8-1988

Effects of Task Difficulty, Performance Consequence, and Social Interaction on Physiological Reactivity in Post-Coronary Patients

A. Janelle Maldonado
Western Michigan University

EFFECTS OF TASK DIFFICULTY, PERFORMANCE CONSEQUENCE, AND SOCIAL INTERACTION ON PHYSIOLOGICAL REACTIVITY IN POST-CORONARY PATIENTS

by

A. Janelle Maldonado

A Dissertation
Submitted to the
Faculty of The Graduate College
in partial fulfillment of the
requirements for the
Degree of Doctor of Philosophy
Department of Psychology

Western Michigan University
Kalamazoo, Michigan
August 1988

EFFECTS OF TASK DIFFICULTY, PERFORMANCE CONSEQUENCE, AND SOCIAL INTERACTION ON PHYSIOLOGICAL REACTIVITY IN POST-CORONARY PATIENTS

A. Janelle Maldonado, Ph.D.

Western Michigan University, 1988

Three experiments were performed to determine the effects of three task variables and Type A behavior pattern on physiological reactivity to time-limited math and anagram tasks. In the first experiment, ten post-coronary patients performed time-limited computer tasks under two performance consequence conditions: Point Reward or presentation of an Auditory Blast combined with two task difficulty conditions (40% and 60% difficult). The findings of Experiment 1 indicated that while the tasks did produce levels of physiological reactivity comparable to those observed in the literature, there were no significant main effects for either variable for any of the five measures. A significant difficulty by consequence interaction was found for skin conductance (EDG). The findings did not provide a demonstration of a statistically reliable interaction between behavior pattern and the consequence or difficulty factors.

Experiment 2 was conducted to evaluate the effects of (a) two task difficulty conditions (10% and 90% difficult) while controlling for the effects of task consequence and (b) the effects of three task consequence conditions Reward, Forced Failure and a no consequence Control while holding task difficulty constant. The results revealed a significant main effect for consequence for systolic blood pressure such that the Reward produced the highest levels of reactivity followed by Forced Failure and then the Control condition. A similar trend was observed for diastolic blood pressure and skin conductance measures but these trends were not statistically significant. No significant behavior pattern by consequence interaction was found for any physiological measure. However, graphic trends suggested that Type B individuals were slightly more reactive across consequence conditions for all measures except frontalis muscle electromyographic (EMG) reactivity.

In Experiment 3, subjects performed three tasks involving social interaction (Impatience, Competition, and Hostility) and two nonsocial tasks (Mental Arithmetic and Computer Arithmetic) while physiological reactivity was monitored. The analysis revealed a group by condition interaction for pulse rate such that Type A subjects' were significantly more pulse rate reactive during Competition than Type B subjects. Although the differences were not significant, social interaction conditions appeared to produce higher elevations than nonsocial conditions for systolic blood pressure, diastolic blood pressure, pulse rate and frontalis EMG. Implications for future research concerning the effects of performance consequence and social demands on psychophysiological responses in Type A and B individuals are discussed.

Order Number 8827686

Effects of task difficulty, performance consequence, and social interaction on physiological reactivity in post-coronary patients

Maldonado, Annette Janelle, Ph.D.

Western Michigan University, 1988

U·M·I
300 N. Zeeb Rd.
Ann Arbor, MI 48106

ACKNOWLEDGEMENTS

I would like to acknowledge the many individuals and agencies without whom this project would not have been possible. I would like to express my appreciation and thanks to my advisor, Dr. Wayne Fuqua, for his guidance and support in all stages of this study. I also wish to thank the members of my doctoral committee for their cooperation throughout my graduate program. My sincere appreciation to Dr. Fred Gault for his assistance and guidance in physiological measurement and calibration procedures. Special thanks go to Ms. Linda Felch for her generous assistance in the areas of statistical analysis and graphics, for psychological pushes she provided, and for her patience and friendship as this project was brought to fruition. To Dr. Janel Harris, I would like to express my many thanks for the opportunity to have collaborated on this and other research, for the excellent professional model she has been, for the many discriminative stimuli she provided for my own professional behavior, and for her most valued friendship over our graduate years. Many thanks and much love to Mr. William Dietz for his friendship and support, his immeasurable contributions to all aspects of this project, and especially for his creative computer programming. Thanks to Ms. Patricia Cole for her many hours of assistance in the running of experimental sessions and data collection. I would like to thank Dr. Fred Garmon, Dr. Michael Macken, and the patients, staff and physicians of the Institute for Cardiac Rehabilitation of Borgess Hospital, Kalamazoo, Michigan, for their cooperation and enthusiastic support in the implementation of this project. To my colleagues at the Behavior Pharmacology Research Unit, Johns Hopkins School of Medicine, I express my heart felt appreciation for your help, resources and friendship which served to keep me on task and made the job so much more enjoyable.

I dedicate this dissertation to all members of my family, to Ken Saad, and Bubba. Thank you for providing me the essential resources of support, humor, patience, and love.

A. Janelle Maldonado

TABLE OF CONTENTS

TABLE OF CONTENTS CONTINUED

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Reactivity is defined as a deviation from a comparison or control value resulting from exposure to a discrete environmental event (Matthews, Weiss, & Detre, 1984). Several lines of research have produced preliminary evidence suggesting that physiological reactivity to stressful stimuli may contribute to the development of coronary heart disease (CHD). In two nonhuman retrospective studies, Manuck, Kaplan, and Clarkson (1983, 1985) examined reactivity in Cynomolgus monkeys to threat of capture, a stimulus that produced cardiac acceleration ranging from 50% to 100% above baseline levels. The monkeys were differentiated into "high" and "low" rate reactive animals according to the criteria of the upper and lower 30% of the distribution. The results showed that monkeys exhibiting high heart rate responsivity to threat of capture (i.e., presentation of a monkey glove) evidenced twice the atherosclerosis found in low heart rate reactive monkeys. However, both of these studies involved assessment of reactivity after a period in which the animals had been exposed to an atherogenic diet. The presence of this disease promoting factor may have influenced both baseline levels and reactivity levels if heart rate was altered by the presence and extent of disease. The findings demonstrate a limited association but not a causal relationship between high heart rate reactivity and extent of atherosclerosis following exposure to an atherogenic diet.

Retrospective research with humans has generated similar findings by contrasting the psychophysiological responses of persons with and without CHD. Corse, Manuck, Cantwell, Giordani, and Matthews (1982) compared blood pressure reactivity in coronary and non-coronary patients who were asked to perform a stressful mental arithmetic task. The results indicated that the coronary patients experienced significantly greater diastolic blood pressure elevations than did the non-coronary patient controls (i.e, mean change of +8.0 and +2.9 mmHg respectively). Steptoe, Melville,and Ross (1984) found that the hypertensive subjects evidenced significantly greater elevations in cardiovascular measures to a mental arithmetic task than did the normotensive patients. These retrospective studies suggest that organisms already evidencing disease may display higher physiological reactivity to stress than do those not evidencing disease.

There is one prospective human study that has examinined the relationship between reactivity and disease development. Keys, et al. (1971) found that physiological reactivity to a standard cold pressor test was associated with a higher incidence of coronary heart disease in humans at a 23 year follow-up . Thus, the prospective data suggest that organisms that are more physiologically reactive may be more predisposed to disease development over time. While this study exemplifies the type of longitudinal studies that will best evaluate the role of reactivity and determine whether reactivity is a risk factor for coronary heart disease, it is limited by its use of the one physical stressor task. Future prospective research should use a range of both physical and psychological stressors in order to more carefully account for the wide variety of stress conditions occurring in real life and how they might differ in their effects on reactivity and disease development.

