williams, 1986). An example of the factors that can influence the variation of physiological measures are anxiety (psychological) and somatic changes throughout the day (circadian rhythms). Another study, also conducted at the CUNY Laboratory of Biobehavior, which used subjects that were an age and occupational cohort of participants in this study, similarly reported low baseline SBPs (e.g., 111 mmHg) (Glass, et al., 1980b). Consider that the men in these studies underwent baseline measurements during their second visit to the laboratory, and were already somewhat familiar with the technician and blood pressure procedure. Consequently, subjects in these studies may not have been as anxious during baseline as those in other experiments. The nature of the setting may have also influenced the relatively low baseline results. Remember that subjects were reclined in a semi-supine position listening to quiet music. The relaxing mood of the experimental room may also have lowered baseline measures, especially pertaining to the subject's reclined position. Previous research has established that postural changes between supine, sitting or standing conditions can affect catecholaminergic reactivity and possibly blood pressure (Glass, et al., 1983; Lake, et al., 1976). Catecholamines increase substantially when a subject moves from a supine to a seated position (Glass, et al., 1983; Lake, 1979). Since subjects in this study participated in a reclined position, postural effects may have contributed to their low baseline values.

TABLE 2

Mean Baseline Values for Each Experimental Group

Measure	n[a]	Type A	(SD)	Type B	(SD)
SBP (mmHg)	21	112.3	(10.8)	110.6	(9.5)
DBP (mmHg)	21	74.7	(5.7)	74.8	(6.8)
HR (bpm)	21	67.5	(8.8)	62.5	(9.6)
Plasma NE(pg/ml)	20	245.5	(130.1)	218.9	(106.8)
Plasma E (pg/ml)	20	35.2	(24.9)	37.0	(46.2)

[a] See footnote.[3]

Cardiovascular Measures

Reactivity from Baseline

Analyses of covariance of change-scores for the cardiovascular measures were conducted for each relating phase of the three emotions (anger, fear and distress). In each analysis, the independent variable was behavior pattern A or B, and the covariate was the appropriate baseline value. The results revealed a reliable A/B effect for systolic

[3] Two cases, one Type A and one Type B, were unavailable for catecholamine analysis because of technical difficulties connected with the blood sampling and assay procedures.

blood pressure, $F(1/39) = 4.68$, $p < .04$ (see Table 3). Surprisingly, Type B's had elevated SBP more than Type A's. Furthermore, contrasts analyses (Weiner, 1971, pp. 529-532) indicated that the difference between Type A's and Type B's, suggested by the A/B x Emotion interaction was pronounced in distress, $F(1/58) = 7.83$, $p < .01$.

Covariance analysis for the remaining cardiovascular measures failed to reveal any other reliable A/B effects (p's $> .15$).

TABLE 3

Systolic Blood Pressure: ANCOVA with Contrasts for Mean Changes from Baseline (in mmHg)

Relating	Type A	(SD)	Type B	(SD)
Anger	+18.8	(12.5)	+23.9	(11.9)
Fear	+19.4	(12.2)	+26.5	(11.4)
Distress	+15.5	(12.0)	+26.0	(10.0)

A/B Main Effect	$F = 4.68$	$p = .04$
Emotions Main Effect	$F = 1.85$	n.s.
A/B x Emotions	$F = 2.63$	$p = .08$

A/B Contrasts

Anger	$F(1/54) = 1.75$	n.s.
Fear	$F(1/54) = 3.64$	n.s.
Distress	$F(1/54) = 7.83$	$p < .01$

Analyses of variance testing cardiovascular recovery values found that SBP and DBP did not return to baseline levels after the experimental trials, $F(1/40) = 21.2$, and $F(1/40) = 24.0$, p's $<.001$, both for Type A's and Type B's. However, heart rate did return to baseline values for both behavior types (p's $>.11$).

Resting Level Reactivity

When working with physiological measures, inevitably the question of a ceiling effect or reactivity plateau must be addressed. Remember in this study a resting phase was given between each experimental task (emotion) to control for plateaus in physiologic elevations. Tests of resting level values were conducted to see if this experimental control was working.

Separate t-tests were conducted on each resting level subsequent to an emotional task, to reveal A/B differences for the cardiovascular measures. No reliable differences were shown for any cardiovascular measure during any resting phase (p's $>.25$, except for SBP-Distress where $p > .08$).

Change-scores were computed from the prior resting phase, for analyses of covariance. Consequently, the appropriate prior resting level was used as the covariate. For any given subject the prior resting phase of a specific emotion might not be the same as that of another person because

of the counterbalancing of emotional sequences (see Figure 1). The independent variable was behavior pattern A or B. Results of these analyses showed an interaction effect between behavior pattern and emotions for systolic blood pressure, $F(2/77) = 5.17$, $p < .01$, indicating the absence of a reactivity plateau on this measure for both A's and B's (see Table 4). An A/B reversal similar to the baseline analysis occurred during distress where B's were higher than A's. No other reliable results were found for these analyses (p's $> .10$).

TABLE 4

Systolic Blood Pressure: ANCOVA with Contrasts

for Mean Changes from Resting Level
(in mmHg)

Relating	Type A	(SD)	Type B	(SD)
Anger	+12.6	(10.3)	+10.7	(10.1)
Fear	+13.9	(9.7)	+13.0	(7.0)
Distress	+7.8	(8.3)	+13.8	(10.6)

A/B Main Effect	$F = 0.11$	n.s.
Emotions Main Effect	$F = 2.35$	$p = .10$
A/B x Emotions	$F = 5.17$	$p = .01$

A/B Contrasts

Anger	$F(1/115) = 0.57$	$F < 1$
Fear	$F(1/115) = 0.16$	$F < 1$
Distress	$F(1/115) = 4.09$	$p < .05$

Analyses of variance for cardiovascular and resting levels revealed reliable differences ($p < .01$) on all measures for all emotions except heart rate during distress ($F(1/40) = 2.77$, $p < .10$). The interpretation of this and other resting level analyses, along with the baseline-recovery ANOVA, indicate that once participants were aroused they never returned to baseline levels (except HR after distress and during rest or recovery). These reactivity elevations persisted throughout the study. However, SFP did not plateau, indicating that the resting period provided some recovery from the prior emotional trial.