The specific mechanism linking physiological reactivity to coronary heart disease is unknown. In fact, no direct evidence is yet available which clearly shows a causal relation between reactivity and disease development. One reason for the scarity of research is that studies cannot be conducted because of ethical or behavior management reasons. For example, it would be unethical to expose individuals to conditions that might promote disease development over an extended period of time. The reactivity research community must first focus on the development of a technology that will allow for the alteration of reactivity in an ethical and systematic manner if we are to use it as an independent variable. Thus far, the data are correlational in nature and it is not clear whether reactivity is a marker of future disease development (e.g., familial history of disease) or a causal link in the pathogenic chain.

The endothelial injury hypothesis is a popular model used to explain how reactivity might be linked to the development of CHD (Ross & Glomset, 1976). According to this model, stressful stimuli produce the initial activation of the sympathetic adrenal medullary system and the pituitary adrenal cortical system which results in increased heart rate and cardiac output. These increases stimulate a vasoconstrictive response with a resulting fixed elevated total peripheral resistance. The increased resistance causes repeated damage or injury to the arteries through mechanical shearing forces and leaves the arteries vulnerable to atherogenic processes including platelet aggregation, the development of fatty streaks and occlusion. Thus reactivity may be linked to CHD by virtue of a chronic tendency of some individuals to show greater adrenal-medullary and sympathetic nervous system responses to environmental challenge, making them more susceptible to pathogenic processes, and to subsequent disease development.

The research on physiological reactivity frequently has included an examination of the relationship of reactivity to Type A behavior pattern. The Type A behavior pattern is an epidemiological construct that was originally formulated by Friedman and Rosenman (1974). Compared with noncardiac patients they were treating, they found that patients suffering from cardiac disorders more often displayed a certain constellation of characteristics with the principal components of extreme aggressiveness, competitive achievement striving, a persistent sense of time urgency, and poorly modulated hostility. The absence of these characteristics denoted non-coronary-prone behavior pattern or Type B behavior. Clinical investigations demonstrated that persons designated as Type A were more likely to develop coronary heart disease and exhibit more severe coronary atherosclerosis than persons designated Type B (Haynes, Feinleib, & Kannel, 1980). Since its early conceptualization, Type A behavior pattern has been the focus of research efforts to identify the specific behavioral subcomponents of the global Type A construct that are most "toxic" in the increased risk for coronary disease. These efforts have resulted in the designation of a number of specific behaviors that are highly correlated with severity of coronary disease including speed of speech, volume of speech, number of interruptions, high self-report of potential for hostility, over-estimation of the passage of time, accelerated work pace regardless of time-demands, and behavioral signs of tension, hyperactivity, and impaired performance when required to work at a slow pace (Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1984; Williams, et al. 1980). Subsequent research has focused on identifying possible differences in physiological responsivity to environmental stress in Type A and Type B individuals.

Approximately fifty studies have investigated reactivity as a psychophysiological correlate of Type A behavior pattern. Generally, these studies have found a moderate relationship between Type A behavior and physiological responses to laboratory stressors. The associations reported, however, vary widely depending on the Type A assessment technique employed, the subject population characteristics, and the type of stressor situations explored. In an unpublished review, Holmes and Zurawski (1983) reported that in 70% of the studies Type A subjects evidenced greater magnitude of cardiovascular reactivity than did Type B subjects. The remaining 30% of the studies did not report significant group differences between Type A and Type B subjects.

Relative to Type B's, Type A's have been reported to show greater reactivity during laboratory stress tasks for (a) systolic and/or diastolic blood pressure (Contrada, et al., 1982; Dembroski, MacDougall, Herd, & Shields, 1979a; Dembroski, MacDougall, & Lushene, 1979b; Krantz et al., 1981; Manuck & Garland, 1979; Manuck, Harvey, Lechleiter & Neal, 1978; MacDougall, Dembroski, & Krantz, 1981; Gastorf, 1981); (b) heart rate (Dembroski, MacDougall, Shields, Petitto, & Lushene, 1978; Dembroski, et al., 1979; Glass, Krakoff, Contrada et al., 1980; Krantz et al., 1981;Van Egeren, 1979); (c) finger pulse amplitude or volume (Scherwitz, Berton, & Leventhal, 1978; Van Egeren, 1979), (d) epinephrine or norepinephrine release (Friedman, Byers, Diamant, & Rosenman, 1975; Glass et al., 1980; Frankenhaeser, Lundberg, & Foresman, 1980), (e) cortisol elevations (Lundberg & Foresman, 1979); (f) cholesterol (Lovallo & Pishkin, 1980); (g) platelet aggregation (Simpson et al., 1974); and (h) skin conductance (Lovallo & Pishkin, 1980). Significant differences in the reactivity of subjects classified as Type A or B are most often observed in studies using the structured interview for classification, with older, white collar subject samples, and with adequate controls for familial history of hypertension (Krantz & Manuck, 1984). Holmes and Zurawski (1983) emphasized in their review that these results should be viewed with caution because the overall increments in cardiovascular measures observed in these studies are not very large and may not be of clinical importance. Furthermore, in those studies reporting reliable differences in reactivity for Type A and Type B individuals, a number of methodological problems were present. These problems include using reactivity change scores that were not adjusted for initial levels, summation of values across conditions, and a failure to counterbalance task presentation across sessions (Holmes & Zurawski, 1983).

Constructing acceptable laboratory stressor tasks that can be used for the systematic study of reactivity is a prerequisite for answering the questions that remain regarding the link between reactivity and the development of disease. This turns out to be no simple task because task characteristics may be quite complicated in their effect on physiology, difficult to quantify, and hard to replicate across laboratories. In their chapter on psychological stressors, Krantz, Manuck, and Wing (1986) suggest several task features that must be taken into account in task construction including the physical nature of the task, the type of stimulation that is applied, and the time-related patterns of physiological responding that may be produced. It is important to examine the intercorrelations between reactivity of different tasks and stimuli as well as the generalizability (of effects) from laboratory assessment to naturalistic settings. Krantz, et al., (1986) point out that

> an ideal stressor for studying reactivity (and its relation to behavior and disease) would be one that previously showed an ability to elicit a stable magnitude and patterning of responses when applied comparably in different laboratories and when presented to different subject groups or to the same individuals on repeated occasions. However, few, if any of the tasks presently meet all of the aforementioned criteria. (p. 102)

One problem in the reactivity literature is the lack of generality of the type of stressor tasks that are used. Each laboratory uses a different task and defines various task parameters differently thus making it almost impossible to compare findings and draw general conclusions. Several types of tasks have been examined in the reactivity literature for their effect on degree of reactivity and pattern of physiological response. Some tasks have been adopted for their standard and reliable effects in producing physiological responses, others for their similarity to daily life stressors, or for the physiological system they affect. The stressor tasks studied thus far include visual-verbal tasks (Manuck et al., 1978), reaction-time shock avoidance (Contrada et al., 1982; Jorgenson & Houston, 1981), auditory reaction time, Super Pong (Glass et al., 1980) and anagrams (Dembroski et al., 1978; Frankenhaeuser et al., 1980), Structured

Interview Type A assessment, history quiz, general interview (Dembroski et al., 1979; Krantz et al., 1981; Smyth, Call, Hansell, Sparacino & Strodtbeck, 1978), unsolvable puzzles with and without noise (Friedman et al., 1975), cold pressor with high or low challenge instructions (Dembroski, et al., 1979a), mental arithmetic (Frankenhaeuser et al., 1980; Lane, White, & Williams, 1984), vigilance tasks, Stroop Color Word Interference task (Steptoe et al., 1984), and watching an aversive movie (Lundberg & Foresman, 1979), tracking tasks, delayed digit recall (Glass et al., 1980), time estimation, repeating words with and without tape of sounds (Price & Clarke, 1978), Wechsler picture completion (Corse et al., 1982), and the Prisoner's Dilemma game (Van Egeren, 1979). Nonlaboratory or natural life situations have been examined for their effects on reactivity although it is not clear they operate as discrete environmental events as specified by Matthews, Weis, and Detre (1984). Natural stressors that have been evaluated include a treadmill test (Simpson et al., 1974) and working day stress (DeBacker et al., 1979). As might be expected, the findings are highly inconsistent depending on the task, subject sample, and measures taken in the various laboratories.

Another limitation in the reactivity literature is the scarcity of studies examining the importance of task consequence on the magnitude of responding to a stressor task. Task consequence, or the relationship between a response and the consequences of that response, is an important but neglected task variable that may effect reactivity to stress. Reasons for the importance of this variable deserve discussion. First, it is known from years of research in biofeedback and experimental and applied behavior analysis research that consequences affect behavior and physiological responding in powerful ways. It is safe to assume that these effects are present in the laboratory as well as in daily living. In addition, psychophysiological research as well as biofeedback research has demonstrated that certain operant conditioning schedules (consequences) and certain methods of feedback produce changes in some physiological responses and response patterns and these schedules and feedback procedures may be arranged to condition the direction of physiological responses (Eliot, 1979). Given this clear link between consequences, behavior and physiological responding, it makes good sense to examine the influence of task consequences in laboratory stressors. From a practical view, describing task consequences will facilitate replication of procedures across laboratories and allow us to more effectively simulate naturally occurring stressors and improve the generality of our results. The present argument is thus, that consequences are potentially important, perhaps even defining features of all stressors that demand active coping. We need to know if this task variable affects reactivity so it can be specified and controlled in future research or manipulated to alter the level of reactivity.

The performance consequences employed in reactivity studies are difficult to pinpoint because the procedures are often vaguely described and most investigators do not state the procedure as a consequence arrangement. Even when these consequences are described, research has rarely aimed at evaluating their effects on reactivity. Consequences may be artificially arranged (e.g., awarded points or money) or naturally occurring (e.g., winning, finding a solution). From a review of the literature it appears that in most of the studies, correct task performance resulted in avoidance of an aversive event, such as shock, noise, or point loss. Almost all the tasks involve an element of time pressure. In most studies, performance was monitored during tasks but it is unclear if subjects received feedback regarding their performance in all cases. In cases where such feedback was available, unspecified performance consequences may have been in effect. Since procedures have been vague in this regard, we do not know for certain what performance consequences were used.

Only four studies have experimentally evaluated the effects of task consequences on reactivity to stressor tasks. The majority of these studies have evaluated the effects positive reward or incentives on the comparative reactivity of subjects classified as Type A or Type B. Blumenthal, et al. (1983) examined the effects of task incentive on task performance and cardiovascular response in Type A and B subjects during a verbal problem solving task. Subjects were randomly assigned to a monetary incentive or nonincentive control condition. The results indicated that Type A subjects showed significant increases in systolic blood pressure and heart rate in both conditions, while Type B subjects showed significant increases in heart rate and systolic blood pressure only when incentives were offered. Glass et al., (1980) had subjects compete individually in a Super Pong game for four games with no monetary incentive (points only) and four games for a twenty-five dollar monetary incentive. Type A subjects responded with significantly greater changes in systolic blood pressure (i.e., 12-25 mmHg) during task performance than did Type B subjects, but the presence of monetary incentive did not enhance or reduce these effects for either Type A's or Type B's. In a similar study, Manuck & Garland (1979) compared blood pressure and pulse rate reactivity during a monetary incentive condition with a no incentive condition and found that Type A's responded with significantly greater elevations in systolic blood pressure (i.e., 3.6-8.3 mmHg) and pulse rate reactivity (i.e., 2.1-6.4 mmHg) that Type B's but, as in the previous study, the presence or absence of an incentive did not produce significantly different pattens in blood pressure or pulse pressure between Type A and Type B subjects. Based on these three studies, it appears that reward or incentive consequences have no effect on reactivity in individuals assessed as Type A but may be related to reactivity in individuals assessed as Type B. Unfortunately, no information is available about the effects of reward on reactivity independent of this personality variable.

Only one study has examined the effects of negative consequences. Perkins (1984) exposed 70 Type A and 70 Type B subjects to three point loss conditions combined with three conditions of performance feedback. Subjects performed a continuous button pushing task involving high response cost (loss of money), low response cost (loss of points), or no response cost. Subjects were further divided into groups that received high, moderate, or low levels of failure feedback. The results indicated greater heart rate reactivity during high response cost conditions relative to the low and no cost conditions. Furthermore, Type A subjects were significantly more heart rate reactive than Type B's, particularly under high cost conditions. This single study suggests that response cost may have a differential effect on the physiological reactivity of Type A and Type B individuals and that this effect is related to magnitude of response cost.

The available reactivity literature has not provided enough data to allow clear explanations of these group differences in reactivity to reward and response cost consequences. One possible explanation is that the differential response to reward consequences in Type A persons reflect the self-selected behavioral standards of Type A individuals independent of programmed consequences. According to Blumenthal et al. (1983) the group differences reflect an interaction of behavior pattern classification and situational demands that produces a shift from one integrated pattern of cardiovascular response to another. The hypothesis is that Type B individuals respond to nonincentive conditions with the pattern of physiological response characteristic of sensory intake tasks (i.e., vasoconstriction and increased heart rate and systolic and diastolic blood pressure) but with the addition of an incentive, their physiological response shifts to a mixed physiological pattern of responses seen both during sensory intake and the defense reaction (i.e., vasodilation and increased heart rate and systolic blood pressure). In comparison, Type A individuals show the "defense" pattern regardless of the consequences operating.

A third task variable that has been examined in the reactivity literature is task difficulty. While several researchers have examined this variable, each has defined difficulty differently. Difficulty has been defined according to degree of failure (Lovallo & Pishkin, 1980), type of task (Price & Clarke, 1978; Contrada, Wright, & Glass, 1984), degree of solvability, pre-task instructions or perceived difficulty (Gastorf, 1981), size of the problem (Holmes, McGilley & Houston, 1984), and ability to avoid shock (Manuck et al., 1978). General conclusions regarding the effects of task difficulty are impossible to draw because of this procedural inconsistency. In some studies, higher levels of difficulty appeared to increase physiological reactivity (Manuck et al., 1978; Obrist et al., 1978). Other studies reported that increased difficulty produced no difference in reactivity (Lovallo & Pishkin, 1980). In all the studies, increased difficulty was evaluated for differential effects on the reactivity of Type A or Type B classified subjects. The results again are inconsistent, with some reports of group differences where Type A subjects were more reactive than Type B subjects during extremely difficult tasks but not during low or moderately difficult tasks (Holmes et al., 1984) and other studies finding no dramatic group differences (Lovallo & Pishkin, 1980; Price & Clarke, 1978).

Several questions remain regarding the effects of task consequence and task difficulty on physiological reactivity. There is no definitive demonstration in the literature of the possible differential effects of level of difficulty or performance consequences on reactivity. The question of whether psychological stressor tasks may differ in their effects depending in the presence of positive or negative performance consequence has not been answered. Nor has the question of the specific effect of objectively defined task difficulty been sufficiently addressed. In all probability, multiple consequences are operating in real life stress situations that produce physiological reactivity. Similarly, real life behavioral challenges are likely to involve a wide range of demands in terms of difficulty. Thus an analysis of physiological reactivity to laboratory simulations of the complex consequences that characterize naturally occurring stressors is needed. In this way, we may better determine the probable effects of naturalistic stressors, the stability of the effects of these task variables over time, the possible differences in resulting reactivity patterns, and the degree to which a given condition enhances or diminishes the stressfulness of a task.