Catecholamines

Analyses of covariance for the catecholamines paralleled the method used for the cardiovascular measures. This statistic revealed a significant A/B main effect for norepinephrine $F(1/37) = 6.98$, $p < .01$ (see Table 5). Type B's had consistently higher elevations than Type A's, and these differences were significant during anger and fear.

Analyses of variance found that norepinephrine levels dropped slightly below baseline values during recovery, for Type A's and Type B's, $F(1/38) = 3.69$, $p < .06$. Epinephrine levels during recovery returned to baseline values.

No other results were found for these measures.

TABLE 5

Norepinephrine: ANCOVA with Contrasts

for Mean Changes from Baseline Level
(in pg/ml)

Relating	Type A	(SD)	Type B	(SD)
Anger	+49.4	(64.5)	+117.2	(96.3)
Fear	+17.4	(106.2)	+98.0	(114.8)
Distress	+33.5	(94.2)	+95.0	(85.2)

A/B Main Effect	$F = 6.98$	$p = .01$
Emotions Main Effect	$F = 1.79$	n.s.
A/B x Emotions	$F = 0.24$	n.s.

A/B Contrasts

Anger	$F(1/73) = 5.11$	$p < .05$
Fear	$F(1/73) = 5.86$	$p < .05$
Distress	$F(1/73) = 3.67$	$p < .10$

Behavioral Measures

Separate factor analyses, using varimax rotations, were conducted for the semantic differential scales in each experimental phase. Initially, the anger-imaging phase yielded reliable positive loadings on three factors (see Appendix F). Two of these factors directly replicated Osgood's affective dimensions and the third factor loaded reliably on scales selected by Hastorf, Osgood and Ono (1966) to represent Control. Subsequently, similar loadings

were found during relating for all three emotions when the third factor was forced. Following Osgood, it was decided that the three factors representing dimensions of affective behavior, would be represented by combining the following scales: Factor I, Pleasantness (pleasant, good, sociable, and agreeable); Factor II, Activation (active, excitable, and emotional); Factor III, Control (tight, hard, and closed).

Multivariate analysis of variance, testing resulting factor scores, during the relating phases, found reliable main effects for the control dimension (see Table 6). Univariate A/B main effect $F(1/40) = 5.03$, $p = .03$, and A/B simple effects for control show reliable differences in anger $F(1/40) = 4.26$, $p = .05$, and fear $F(1/40) = 5.78$, $p = .02$, similar to the norepinephrine effects. Type B's were rated higher on the control dimension for all emotions, than were Type A's (a lower score on the control factor indicates a greater amount of that dimension, see scale Appendix D).

It is perplexing that Type B's who were more physiologically reactive should rate higher on Control. According to Zuckerman et al. (1981), the higher reactive Type B's should show more facial arousal, and in fact they do when the individual scale items that comprise the Control dimension are examined (tight-loose, hard-soft, and closed-open). Osgood (1966, pp. 21-23), in the first of a three paper series, delineated the problem of naming the third affective

TABLE 6

Video Facial Affect: MANOVA

for Mean Semantic Differential Factor Scores

RELATING

Semantic Factors	ANGER Type A	ANGER / B	FEAR Type A	FEAR / B	DISTRESS Type A	DISTRESS / B
Pleasant	4.5	4.7	4.7	4.7	4.6	4.7
(SD)	(0.5)	(0.5)	(0.5)	(0.3)	(0.5)	(0.4)
Active	3.8	3.8	3.6	3.7	3.7	3.7
(SD)	(0.8)	(0.7)	(0.7)	(0.5)	(0.8)	(0.6)
Control	4.0	3.7	4.0	3.6	4.0	3.8
(SD)	(0.1)	(0.5)	(0.5)	(0.5)	(0.5)	(0.4)

Multivariate Effects

A/B Main Effect	$F = 0.84$	$F < 1$
Emotions Main Effect	$F = 0.34$	$F < 1$
A/B x Emotions	$F = 0.57$	$F < 1$
Factors Main Effect	$F = 40.84$	$p = .03$
A/B x Factors	$F = 2.28$	n.s.

Univariate Effects

CONTROL

A/B Main Effect	$F = 5.03$	$p = .03$
Emotions Main Effect	$F = 2.62$	$p = .08$
A/B x Emotions	$F = 1.36$	n.s.

A/B Simple Effects

Control/Anger	$F(1/40) = 4.26$	$p = .05$
Control/Fear	$F(1/40) = 5.78$	$p = .02$
Control/Distress	$F(1/40) = 2.25$	n.s.

dimension - Control. An issue among investigators has been whether a minimum of three dimensions are necessary to represent emotion, or whether two dimensions, namely Pleasantness and Activation, are sufficient. There is agreement in the literature on Pleasantness being one dimension of emotion, and nearly complete agreement on Activation (Osgood, 1966). The third dimension, Control, if used at all, seems open to debate. One of the experiments reported by Hastorf et al. (1966) showed that the scales usually representing Control were used to indicate Pleasantness, necessitating tight - hard - closed being Unpleasant. In the aforementioned study, the scale intentional-unintentional, was a better representation of the Control dimension. Upon reevaluating the tight - hard - closed scales, Osgood concluded that they describe a physical representation of the face, which could be interpreted as unpleasant affect (Osgood, 1966, p. 23). Data in the present study is congruent with Osgood's reinterpretation of the Control dimension to one of Unpleasantness.

To check Osgood's contention further, intercorrelations were performed on factor scores of the three dimensions, Pleasantness, Activation and Control. A reliable negative correlation ($p = .03$) was found between Pleasantness and Control. These opposing factors were combined into a new factor, Unpleasantness. An analysis of variance was conducted, using the new dimension Unpleasantness as the dependent

measure, for the three emction trials during relating. As in prior analyses the independent variable was behavior pattern A or B. The results showed a reliable A/B effect for Unpleasantness, $F(1/40) = 4.34$, $p = .04$. As with the original Factor III - Control (which was renamed Unpleasantness), Type B's evidenced more unpleasant affect than Type A's during the emotional trials.