The first experiment examined the effects of two task consequences and two levels of objectively defined task difficulty on physiological reactivity during performance of a standard laboratory task. The purpose of the first experiment was twofold: (1) to determine the effects of task difficulty and task consequence on degree and pattern of reactivity, and (2) to examine possible interactive effects of task consequence, task difficulty and Type A classification across several physiological measures. Efforts were made to address the methodological problems noted in previous reactivity research by controlling for initial levels of physiological activity, using repeated exposure to experimental conditions, and counterbalancing the order of presentation for experimental conditions. Coronary patients were chosen as subjects in these experiments because past research has demonstrated that both coronary patients and Type A individuals without manifest symptoms of CHD respond to experimental tasks with greater magnitude of physiological responding than do Type B subjects (Rosenman, 1978) and that Type A pattern is significantly predictive of recurrent myocardial infarction (Zyzanski, Jenkins, & Ryan, 1976).

The purpose of Experiment 2 was to compare the effects of two alternative task consequences (reward and forced failure) to a no consequence control condition on physiological reactivity to a behavioral stressor while controlling for level of difficulty. In addition, an extremely low level of task difficulty and an extremely high level of difficulty were compared for their effects on physiological reactivity while task contingency was held constant. The second experiment further examined the interactive effects of task consequence, task difficulty and Type A classification as well as the comparative task performance of Type A and Type B subjects.

Experiment 3 examined the effects of two nonsocial and three social interaction tasks on physiological reactivity in Type A and Type B subjects based on the Goldband (1980) and the Dembroski et al., (1984) formulations regarding Type A, anger and hostility.

EXPERIMENT 1

Method

Subjects

Ten participants (9 male, 1 female) were recruited from a cardiac rehabilitation program to participate in a study of physiological effects of psychological stress. The subjects ranged in age from 36 to 73 (x = 58.7) years. All ten subjects presented with a diagnosis of coronary heart disease and were in the maintenance phase of the cardiac rehabilitation program. Four of the ten subjects had experienced myocardial infarction and seven had undergone bypass surgery. Five of the subjects had a diagnosis for hypertension and four were taking beta blocker medication (i.e., inderol or tenormin). The Framingham Type A Scale (Haynes, Feinleib, & Kannel, 1980) was administered to all subjects and scored by the experimenter. Five subjects were assessed as Type A and five were assessed as Type B. According to nutritionist records, no major changes in diet, exercise, or medication had occurred over the previous one year period. The subjects engaged in a supervised aerobic exercise program one to three times weekly which was supplemented by home exercise. Table 1 provides a summary of all relevant subject characteristics with grouping according to Framingham Type A Scale scores.

Setting

The study was conducted at the Institute for Cardiac Rehabilitation of theBorgess Medical Center (Kalamazoo, Michigan). A graduated aerobic exercise

Table 1

Subject Profile Summary

Sub #	Age (yrs)	Type A Assessment FTAS	Beta-blockers	High blood pressure	Coronary heart disease	History myocardial infarction
1	36	B	yes	yes	no	no
2	73	B	yes	yes	yes	no
3	70	B	yes	yes	yes	no
4	8	B	yes	yes	yes	yes
6	46	B	no	no	yes	yes
mean= 54.6						
5	52	A	no	no	yes	no
7	58	A	no	no	yes	no
8	68	A	no	yes	yes	yes
9	57	A	no	no	yes	yes
10	49	A	no	no	yes	no
mean = 56.8						

program was the major component of the rehabilitation program. Patients were treated by an interdisciplinary team of cardiologists, exercise physiologists, nurses, dietitians, psychologists, and exercise leaders.

Experimental sessions were conducted in a well lighted room furnished with a small table, two chairs and a counter. The subject was seated at the table facing a computer monitor and keyboard. The subject's left arm and hand were loosely strapped to a padded arm rest in order to limit movement during physiological monitoring of blood pressure, heart rate and skin conductance. The right arm was free for typing responses to computer problems (all subjects were right handed). The research assistant was seated to the left of the subject in order to monitor digital readouts, activate the blood pressure unit, and operate the computer. All equipment was positioned on the counter to the left of the subject such that only the research assistant could observe digital displays, lights or meters.

Physiological Measures

Frontalis EMG was monitored using a J & J EMG (Model M-52) with the frequency bandpass set at 100-200 Hz. The subject's forehead was cleansed with a mild abrasive followed by an alcohol wipe. The J & J silver-silver chloride electrodes were filled with a conductive gel and then applied to the frontalis muscle in the manner described by Lippold (1967).

Skin conductance level (SCL) was measured using a J & J EDG (Model R-72) with the high sensitivity range set at + 2 µmho/mv full scale. The subject's hands were cleansed using a mild soap. Two J & J element lead annular finger electrodes prepped with a thin layer of conductive gel were applied to the volar surface of the first and third distal phalanxes of the left hand.

Both EMG and SCL were recorded from a J & J Digital Integrator (Model D-200) which displayed mean integrated values for successive 1 minute intervals on a continuous basis throughout the session.

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) and pulse rate (PUL) were electronically measured in the final minute of each rest and stress period using an Astropulse 88 (Marshall Electronics, Inc.) microphone-triggered sphygnomanometer with digital display. An automatically inflating cuff was placed over the brachial artery of the subjects' left arm. Cuff inflation was preset for each subject to a level 30 mmHg above the mean of three screening blood pressures (taken during exposure to stressor tasks) and was activated manually. Cuff deflation was at the constant rate of 2 mmHg per second.

Physiological recording instruments (EMG and EEG) were calibrated by delivering test signals generated by a Hewlett Packard 200 AB oscillator through a General Radio type 546 C Microvolter. Signals of known frequency and amplitude were applied to the equipment to detect and correct measurement error. Calibration occurred at 2 week intervals throughout the study. Blood pressure and pulse instrumentation was controlled by computer microchip and therefore could not be calibrated. However, equipment specifications indicated an error factor of $\pm$ 2 mmHg.

Type A Measures

The Framingham Type A Scale (FTAS; Haynes et al., 1980) were completed by each subject during the initial screening session in order to assess Type A behavior pattern. Research assistants were blind to Framingham scores throughout the study. Group assignment for data analysis utilized the Framingham assessment results.

Performance Measures

Math and anagram tasks were presented using a Commodore VIC-20 computer and television monitor. The task presentation program was designed so that during selected conditions, correct answers resulted in a high pitched computer tone and incorrect answers resulted in a low pitched computer tone. The program also made it possible to display a point counter on the monitor screen during selected conditions. Typed responses were recorded and analyzed to yield measures of the total number of problems attempted, the percentage of correct responses, the average response latency, and the average trial duration for each condition within a session. Exchange rates for the amount of money subjects earned for correct task performance were based on percent correct data such that subjects earned $ 0.05 for each 5 percentage points, up to $ 0.50 per problem period or a maximum or $ 3.00 per session.

General Procedures

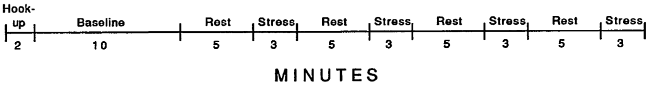
Subjects attended an initial screening session during which they signed informed consent forms. The subjects were asked to provide medical information and then they completed the Framingham Type A Scale. Training on the computer tasks was provided and individual time-limits for task performance were determined by presenting two three minute task trials. During the first trial, all subjects performed the tasks with a 10 second time-limit and their percent correct score was displayed on the screen. In an effort to set the time limit to a level that would produce approximately 50% accuracy performance such that one second was added or deducted from the 10 second time-limit for each 5 percentage points above or below 50 %. For example, if a subject received 60% correct, his time-limit was decreased to 8 seconds and if he received 40% correct, his time-limit was increased to 12 seconds. Subjects then performed a second trial with further adjustment according to the above algorithm. Blood pressure inflation levels were also determined based on maximal blood pressure elevations observed during stress task practice trials.