Few consistent results were found when the three affective dimensions were correlated with physiological measures. Unpleasantness on Factor I was correlated ($p < .03$) with SBP and DBP during fear and distress for Type B's. Whereas, Type A's showed reliable correlations ($p < .03$) between pleasant affect on Factor III and SBP, for all three emotions. However, intercorrelations among the five physiological measures showed that these responses were dissociated during the experimental trials. The expected response pattern would have systolic blood pressure, heart rate and epinephrine elevating together, with diastclic blood pressure paralleling changes in norepinephrine.

To test the assumption that Type B's, or Type A's, might have been more involved in performing the emotional imagery tasks, ratings were made, in each phase of the study, concerning participants' intensity of affect and facial expression (see Table 7, and Appendix D).

TABLE 7

Mean Level of Involvement from Video Ratings

Emotion	n	Type A	(SD)	Type B	(SD)
Anger	21	4.4	(0.6)	4.3	(0.5)
Fear	21	4.5	(0.7)	4.4	(0.4)
Distress	21	4.4	(0.7)	4.5	(0.7)

T-tests on level of involvement showed no differences between Type A's and Type B's. The scale means of both groups varied between moderate involvement, moderate intensity and involved with emotion, intense expression.

One would expect the Osgood scale and the intensity measure to reflect each other, but they did not. Since the level of involvement scale was unvalidated, possibly it is a less discriminating or unreliable measure.

No other results were found for the behavioral measures.

DISCUSSION

This study explores the differential impact cognitive imagery of strong emotional situations has upon cardiovascular and plasma catecholamine activity in Type A and B individuals. Reliably greater elevations in systolic blood pressure and plasma norepinephrine were found for Type B's compared to Type A's. The increased reactivity of Type B's was consistently higher for each emotion, although not always significantly so, than that of Type A's. Conversely, diastolic blood pressure, heart rate and epinephrine did not discriminate A and B subjects. Contrary to this finding previous research has shown that Type A's are usually more physiologically reactive than Type B's (Baker, et al., 1984; Dembroski, et al., 1977, 1978, 1979; Glass, 1977b; Glass, et al., 1980, 1983; Rosenman, 1978). However, the present finding is not completely unusual as other studies have also found A/B reversals (Contrada, et al., 1982; Glass, 1977b; Glass & Contrada, 1984).

The first hypothesis, that Type B's would be more physiologically reactive than Type A's when relating incidents of fear or distress was confirmed. Type B's showed higher elevations in SBP during distress and in NE during fear than

their Type A counterparts. Furthermore, mean changes for both SBP and NE were higher for Type B's during all emotions, including anger, with a reliable effect occurring for NE contrast during anger. This finding leads to a rejection of the third hypothesis, that Type A's would be more physiologically reactive when relating incidents involving anger.

Interpretation of the behavioral measures indicates that, during experimental trials, Type B's showed more facial arousal, as measured by greater ratings of unpleasant affect. Unpleasant affect for Type B's was correlated with increased SBP during fear and distress. This finding confirms Zuckerman's contention that higher levels of facial expressiveness are accompanied by increased physiological arousal (Zuckerman, et al., 1981). The expectation, expressed by the second hypothesis, that Type A's would attempt to conceal their affect by suppressing facial expressiveness when relating fearful and distressing situations, was supported by lower ratings of unpleasant affect and lessened physiologic arousal. This finding held for all emotions, and the fourth hypothesis that Type A's would be more facially expressive when relating incidents involving anger, than Type B's, was rejected.

While reverse A/B effects during two of the three emotions were expected, these expectations were primarily based on the predicted reactions of Type A's. However, the actual findings address other response categories in addition to those of Type A, and as such, require examination.

One of the difficulties in interpreting the results of this study is that few differential effects were found. Also, these findings did not conform to previous studies of Type A behavior which usually focused on performance outcomes during a variety of Type A relevant situations, i.e. competitiveness and time-urgency (Glass & Contrada, 1984; Matthews, 1982).

This study certainly presented participants with a unique psychosocial situation, that being the redintegration of actual emotional experiences while catheterized and monitored for physiological responses. At this juncture it is difficult to evaluate what effect the combination of imaging and measurement techniques had on experimental results. It seems that the results of this study may be attributed to situational specificity as well as behavioral characteristics of the participants. The notion of situational specificity lends itself to other interpretations of the results. Remember, the present study did nct restrict subjects to Type A relevant events. Subjects were allowed to freely choose the situation they applied to an emotion they were asked to image. Although these subjects could not dictate which emotion to image, they nevertheless could control the experimental situation by deciding which event they would relate to a given emotion. As such the imaging task was not an environmentally imposed demand, and in this respect was different from previous research. This controlable aspect

of the experimental setting may have been important for Type A's, leading them tc select an emotional event that allowed them tc remain in control of themselves, the situation, and in effect their reactivity. After all, an experimental session may be viewed as a personal evaluation, and Type A's propensity for control during such situations is a characteristic by which they are defined (Jenkins, 1975).

Other explanations may also account for Type A's responses. Possibly Type A's did not elevate during anger, when increased reactivity was expected, because A's only exhibit anger reactions in appropriate situations having demand conditions such as threat or challenge (Glass, 1977b; Rosenman, 1978). The experimental room had the appearance of a clinical setting, in an attempt to reduce the anxiety induced by catheter insertion. Although the room itself was not part of an experimental manipulation, Type A's may have perceived and used this setting differently than Type B's. It is possible that Type A's experienced relief when relating their emotions, and did not elevate because increased reactivity is not the expected response in this situation (Williams, 1986). The only measure remotely addressing this issue found that A's and B's performed emotional tasks with the same level of intensity, when increased intensity during anger was expected for Type A's.