Subjects attended 3 experimental sessions scheduled to coincide with their regular visits to the institute. Each session was approximately one hour in duration and preceded the cardiac program exercise periods. The session began with the placement of the electrodes and blood pressure cuff. All physiological measures were monitored for an initial ten minute adaptation period. Subjects were then exposed to four experimental stress conditions presented in random order. Each stress condition was 3 minutes in duration and was preceded by a 5 minute rest/recovery period.

Physiological measures were taken throughout all rest and stress periods. Frontalis EMG and skin conductance were recorded once per minute. Blood pressure and pulse were taken only for the final minute of each rest and stress period in order to minimize discomfort to subjects. Figure 1 depicts the procedure.

Experimental Stimuli

Experimental stimuli consisted of time-limited math problems alternated with time-limited anagram problems arranged in series of 120 problems with equal numbers of each type of problem (e.g., 60 math and 60 anagram). Math problems were two operation arithmetic problems with two or three digit solutions (e.g., 54 - 13 + 72 = ?) Anagram problems were 3, 5 or 7 letter scrambled words (e.g., IRLAT = ?). During stress periods, problems were presented on the television monitor in rapid succession. The amount of time a subject was allowed to solve the problems (e.g., the time-limit), was determined individually. Time-limits ranged from 10-20 seconds (Mean = 16 sec.). A different series of problems was used for each session in order to minimize



Rest = EMG, EDG measured once per minute
SBP, DBP and Pulse measured during minute 5

Stress = Presentation of time-limited tasks
EMG, EDG measured once per minute
SBP, DBP and Pulse measured during minute 3

Figure 1. Session Timeline for Experiments 1 and 2.

practice effects. Subjects responded by typing answers into the computer keyboard with their right hand and then pressing the enter key. For anagrams, a correct answer consisted of any word constructed from the scrambled letters. The computer program was designed to accept all possible answers. Correct answers to math problems were simply the correct mathematical solution. Answers were correct only if they were entered before the expiration of the time-limit. Corrections could be made to typed answers provided they were completed before the time-limit expired. To correct an answer, subjects erased the wrong answer using the backspace key and then typed the new answer.

Levels of Difficulty

Problem series were specially constructed for levels of difficulty by changing the ratio of easy to hard problems. Easy problems were operationally defined as three and five letter anagram (e.g., YFR = FRY or RAWTE = WATER) and two-operation arithmetic problems with 2 digit answers.(77-16+20= 86). Hard problems were five and seven letter anagrams alternated with two-operation arithmetic problems with 3 digit answers (e.g., TORIHSY = HISTORY, 61+85-29= 117). Thus, there was some overlap in the operational definition of easy and hard problems in that both contained five letter anagrams. Levels of difficulty within problem series were objectively constructed at 40% and 60% where the percentage refers to the percent of hard problems relative to the total problems in a series. Within a series of 120 problems,there were four sets of 30 problems each with one of the 2 possible levels of difficulty. For example a 40% difficult series contained four sets of problems each with 12 hard problems and 18 easy problems (i.e., 12 equals 40% of 30). The order of easy and hard problems within a set was random. No attempt was made to independently verify the classification of problems into easy versus hard categories by pretest of percentage correct on the problems. However, this strategy was used in a previous study (Maldonado, 1982) in which percent correct data and informal reports by subjects supported the present classification.

Task Consequences

During Reward (R) conditions subjects earned points that could be exchanged for small amounts of money at the end of the session. Points were displayed on a counter at the top of the television screen.

During Noise (N) conditions subjects received a sharp 90 db auditory blast via headphones for all incorrect responses. Auditory blasts used during Noise conditions were generated by depressing a telegraph key wired to a Hewlett Packard Audio Oscillator (Model 200 ABR) which was set to produce a 1,300 cycle per second tone with a sound pressure of 90 decibels. Table 2 summarizes the experimental conditions for Experiment 1.

In all experimental conditions the computer delivered performance feedback by emitting a high tone for correct responses and a low tone for incorrect responses. Instructions were read to the subjects prior to the start of each stress period and displayed on large colored cards stating the difficulty of the problems as easy or hard, whether points or auditory blasts would be presented and whether some of their correct responses would be rejected. Additionally, subjects were instructed to work as fast as they could and to try to get as many problems correct as possible (see Appendix for instruction scripts).

Table 2

Experimental Conditions Experiment 1

	Task Consequence	Task Difficulty
Condition		
1	Reward	40%
2	Auditory Blast	40%
3	Reward	60%
4	Auditory Blast	60%

Data Reduction and Analysis

The final three readings of the rest period that preceded a specific stress period were averaged to a yield prestress (rest) value for that stress condition. For EMG and EDG (which were measured once per minute) three reactivity scores were calculated for each condition by subtracting theaverage prestress value from the stress values for minutes 1, 2, and 3 of stress induction. For blood pressure and pulse measures (which were measured during minutes 5 of prestress and minute 3 or stress) one reactivity score was calculated by subtracting the value for each stress condition from its corresponding prestress value. All data were then subjected to statistical analysis using an analysis of covariance (ANCOVA) (Dembroski et al., 1979a; 1979b; Weidner

& Matthews, 1978) in order to control for the effects of the the "law of initial values."[1] The ANCOVA analysis was chosen because this approach permits the comparison of changes between rest and stress periods while controlling for the effects of baseline differences. Differences in baseline levels may greatly effect the degree of observed reactivity. For example, the higher the heart rate is before a subject performs a task, the less the heart rate will increase during a task that usually increases heart rate but the more the heart rate will decrease during a task that usually depresses heart rate (Holmes & Zurawski, 1983). The covariate in these analyses was the mean of the last three minutes of the ten minute resting baseline for each session. This value was chosen because it best represented the initial values for each session. The ANCOVA analysis performs a regression analysis between the baseline levels and the reactivity scores. Variability resulting from baseline differences was determined and beta weight values were used to weight the reactivity scores so the effect of baseline variability is weighted out. The new weighted values or adjusted means represent the changes from rest to stress apart from the effects of initial differences. Resulting adjusted means were then graphed for visual analysis of trends. Variability measures can not be determined for the adjusted means as part of the variability has been removed to control for the law of initial values and no acceptable strategy is available to determine standard error for these data. Pairwise comparisons were conducted for all significant differences $p < .05$ using the Tukey HSD analysis. Percent correct data or task performance were analyzed using a analysis of variance (ANOVA) to test for stability across sessions.

[1]The law of initial values asserts that the magnitude of a response to a task is determined not only by the task but also by the level of the activity preceding the task (Wilder, 1962).

Results

The results clearly demonstrate that the experimental stressors were effective in producing physiological adjusted mean reactivity scores across all conditions. Adjusted mean reactivity scores for systolic blood pressure ranged across conditions from 3 to 8 mmHg and from 4 to 8 mmHg in diastolic blood pressure during exposure to experimental stimuli. Adjusted mean pulse reactivity scores ranged across conditions from 2 to 8 beats per minute. Frontalis EMG mean reactivity scores ranged across conditions from 8 to 12 microvolts. Finally, mean skin conductance reactivity scores ranged from 1.0 to 2.0 µmho/mv.

The effects of reward or auditory blast consequences combined with either 40% or 60% task difficulty was evaluated using a three factor (Difficulty x Consequence x Session) repeated measures analysis of covariance (ANCOVA) conducted on mean reactivity scores for each of the five physiological measures; systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse (PUL), frontalis EMG, and skin conductance level (EDG) during task performance. The covariate in these analyses was the mean of the last three minutes of the ten minute resting baseline for each session (see Figure 1.) The resting period scores were used as the covariate to eliminate any potential influence of the law of initial values (Lacey, 1956; Wilder, 1956; 1968). The analysis revealed that there were no significant main effects for either Consequence or Difficulty.for any of the five dependent measures. A significant Consequence by Difficulty interaction was found for skin conductance (EDG) $F, (1,8) = 4.84, p < .05$ where the effects of the Reward consequence combined with the 60% level of difficulty produced the largest adjusted mean reactivity score for EDG.

Figure 2 summarizes the effects of the four experimental conditions on mean

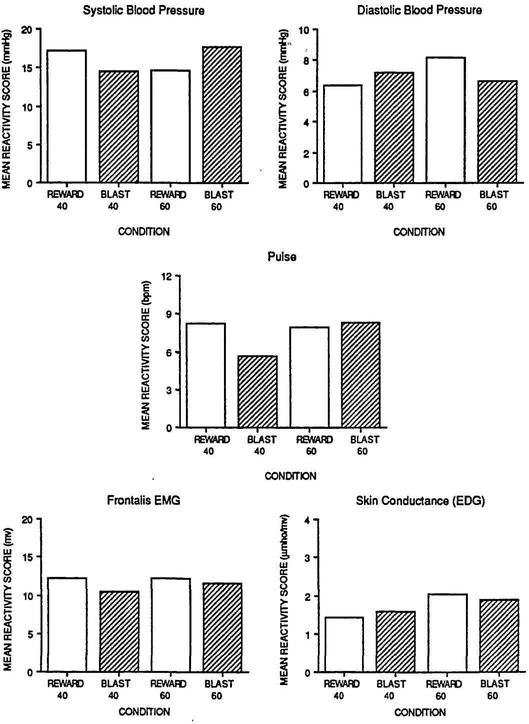


Figure 2. The Effects of Two Task Contingencies (Reward and Auditory Blast) and Two Levels of Task Difficulty (40% and 60% difficult) on Adjusted Mean Reactivity Scores for Systolic Blood Pressure, Diastolic Blood Pressure, Pulse, Frontalis EMG, and Skin Conductance (EDG) Measures.

adjusted reactivity scores for the five measures summarized across all subjects and all sessions. The vertical axis represents mean adjusted reactivity scores and thehorizontal axis depicts the four experimental conditions. The open bars represent Reward conditions and the colored bars represent Auditory Blast conditions.[2] According to Figure 2 mean adjusted reactivity levels differed by condition but the differences were quite small and were inconsistent across measures. Visual examination of the graphs indicates that when subjects received an auditory blastcontingent on incorrect responses, they evidenced slightly greater mean adjusted reactivity scores for systolic blood pressure and pulse rate when performing the more difficult tasks than they did when performing the tasks at the lower level of difficulty. However, this difference was not statistically significant. Figure 2 also depicts a visibly greater mean diastolic blood pressure reactivity for the more difficult task condition compared to the less difficult task condition when subjects performed under Reward conditions but again, this difference was not statistically significant.

Multivariate ANCOVA analyses were performed for all five physiological measures to examine Group by Condition interactions for the Framingham Type A

Scale (FTAS) classification grouping variable. A second set of ANCOVA's were performed to evaluate the effects of beta blocker medication (BETA) on reactivity. Each was a mixed ANCOVA with the relevant grouping factor and the other within

---

[2] Measures of variability are not presented for any of the present figures. The reason for no variability measurements involves the analysis of covariance that was used to analyze those data. The data used in the analysis were adjusted for baseline differences, and the means presented in all figures reflect these adjustments. The statistical package used (BMDP) does not provide variability estimates for these adjusted means. In order to calculate variability estimates, we would need to convert data points to z-scores, calculate an adjusted score for each data point in the analysis based on the regression coefficients provided by the BMDP output, and then convert back to the original units. Thus, it is conventional to omit measures of variability when reporting adjusted means from analyses of covariance.

subjects factors of difficulty, consequence, and session. According to the analysis, there was no significant Group by Condition interactions for Type A classification. However a significant Group by Consequence by Difficulty interaction occurred for the beta blocker medication variable for SBP, $F(1,7)=3.44$, $p < .01$ such that subjects receiving beta blocker medication evinced significantly higher mean reactivity scores than did subjects not on medication during Reward plus 40% Difficulty (i.e., 19.58 mmHg and 15.37 mmHg respectively) and during Auditory Blast plus 60% Difficulty (i.e., 20.16 mmHg versus 15.85 mmHg respectively).

Figure 3 illustrates the effects of the four conditions on adjusted mean reactivity scores for subjects assessed as Type A according to the FTAS. The stripped bars depict reactivity for Type A subjects and the white bars represent reactivity scores for Type B subjects. Visual examination of Figure 3 suggests that Type A subjects differed from Type B subjects in the degree of physiological reactivity to the four conditions. However, there were no consistent patterns or group differences across the five measures. Visual trends indicate differences in reactivity such that Type B group mean adjusted reactivity appeared to be slightly above those produced by Type A group for systolic blood pressure during Reward 40%, Blast 40%, and Blast 60% ; for diastolic blood pressure during Reward 40%, and Blast 60%; for pulse rate during Blast 40% and Blast 60%; and for skin conductance during all four experimental conditions. According to Figure 3, the Type A group produced mean adjusted reactivity scores for frontalis EMG that were consistently above those of the Type B group for all four experimental conditions. None of these graphically observed differences was statistically significant.

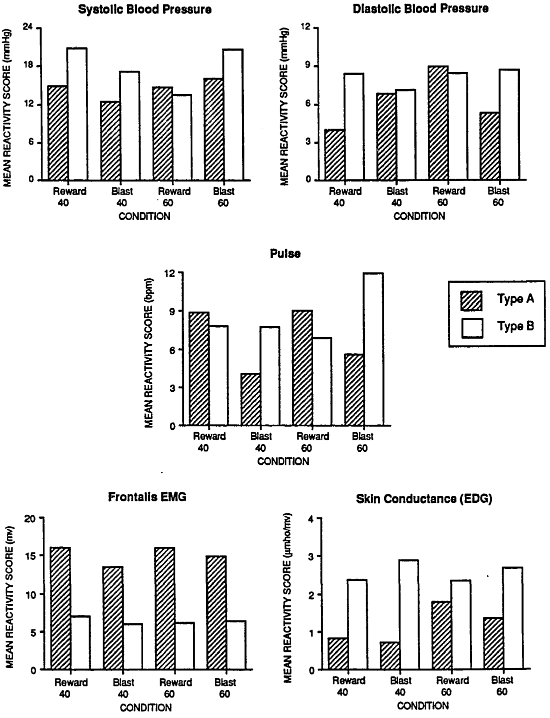


Figure 3. Comparison of Type A and Type B Group Differences for the Effects of Two Task Contingencies (Reward and Auditory Blast) and Two Levels of Task Difficulty (40% and 60% difficult) on Adjusted Mean Reactivity Scores for Systolic Blood Pressure, Diastolic Blood Pressure, Pulse, Frontalis EMG, and Skin Conductance (EDG) Measures.