An anomaly reported earlier was that the physiological effects were dissociated with each other. Usually cardio-

vascular and neuroendocrine reactivity present distinct patterns of response, i.e. elevations in systolic blood pressure parallel increases in heart rate and epinephrine. These responses are mediated by the sympathetic-adrenomedullary (SAM) system and may be influenced by physical, cognitive or situational events (Mason, 1972; Lacey, Kagan, Lacey, & Moss, 1963). However, Lacey (1967) emphasizes that SAM responses are frequently disscciated and should not be considered unusual, because other unnoticed patterns may be transpiring.

The existance of several stress response patterns is suspected by theorists, although at present only two have been well documented (Williams, 1986). These patterns of reactivity, currently designated Pattern 1 and Pattern 2 by Williams (1986), are defined by distinct cardiovascular and neuroendocrine components. Both patterns have characteristic hemodynamic markers, these being vasodilation (followed by increased cardiac and neuroendocrine output) for Pattern 1 and vasoconstriction indicative of Pattern 2 (Williams, 1986; Williams, et al., 1975). Research conducted by Williams and his colleagues showed that Type A's evidenced more Pattern 1 reactivity during a mental arithmetic task than Type B's (Williams, et al., 1982). However, when presented with a reaction time task, Type A responses were quite different than expected. First, most measures did not distinguish between Types A and B. Second, in contrast to

the cognitive task Type A's did not produce a clearly differentiated pattern of responses, but did show elevations of testosterone that previously have been associated with Pattern 2. Williams work points out that different situations evidence different patterns of physiological response. Also, that it is possible to deduce the occurance of a prevalent response pattern if measurement of a known contributor is available. In relation to the present study, possibly no response patterns were found because the experimental situation was unique and traditional measures could not reveal ongoing patterns. For instance, measures that were not used in the current study, but used by Williams and others to reveal reactivity patterns, were forearm blood flow, forearm vascular resistance, plasma cortisol, prolactin, growth hormone and testosterone. These and many other measurements can and have been used in stress related research (Frankenhaeuser, 1975; Herd, 1986; Mason, 1972; Obrist, 1973). Obviously, for practical reasons, any single study must limit itself to use only a few of the known measures. Consequently, the present study only used the prevailing measures associated with Pattern A research.

Thus far, three mediating factors which probably contributed to experimental results have been proposed, these being situational specificity, control and suppression for Type A's. Type B's, however, did not evidence the aforementioned mediating factors as did Type A's. It should be

remembered that Type B's, who were high on unpleasant affect resulting from emotional imagery, were also high on arousal. One explanation for this response sequence suggests that a cognitive mediating factor, termed effort, is responsible for the control of psychophysiological arousal and neuropsychological attention, such as imagery or affect (Pribram, & McGuinness, 1975). In other words, the more Type B's became engaged in emotional tasks the more they experienced unpleasant affect and through their expenditure of cognitive effort to reduce unpleasantness they became more highly aroused. Of course, in the absence of supporting data, this scenario is speculative. Other psychological factors or a combination of factors could also account for Type B reactions, such as active coping strategies, intrapsychic defense mechanisms, or even cynical hostility. Any of these explanations applied to Type B's responses is consistent with research describing factors which elicit increased physiological reactivity (Mason, 1972; Williams, 1986).

In the present study, a series of situations was constructed to engender psychological stress without requiring performance outcomes. The combined psychological and physiological techniques, used here for the first time in a behavior pattern study, seemed to work as intended. Current results indicate that Type A's propensity for anger was either overestimated or Type B's capacity for this affect greatly underestimated. It would seem that the latter was

the case. As Matthews (1982) has pointed out, very little is known about the psychological characteristics of Type A individuals, and virtually nothing is known concerning Type B's. What is needed in the future is an exploration of intervening psychological processes that contribute to Type A and B behaviors. Such as, given similar situations, what are Type B's cognitive responses in addition to their physiological responses (i.e. anger imagery)? Already factors such as cynical hostility and generalized physiological reactivity play an increasing role in describing coronary proneness (Smith & Frohm, 1985; Williams, 1975, 1985; Williams, et al., 1985). Other psychological factors, presently unresearched, associated with a Type B typology independent from a Type A continuum of behaviors, must also be investigated. What seems to be emerging from the results of this and other studies is that the Type A and Type B constructs represent different behavioral characteristics rather than a continuum of behaviors (Contrada, et al., 1982; Glass, 1977b). While it is true that most outwardly observable Type B behaviors are generally opposite those of Type A, current findings imply these may only be surface characteristics, and more substantive psychological differences have yet to be defined (Matthews, 1982). For example, Glass (1977b, 1982) has suggested that Type A's are persons who anticipate both challenging and threatening or potentially harmful situations, thereby potentiating physiologi-

cal reactivity and subsequent CHD. Type A reactions in laboratory settings strongly support this contention, but little is known about Type A attributions in frequent everyday occurrences. The theory that people move along a continuum of behaviors, represented at the extremes by Patterns A and B, dependent upon their psychological disposition under particular situations, may be too simplistic. Rather, both behavior types may exhibit separate patterns of previously unrecognized responses under many commonplace situations which are typically not Type A relevant. Possibly, Type A's also anticipate threat or challenge in situations where neither exists. When Type A's finally perceive the nonstressful nature of the situation, their physiological reactivity has already begun, and cannot be abated by encountering an appropriate stressor. In effect, under nonthreatening everyday conditions Type A's may stress themselves needlessly. It is this consistent reactive - nonreactive behavior that is presumed to increase Type A's risk for CHD (Glass, 1977b, 1982). No doubt, Type B attributions to similar situations differ from those of Type A. In light of foregoing research and current questions, clinical coronary prone behavior must be more than the typical Type A characteristics.

Individual's perceptions of emerging situations, coping styles and defense mechanisms, undoubtly contribute to their resulting behavior patterns, yet all have been understudied.