Discussion

The results of the present experiment indicate that, as expected, varying the level of difficulty and consequence within a standard laboratory stressor may differentially effect patterns of physiological reactivity and that the magnitude of the effects of these variables may vary in different physiological measures. These findings are not surprising given the vast research that has documented response pattern differences in different physiological measures since the 1920s (Darrow, 1929; Davis, Buchwald & Frankmann, 1955; Lacey, 1956). However, the present findings suggest that the two task consequences and two levels of task difficulty employed in this study did not produce the dramatic between condition differences in physiological reactivity that were expected. It was hypothesized that the contingent presentation of the auditory blast would produce higher levels of reactivity than the point reward consequence. It was also expected that the higher level of difficulty (60%) would produce higher levels of reactivity than the lower level (40%). None of the statistical findings supported the first hypothesis, and in fact, when differences were visible, the reward condition was more often the condition that produced higher levels of physiological reactivity. Only the results for skin conductance reactivity supported the second hypothesis, where there was a significant interaction for difficulty and consequence conditions. For both consequence conditions, the higher level of difficulty produced higher levels of skin conductance reactivity than did the lower level of difficulty. Thus the present experimental conditions appeared to impact skin conductance reactivity as expected, but did not similarly affect cardiovascular measures or frontalis muscle tension. Reasons for this outcome are unclear, but may reflect the subjects tendancy to perspire more when performing more difficult tasks rather than any stimulus specific reactivity pattern.

Although no definitive conclusions can be drawn based on the present findings because of the paucity of statistically significant results, it may be worthwhile to discuss the more robust trends that appeared in the graphic presentation of the data. According to the figures, different levels of difficulty affect physiological reactivity differently depending on the consequence in effect. When the consequence was point reward contingent on correct responding, increasing task difficulty produced increases in DBP reactivity and decreases in SBP, PUL, and EMG reactivity. However, when the consequence was an auditory blast contingent on incorrect responding, higher levels of difficulty produced the opposite effect, or decreases in DBP reactivity and increases in SBP, PUL, and EMG reactivity. Reasons for this pattern reversal are unclear. Perhaps the pattern reversal reflects a covariation of cardiac output and body tension that was sensitive to the presentation of an auditory blast only at high levels of difficulty. Or it may be that at the higher level of difficulty, subjects merely gave up and physiological responding was in turn diminished.

According to the trends in the graphic data presentation, there was an additive effect of increased reactivity during the auditory blast condition performed under the higher level of task difficulty . Perhaps when a highly difficult task was presented in conjunction with an unpleasant consequence such as an auditory blast, the negative effects of the combination may have had an interactive effect such that the resulting reactivity scores were higher than they would have been for either higher difficulty alone or auditory blast alone. High difficulty conditions would likely produce more errors and thus more auditory blasts would be presented than during low difficulty periods. It could therefore be that subjects were exposed to more blasts during the low difficulty condition and this difference may account for the observed differences in reactivity. In the present data increases in reactivity did occur for several measures during the high difficulty and auditory blast combination but only for two measures (i.e., skin conductance and diastolic blood pressure) during the high difficulty and reward combination. Unfortunately, the present experiment did not independently assess the effects of consequence and difficulty and so how much of the present effects are due to the effects of the blast can not be specified. Another explanation is that these findings merely represent the unpleasant effects of the the auditory stimulus independent of either its contingent relation to performance or the level of task difficulty. Given that the differences in the levels of reactivity between the two auditory blast conditions were modest across all five measures, this explanation can not be dismissed. A consequence involving only point loss on a counter rather than a tone presentation or a noncontingent auditory blast condition would be desirable alternatives to control for this confound.

The increases in reactivity seen between the conditions of 40% and 60% difficulty were not statistically significant. There are several possible explanations for this. First, it may be that the subject sample was too small to allow for the observed trends to reach significance. Second, perhaps a 20% difference in level of difficulty was not adequate to produce more dramatic differences in reactivity that might have resulted from a larger difference in conditions. It is also possible that subjects were simply not as sensitive to this increment in difficulty as was expected and thus the increment in difficulty was not real. A fourth factor is that the procedure did not control for individual performance and thus if a subject's performance was constant across conditions, they may not have contacted the stimulus changes as expected. For example, if a subjects' performance was poor whether given easy or hard problems, their reactivity would not be expected to reflect the increased difficulty since the conditions were experienced as equally difficult. Unfortunately, no data were collected on subjective perceptions of difficulty. Nor is there any breakdown in performance data by condition. Both of these types of data would be useful in addressing this issue and should be included in future procedures.

The present findings did not provide a demonstration of a reliable interaction of Type A behavior pattern classification and either consequence or difficulty factors. It was hypothesized that the Type A group would be more reactive than the Type B group to the experimental conditions. However, the results of the statistical analysis did not support this hypothesis. Analysis of trends observed in the graphic presentation of the data suggested that there may be consistent group differences in reactivity for skin conductance (B's more reactive than A's) and frontalis EMG (A's more reactive than B's). There wa no dramatic group differences for any of the cardiovascular measures, but the differences that were observed suggested that Type A subjects were more reactive than Type B subjects. Finally, contrary to expectation, neither task difficulty nor task consequence appeared to differentially effect reactivity for any measure in either group. Thus the present experimental manipulations did not replicate the differences in physiological reactivity attributed to either Type A behavior, task difficulty, or task consequencesthat have been reported in the previous literature.

In summary, the findings of Experiment 1 suggest that, while the time-limited math and anagram problems did produce levels of physiological reactivity comparable to those observed in the literature (Holmes & Zurawski, 1983), the manipulations of task consequence and task difficulty were ineffective in producing significantly different degrees of reactivity for any of the five measures. Thus, contingent reward and contingent presentation of an auditory blast are two task consequences that effect reactivity similarly. Furthermore, task difficulty conditions that are defined according to a 20% difference in the ratio of easy to hard problems was not sufficient to produce differential physiological responding in any physiological measure. Future efforts to evaluate the effects of task consequence and task difficulty might prove more successful in detecting existing differences in reactivity with comparisons of consequences other than point rewards and the auditory blast, and with comparisons of levels of difficulty that involve a much larger range between easy and hard task conditions.

EXPERIMENT 2

The less than remarkable findings of Experiment 1 may have been due to the complexity of the design and the failure to evaluate task contingency independent of task difficulty. This complex design may have obscured differences in reactivity that may have been observed within an independent analysis of these two variables. Although the effects of both of these variables may be at work in the physiological responses that occur in natural settings, examination of their effects in combination rather than separately was premature at best. Another difficulty with the procedures of Experiment 1 was the disappointing levels of reactivity produced by the consequence condition involving the auditory blast. Surprisingly, this consequence condition was more often associated with lower levels of reactivity than those produced by the reward consequence condition. The reward condition appeared to produce higher and more consistent levels of reactivity than the auditory blast condition for several measures and thus appears to be a consequence condition warranting further attention. Experiment 2 was an effort to improve on some of the methodological limitations of Experiment 1 while addressing further the relationship of task difficulty, task consequence and Type A behavior classification on physiological reactivity to a standard laboratory stressor.

The purposes of Experiment 2 were (a) to compare the effects of two task consequences (reward and forced failure) and a no consequence control condition on physiological reactivity during exposure to a behavioral stressor while controlling for level of difficulty and (b) to compare the effects of an extremely low level of task difficulty and an extremely high level of task difficulty on reactivity while holding task contingency constant, and (c) to examine possible interactive effects of consequence and difficulty and Type A classification across several physiological measures. In addition, the present experiment involved an attempt to compare task performance for Type A and Type B subjects across sessions.

Method

Setting and Subjects

Experiment 2 was conducted in the identical setting used in the previous experiment. All of the ten subjects who participated in Experiment 1 served as subjects for Experiment 2.