Future research in this area should investigate these and other factors that could influence coronary prone behavior. Even though the results of this study were different from what was expected, it is still possible that emotional imagery is a viable technique for tapping everyday experiences related to at-risk behavior. The advantage of imagery techniques is that they impart ecclcgical validity without placing the subject in actual situations that could be dangerous or unethical. Admittedly, imagery techniques present potential methodological problems pertaining to experimenter control and subject involvement. However, I think these problems can be overcome, and will present some possible solutions.

Remember that not all Type A's develcp CHD and conversely, some Type B's are also affected by this disease. Considering that few new factors have been fcund which contribute to the pathogenesis of CHD, ccntinued experimentation with the coronary prone behavicr pattern and psychological stress could produce useful results. For these reasons, non-Type A relevant emotional situations, e.g. fear and distress, that promote stress may yet reveal additional psychosocial or cognitive factors involved in the development of CHD. Admittedly, it will be important to take steps to minimize the exercise of control by Type A's during the imaging situation. However, experimenter control may be maintained by the use of specific pre-selected stan-

dardized scenarios, as the imagery task, along with experimental manipulation checks.

In order to better understand the results, further work should be focused on specific non-Type A relevant situations that lead to particular emotions. For example, subjects could be screened prior to the experiment for different types of recent experiences that resulted in emotional reactions. Subjects will be asked to rate these events for importance and intensity. With this information, standardized or individualized scenarios that have a high likelihood of paralleling subjects' actual experiences would be selected. The resulting series of situations would be presented to subjects as imaging tasks, requiring them to account for their perceptions of the situation, anticipation of possible outcomes, and their resulting behavior. A manipulation check should be performed after each imaging task, consisting of self ratings of how deeply each subject felt the emotion, and for this person, how strong an example was this emotional event. Preparatory behavioral and medical screening would be the same as the present study. Standarized measures of coping and defending should be included in each experimental trial. Other factors that should be considered are an index of hostility, individual attributions about time and punctuality, frequency of subjects actual emotional experiences, and physiological measures including indicators of vasodilation and vasoconstriction.

The expected results of this study would identify non-Type A relevant situations which potentiate physiolcgical reactivity, describe cognitive mediators of behavioral and physiological responses, and hopefully detail specific descriptors of Type B behavior. For the present it remains for future research to continue to delineate and explain relevant psychosocial and environmental factors associated with coronary prone behavior.

Appendix A

BEHAVIOR PATTERN INTERVIEW

INTRODUCTION: "I would appreciate it if you would answer the following questions to the best of your ability. Your answers will be kept in the strictest confidence. Most of the questions are concerned with your superficial habits and none of them will embarrass you." (Begin taping now.)

Your code number is _____.

1. May I ask your age? PLEASE.
2. What is your occupation or job?
   a) How long have you been in this type of work?
3. Are you SATISFIED with your job level? (Why not?)
4. Does your job carry HEAVY responsibility?
   a) Is there any time when you feel particularly RUSHED or under PRESSURE?
   b) When you are under PRESSURE does it bother you?
5. Would you describe yourself as a HARD-DRIVING, AMBITIOUS type of man in accomplishing the things you want, getting things done as QUICKLY as possible, OR would you describe yourself as a relatively RELAXED and EASY-GOING person?
   a) Are you married?

b) How would your WIFE describe you -- as HARD-DRIVING and AMBITIOUS or as relaxed and easy-going?

c) Has she ever asked you to slow down in your work? NEVER? How would SHE put it -- in HER OWN words?

6. When you get ANGRY or UPSET, do people around you know about it? How do you show it?

7. Do you think you drive HARDER to ACCOMPLISH things than most of your associates?

8. Do you take work home with you? How often?

9. Do you have any children? When they were around the ages of 6 and 8, did you EVER play competitive games with them, like cards, checkers, Monopoly?

a) Did you ALWAYS allow them to WIN on PURPOSE?

b) Why (or why NOT)?

10. When you play games with people your OWN age, do you play for the FUN of it, or are you REALLY in there to <u>WIN</u>?

11. Is there any COMPETITION in your job? Do you enjoy this?

a) Are you competitive off the job -- sports for example?

12. When you are in your automobile, and there is a car in your lane going <u>FAR TOO SLOWLY</u> for you, what do you do about it?

a) Would you MUTTER and COMPLAIN to yourself?

b) Would anyone riding with you know that you were ANNOYED?

13. Most people who work, have to get up fairly early in the morning -- in your particular case, uh-what-time-uh-do-you-uh, ordinarily uh-uh-uh-get-up?

14. If you make a DATE with someone for, oh, two o'clock in the afternoon, for example, would you BE THERE on TIME?

a) If you are kept waiting, do you RESENT it?

b) Would you SAY anything about it?

15. If you see someone doing a job rather SLOWLY and you KNOW that you could do it faster and better yourself, does it make you RESTLESS to watch him?

a) Would you be tempted to STEP IN AND DO IT yourself?

16. What IRRITATES you most about your work, or the people with whom you work?

17. Do you EAT RAPIDLY? Do you WALK rapidly? After you've FINISHED eating, do you like to sit around the table and chat, or do you like to GET UP AND GET GOING?

18. When you go out in the evening to a restaurant and you find eight or ten people WAITING AHEAD OF YOU for a table, will you wait? What will you do while you are waiting?

19. How do you feel about WAITING in lines: Bank lines, or Super-market lines? Post Office lines?

20. Do you ALWAYS feel anxious to GET GOING and FINISH whatever you have tc do?

21. Do you have the feeling that TIME is passing too RAPIDLY for you to ACCOMPLISH all the things you'd like to GET DONE in one day?

    a) Do you OFTEN feel a sense of TIME URGENCY? TIME PRESSURE?

22. Do you HURRY in doing most things?

All right, that completes the interview. Thank you very much.

Appendix B

EXPERIMENTAL SCRIPT

Subject (S) waits in the lounge area near the elevators upon arriving at the Graduate Center. Experimenter (E) greets S by saying, "Hello, Mr. (S's name), I'm Mr. Hilton. Will you come with me." Escort S to room 618 and seat S in the Barca lounge chair, facing the T.V. camera (E remains standing).

"Before we begin today's session, we would like to check your blood pressure. Miss Kehoe is going to do that now, and I will return in a few minutes to tell you what we are going to be working on."

(E leaves, medical technician (MT), Kathleen Kehoe, enters and takes a manual blood pressure (BP), with S in a semi-supine position, after which E returns and delivers the general instructions).

"In today's session, we're interested in a variety of factors that may enhance a person's risk of developing coronary heart disease. You are probably familiar with some of

the risk factors for heart disease -- such as high levels of serum cholesterol, high blood pressure and cigarette smoking. In addition to these risk factors, we and other researchers believe that a person's life style -- for example, the way in which a person goes about getting what they want in life, may be involved in this disease. Therefore, we are interested in studying how your day-to-day experiences, annoyances, and hassles may affect the functioning of your heart. In order to do this, we utilize standard laboratory conditions, for it is only in a standardized setting - where everybody works on identical tasks, with known properties, that we can be sure of the type of behaviors that may be responsible for specific bodily responses."

"What you will do today, then, is work on a series of standard tasks, with which we have had a good deal of experience, and that capture the essence of various skills and activities that are important in the daily routine of most people. These activities include problem solving, as well as visual tracking, motor coordination, and tests of thinking and feeling. I want to assure you that nothing in today's session will cause you any discomfort. We are <u>not</u> using noise, shock or any other type of noxious stimuli. We are interested in how performance on these tasks affects two blood components known as catecholamines. Recent research has implicated these substances in acceleration of damage to coronary artery walls over time. Hardening of the coronary

arteries is the real problem underlying heart disease. Indeed, heart attacks that appear suddenly may be the result of arterial damage that has taken many years to develop."

"Knowledge concerning even small elevations of catecholamine levels, as a function of your performance on the tasks we will be using, can shed light upon the long term effects of such activities on the development of heart disease."

"In order for us to examine the blood components, it will be necessary to take a series of very small blood samples during today's session. This will be done by Miss Kehoe. The total amount of blood we will draw is less than what you would normally give for a routine blood test."

"To take these blood samples, a catheter will be inserted in your arm and taped in place. It will remain there during the entire session. The catheter we use is very flexable and won't cause you any discomfort while it's in place."

(Demonstrate the catheter).

"This procedure is completely routine and entirely safe. If this is clear, please read, sign, and date this form. It describes what I've been telling you so far."

(Hand S consent form. Only date and signature are required).

"Incidentally, we will also be taking blood pressure readings from you today, using an Arteriosonde. This device works in the same way as the usual blood pressure cuff, except that it allows us to monitor your readings from the next room. Your heart rate will be automatically monitored also, with the use of this instrument known as a photocell plethysmograph. There is a sensor in the tip of this device that records the pulsation of the blood moving through your finger. This gives us a pulse rate, which we convert to a heart rate."

"Now, before I describe the tasks you'll be working on today, Miss Kehoe will come in to insert the catheter. By the way, the measurements we will be taking are extremely sensitive and it's necessary that we remove your watch for this purpose. Just give it to me now and I'll return it later."

(E takes watch and places it on top shelf out of sight).

"I'll return in a few minutes and we'll go on."

(E leaves as MT enters. The general routine that will be followed by the MT at this point, including baseline BP, HR & base blood, is listed below).

1. MT enters, inserts catheter and begins stop watch at the end of venipuncture (VP).
2. Arteriosonde cuff placed on arm opposite catheter, and photocell plethysmograph placed on same arm as catheter. S is instructed to relax, and not to move his arms while reclining (to extend arm that has Arteriosonde cuff palm down). Music is played via a tape recorder.
   a) 5 minutes from VP, Arteriosonde and photocell plethysmograph are activated. Readings are taken at 2 minute intervals.
   b) 30 minutes from VP, BASELINE BP and HR readings are recorded.
3. MT enters the experimental room after these recordings. First blood sample is taken from S (Base blood).
4. E returns and gives specific instructions as described below.

"Now we're ready to proceed with the study. Miss Kehoe will come in periodically during the session to take blood samples. Of course, the pressure cuff will inflate automatically at fixed intervals throughout the session."

"I should also add that we will be using a tape recorder in parts of today's session. The microphone used for recording is this little lapel device, which I'm sure you're

familiar with. (E attaches microphone to S's shirt). All of your responses will be kept in strict confidence."

"As I said before, you are going to work on several tasks today. The first is concerned with your feeling states. The specific instructions for this task are tape-recorded. You will hear them over headphones. I will be able to see and hear you from the control room."

"Now, the success of this phase of the study will depend very much on how INTENSELY you are able to feel the emotions we give you. THIS IS VERY IMPORTANT: You must do your best to ACTUALLY FEEL whatever emotion we ask you to experience. For example: If we ask you to feel sad, try to get yourself into a state where you are ACTUALLY as sad as possible RIGHT NOW. Try to experience what you usually feel when you are sad. For example, some people feel a heaviness in their chest, a queasiness in their stomach and a general feeling of lethargy. If we give you other emotions, experience them the way you normally would feel. Remember to experience the emotions in this way HERE and NOW. Just let yourself go and feel the emotion fully. (Pause)."

Also, select an example of the emotion that is the strongest one you can think of. If you are asked to feel sad, for example, choose the saddest situation that you have ever experienced. And remember, try to experience the emotion here and now.

After you create this feeling in yourself, there will be a period in which I will ask you to describe the situation leading to your feelings and the intensity of your feelings (pause). You will be asked to experience and describe a succession of four different emotions."

"When you're asked to <u>think</u> about an emotion, or to <u>talk</u> about it, please continue to do this until you hear the next set of instructions. In other words, take all of the time given to you to think or talk about the emotion. (Brief pause). O.K., let me start the tape-recorded instructions, and you can begin with the first emotion."

(E leaves room and begins the taped instructions).

> Let us begin with physical fatigue. I want you to think of a situation which made you very tired, even exhausted. Think of a situation that you might have encountered in the past, or choose an imaginary situation that <u>would</u> make you tired. Choose a situation that can make you tired when you think about it <u>now</u>. Imagine in your head the events which contribute to increasing fatigue. If you need a series of situations, that's okay, too. What is important is to be as tired as you possibly can. You will be given a couple of minutes to build your felt fatigue up to its maximum intensity. Please close your eyes so the room does not distract you. You may begin now.

(Start BP cuff inflation, but do not record this task. At the end of the 3 min. period begin the next set of taped instructions).

Now, what I would like you to do is describe the situation which made you physically tired. Tell me how you reacted. Pretend that you are telling this to a sympathetic friend who is listening carefully. Describe the situation to your friend in such a way that he can also begin to be tired. Take the whole 3-minutes allotted to tell the story. O.K. begin talking.

(Start BP but do not record this task. At the end of the 3 min. period begin the taped instructions for the rest period).

I want you to stop thinking about being tired. At this point you are going to have a brief rest period. Try to relax. You may close your eyes if this will help you to relax. After the rest period, the recorded instructions will begin again for the next emotion.

(At the end of the 5 min. rest period, begin the taped instructions for the next emotion).

I want you to think of a situation which made you (angry) in the past, or choose an imaginary situation that <u>would</u> make you (angry). Choose a situation that can make you (angry) when you think about it <u>now</u>. Imagine in your head the events which contribute to increasing (anger). If you need a series of situations, that's okay, too. What is important is to feel as (angry) as you possibly can. You will be given a couple of minutes to build your (anger) up to its maximum intensity. Please close your eyes so the room does not distract you. You may begin now.

(Record BP & HR 5, 65 & 125 secs. into the task. At the end of the 3 min. period, MT enters and takes second blood sample "Experimental 1," then leaves. After MT leaves begin taped instructions and turn on second tape recorder for S's verbal description of his imaging).

> Now, what I would like you to do is to describe the situation which made you (angry). Tell me how you reacted. Pretend that you are telling this to a sympathetic friend who is listening carefully. Describe the situation to your friend in such a way that he can also begin to feel (angry). Take the whole 3-minutes allotted to tell the story. O.K., begin talking.

(Record BP & HR 5, 65 & 125 secs. into the task. After the relating, MT enters and takes third blood sample "Experimental 2," then leaves. Begin taped instructions for the 5-minute rest period).

> I want you to stop thinking about being (angry). At this point you are going to have a brief rest period. Try to relax. You may close your eyes if this will help you to relax. After the rest period, the recorded instructions will begin again for the next emotion.

(At the end of the 5 min. rest period, begin the taped instructions for the next emotion).

> Now, I want you to think of a situation which made you (afraid of being hurt physically). Think of a situation that you might have encountered in the past, or choose an imaginary situation that would make you (afraid of being hurt). Choose a situation that can make you (afraid) when you think about it now. Imagine in your head the events which contribute to increasing (fear). If you need a series of situations, that's okay, too. What is important is to (be afraid of physical injury) as much as you possibly can. You will be given a couple of minutes to build your (fear) up to its maximum intensity. Please close your eyes so the room does not distract you. You may begin now.

(Record BP & HR 5, 65 & 125 secs. into the task. At the end of the 3 min. period, MT enters and takes fourth blood sample "Experimental 3," then leaves. After MT leaves begin taped instructions and turn on second tape recorder for S's verbal description of his imaging).

> Now, what I would like you to do is to describe the situation which made you (afraid of being hurt). Tell me how you reacted. Pretend that you are telling this to a sympathetic friend who is listening carefully. Describe the situation to your friend in such a way that he can also begin to feel (afraid). Take the whole 3-minutes allotted to tell the story. C.K., begin talking.

(Record BP & HR 5, 65 & 125 secs. into the task. After the relating, MT enters and takes fifth blood sample "Experimental 4," then leaves. Begin the 5-minute rest period with the taped instructions).

I want you to stop thinking about (fear). At this point you are going to have a brief rest period. Try to relax. You may close your eyes if this will help you to relax. After the rest period, the recorded instructions will begin again for the next emotion.

(At the end of the 5 min. rest period begin the taped instructions for the next emotion).

I want you to think of a situation which made you feel (upset and distressed) in the past, or choose an imaginary situation that would make you (upset and distressed). Choose a situation that can make you (distressed) when you think about it now. For example: Failing a promotional exam, or any other situation that would upset you and make you anxious. Imagine in your head the events which contribute to increasing (distress). If you need a series of situations, that's okay, too. What is important is to feel as (distressed) as you possibly can. You will be given a couple of minutes to build your (distress) up to its maximum intensity. Please close your eyes so the room does not distract you. You may begin now.

(Record BP & HR 5, 65 & 125 secs. intc the task. At the end of the 3 min. period, MT enters and takes sixth blood sample "Experimental 5," then leaves. After MT leaves begin taped instructions and turn on second tape recorder for S's verbal description of his imaging).

Now, what I would like you to do is to describe the situation which made you (upset and distressed). Tell me how you reacted. Pretend that you are telling this to a sympathetic friend who is listening carefully. Describe the situ-

> ation to your friend in such a way that he can also begin to feel (distressed). Take the whole 3-minutes allotted to tell the story. O.K., begin talking.

(Record BP & HR 5, 65 & 125 secs. into the task. After the relating, MT enters and takes seventh blood sample "Experimental 6," then leaves. Begin the 5-minute rest period).

Whatever the last emotion, distress, anger or fear, the taped instructions for the rest period are modified to lead into the longer, final rest period. The modification is as follows:

> I want you to stop thinking about being (upset and distressed). At this point you are again going to have a rest period. It will be somewhat longer than before. Try to relax. You may close your eyes if this will help you to relax. After the rest period, we will continue with the remainder of today's session.

The final rest period lasts 15 minutes. After this period, E returns and conducts the post-experimental interview and debriefing).

Appendix C

POST-EXPERIMENTAL INTERVIEW AND DEBRIEFING

POST-EXPERIMENTAL INTERVIEW

"Now, I'd like to ask you a few questions about the session up to this point."

"To begin with, have you ever participated in psychological research before? How about biomedical research?"

(Ask for elaboration about the research, and S's reaction to the previous study).

"Have you ever had your blood taken with a catheter before? When? How did you react to it today? Is the catheter uncomfortable?"

"In general, how have you reacted to the session so far? Is there anything particularly striking or unusual about it?"

"Let me ask you about the blood pressure cuff. Is it uncomfortable? Distracting? Annoying? Did it affect your performance in any way?"

Next, E probes about instructions for the day: Exercise, coffee/tea/cocoa, smoking, drugs. Stress it is essential that we know now about his compliance with our instructions not to drink caffeine-related drinks, smoke (if any smokers are included), or take drugs (including aspirin or cold medications) -- before we analyze the blood. E says:

"The type of blood analysis we do is very expensive and therefore we do not want to find out about these things after we get the results of the analysis. So please be frank about the amount of smoking, coffee, cola drinks, and any drugs or medication you may have taken recently -- i.e., the last four (4) hours."

DEBRIEFING

At this point, E reiterates the purpose of the study - as given in the introduction. Also, he explains why no other tasks were presented and that he was told about the other tasks in order to maximize his interest in the session. E emphasizes the purpose of the study: To examine the effects of behavior on catecholamines and blood pressure -- factors related to heart disease.

After this explanation, E thanks S for participating and explain the importance of his not discussing the specific nature of this research with anyone else, since they too may participate. This point is emphasized. E explains that if the next S knows about the study ahead of time, important information about natural, spontaneous behavior, may be lost.

"Your own participation will be wasted and knowledge that could be important to all of us in eventually preventing heart attacks may be sacrificed. LET ME STRESS AGAIN: If future participants know anything about the study, their participation in this research could distort the results and produce misleading conclusions. Therefore, although it is tempting to discuss your experiences here with your friends and co-workers, PLEASE be extremely careful not to do so."

Next, E thanks S, returns his watch, and pays him his fee. Also, E tells S that he can get the results of the study, including his own catecholamine and blood pressure responses, if he wants them. E explains that it will take several months to complete the analyses. However, if S wants the results, E will mail him a copy. If S says "Yes," E notes this on his data folder.

Appendix D

VIDEO FACIAL RATINGS

Each tape segment will have its own ID number which you must write on the rating sheet.

ID#:_____ Segment:_____

Rater:_____ Date:_____

INSTRUCTIONS: You are going to watch video tape recordings of people's faces. Their speech has been deleted. What you must do is rate the emotion, conveyed by the facial expressions, using the following seven-point scales. Circle a number on the scale to indicate your rating.

Pleasant	1	2	3	4	5	6	7	Unpleasant
Sociable	1	2	3	4	5	6	7	Unsociable
Good	1	2	3	4	5	6	7	Bad
Agreeable	1	2	3	4	5	6	7	Disagreeable
Active	1	2	3	4	5	6	7	Passive
Excitable	1	2	3	4	5	6	7	Calm
Emotional	1	2	3	4	5	6	7	Unemotional
Tight	1	2	3	4	5	6	7	Loose

Hard	1	2	3	4	5	6	7	Soft
Closed	1	2	3	4	5	6	7	Open
Clear	1	2	3	4	5	6	7	Hazy
Intentional	1	2	3	4	5	6	7	Unintentional

Level of Involvement: Intensity of affect and expression.

1. No response, failure to engage in task.
2. Disassociated from emotion.
3. Low involvement, low intensity.
4. Moderate involvement, moderate intensity.
5. Involved with emotion, intense expression.
6. Highly involved, very intense expression.
7. Extremely involved, extremely intense expression.

Appendix E

SUBJECTS CONSENT STATEMENT

CITY UNIVERSITY OF NEW YORK
THE GRADUATE SCHOOL AND UNIVERSITY CENTER
Principal Investigator(s): Dr. David C. Glass
Project: BEHAVIOR PATTERNS AND CATECHOLAMINES

I hereby acknowledge that on _____, 19__ I was informed by William Hilton of the Graduate Center, of a project concerning or having to do with the following: (Describe briefly) The effects of task performance upon various blood components and blood pressure.

I was told with respect to my participation in said project of the possible risks involved, the procedures involved; possible alternative procedures and the expected benefits from the program, i.e., that: (State information given)

I will be working on a variety of psychomotor and verbal tasks. A series of small blood samples will be taken from my arm through an indwelling catheter by a qualified medical technician. I was informed that these procedures follow standard clinical practice and techniques which entail no danger to my health.

I am fully aware of the nature and extent of my participation in said project and possible risks involved or arising therefrom. I hereby agree, with full knowledge and awareness of all of the foregoing, to participate in said project. I further acknowledge that I have received a complete copy of this consent statement.

I also understand that I may withdraw my participation in said project at any time and that I may inspect a copy of the Institutional Assurance filed by the Research Foundation, CUNY, with the U.S. Department of Health, Education and Welfare.

Dated: New York _____, 19__

_____
(Signature of Subject)

_____
(Printed Name of Subject)

_____
(Residence of Subject)

Appendix F

FACTOR ANALYSIS OF VIDEO FACIAL RATINGS

Individual Items of the Semantic Scale
Combined Across Raters for
Anger - Imaging, HI/SBP

VARIMAX ROTATED FACTOR PATTERN

SEMANTIC SCALE	FACTOR 1 (Pleasantness)	FACTOR 2 (Activation)	FACTOR 3 (Control)
Pleasant	0.83	-0.24	0.12
Sociable	0.89	-0.15	-0.21
Good	0.80	-0.10	-0.11
Agreeable	0.76	-0.12	0.06
Active	-0.15	0.78	-0.06
Excitable	-0.19	0.88	-0.07
Emotional	-0.17	0.82	-0.10
Tight	-0.30	0.49	0.55
Hard	-0.53	0.34	0.43
Closed	-0.02	-0.16	0.72
Clear	-0.16	0.32	-0.24
Intentional	-0.05	0.39	0.15

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