Physiological Measures

The measurement of systolic blood pressure, diastolic blood pressure, pulse rate, skin conductance and frontalis EMG was achieved using the same apparatus and monitoring procedures as used in Experiment 1 and followed the same timeline (see Figure 1).

Type A Measures

The Framingham Type A Scale (FTAS; Haynes et al., 1980) scores from Experiment 1 were used for group assignment for data analysis. As in Experiment 1, five of the subjects were classified as Type A and five were classified as Type B.

Performance Measures

Math and anagram tasks were presented using a Commodore VIC-20 computer and television monitor following the same general procedures as in Experiment 1. Subjects' typed responses were recorded and analyzed to yield the percentage of correct responses and were then summarized by group across the three experimental sessions.

Procedure

The general procedure was essentially the same as in the first study. The major differences were the exclusion of the Auditory Blast consequence, the addition of Forced Failure and Control consequence conditions, and the adjustment in levels of task difficulty to 10% and 90% difficult. As in Experiment 1, subjects attended 3 experimental sessions scheduled to coincide with their regular visits to the institute. Each session was approximately one hour in duration and preceded the cardiac program exercise periods. Figure 1 depicts the session time-line and number of repeated measures for each condition for Experiment 2. Subjects were exposed to three consequence conditions, Reward, Forced Failure, and Control. During these three conditions, tasks were presented at the 10% or easy level of difficulty so that the effects of consequence could be compared. In addition, subjects were exposed to a second Reward condition during which they performed tasks at the 90% or hard level of difficulty. A comparison was performed of the effects of the two levels of difficulty (i.e., 10% and 90%) during Reward consequence conditions. Thus Experiment 2 involved a two factor (i.e., Consequence x Session) repeated measures design with the effects of three consequence conditions assessed across three experimental sessions, and a two factor (i.e., Difficulty x Session) repeated measures design with the effects of two levels of task difficulty assessed across three experimental sessions. Table 3 presents a summary of the experimental conditions for Experiment 2.

Levels of Difficulty

Levels of task difficulty were constructed to enhance the difference between low difficulty and high difficulty by changing the ratio of easy to hard problems to 10% and

90%. Easy and hard problems were defined as in the previous experiment. Low

Table 3

Experimental Conditions Experiment 2

	Task Consequence	Task Difficulty
Condition		
1	Reward	10%
2	Reward	90%
3	Forced Failure	10%
4	Control	10%

difficulty conditions were defined as the presentation of problem series in which the ratio of easy to hard problems was 3 hard problems and 27 easy problems in each set of 30 problems. High difficulty conditions were defined as the presentation of problem series in which the ratio of easy to hard problems was 27 hard problems and 3 easy problems in each set of 30 problems. The order of easy and hard problems within a set was random.

Task Consequences

During Reward (R) conditions subjects earned points that could be exchanged for small amounts of money at the end of the session. Points were displayed on a counter at the top of the television screen. Reward was presented at two levels of difficulty (10% and 90% difficult) in order to determine if differences in task difficulty would result when the difference between easy and hard conditions was expanded beyond those examined in the previous experiment while consequence was held constant.

In the Forced Failure (FF) condition 50% of the subjects responses were rejected by the computer even if they were correct. Such responses were treated as incorrect and thus resulted in presentation of the low computer tone and no point increment on the counter. During forced failure tasks were performed only at the 10% level of difficulty so that consequences could be compared while level of difficulty was held constant.

For the no consequence Control (C) condition, subjects performed tasks as in the reward condition but no tones were presented to signal performance feedback and there was no counter displaying points on the monitor. Tasks were performed during Control only at the 10% level of difficulty for the same rationale as for the FF condition.

Data Reduction and Analysis

Data reduction and analysis procedures were identical to the procedure used in Experiment 1.

Results

To examine the effects of three task consequence conditions, Reward, Forced Failure, and No Consequence Control, a two factor (Consequence x Session) repeated measures analysis of covariance (ANCOVA) was conducted on mean reactivity scores for each of the five physiological measures; systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse (PUL), frontalis EMG, and skin conductance level (EDG). The covariate in these analyses was the mean resting period score for the period immediately following baseline adaptation for each session (see Figure 1). Significant main effects for Consequence were found for SBP, $F(2,17) = 4.23$, $p < .032$. Figure 4 displays the effects of task consequence on mean adjusted reactivity scores for the five dependent measures. Reward produced the largest levels of systolic blood pressure reactivity followed by Forced Failure, and then by the Control condition. A similar trend is depicted in Figure 4 for DBP and EDG but these trends were not confirmed statistically. The figure also indicates that pulse reactivity to the Control condition may have exceeded those produced by the two consequence conditions. Significant session effects were found for EDG, $F(2,17)= 4.52$, $p < .047$, and DBP, $F(2,17)= 7.87$, $p < .004$. These session effects reflect a decrease in reactivity from session 1 to 3 observed for these two measures (i.e., DBP and EDG).

Separate ANCOVA's were performed for the two grouping variables of Type A classification according to the Framingham Type A Scale and presence or absence of beta blocker medication with the two within subjects factors (i.e., Consequence and Session). Results of these analyses indicated no significantly reliable interaction between either grouping variable and either consequence or difficulty. The effects of the three consequences on mean adjusted reactivity scores in Type A and B subjects are presented in Figure 5. Stripped bars represent the reactivity for the Type A group and white bars depict reactivity for the Type B group. The reactivity patterns presented in Figure 5 suggest that the three consequence conditions may differentially effect the five measures and that Type A's may respond differently than Type B's, but the differences varied by measure, were often quite modest, and were not confirmed statistically. For example, compared to the Type A subjects, the Type B subjects seemed to respond with.greater DBP reactivity across all consequence conditions. Similarly, adjusted mean SBP for Type A's appeared to be consistently above those of Type B's for all consequence conditions. Neither of these trends were confirmed in the statistical analysis.

To examine the effects of the two levels of task difficulty, a two factor (Difficulty x Session) repeated measures analysis of covariance (ANCOVA) was conducted on mean reactivity scores for each of the five physiological measures; systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse (PUL), frontalis EMG, and skin conductance level (EDG). The covariate in these analyses was the mean resting period score for the period immediately following baseline adaptation for each session (see Figure 1). The ANCOVA revealed no significant main effect for task difficulty for any measure. Thus extending the difference between the conditions of low and high difficulty did not produce significantly different levels of physiological responding. A significant session effect was found for PUL, $F(2,17)=9.74$, $p<.002$ indicating that pulse rate reactivity consistently decreased across sessions. Separate ANCOVA's were performed for the Type A and beta blocker grouping variables in order to evaluate their predictive effects on reactivity. The analyses revealed no significant interactions between either of the two grouping variables and physiological reactivity for any measure. Table 4 provides a summary of mean adjusted reactivity according to Type A pattern classification, physiological measure, and difficulty condition.

Table 4

Effects of Two Levels of Task Difficulty on Physiological Reactivity
Adjusted Mean Reactivity Score

Measure	Type	Task Difficulty 10%	Task Difficulty 90%
Systolic (mmHg)	A	4.89	14.67
	B	8.05	11.42
Diastolic (mmHg)	A	6.4	12.33
	B	5.93	7.22
Pulse rate (bpm)	A	4.28	3.92
	B	8.83	7.83
EMG (mv)	A	12.94	7.78
	B	6.84	7.85
Skin conductance	A	1.42	1.31
(umho/mv)	B	1.86	2.54

sessions 2 and 3. Figure 6 displays the session effects for the percent correct performance data for the two groups. According to Figure 6, task performance improved between session 1 and 2 for both Type A and Type B groups but did not increase further between sessions 2 and 3. The magnitude of improvement in performance was an increase from 48 to 58 percent correct. Although Figure 6

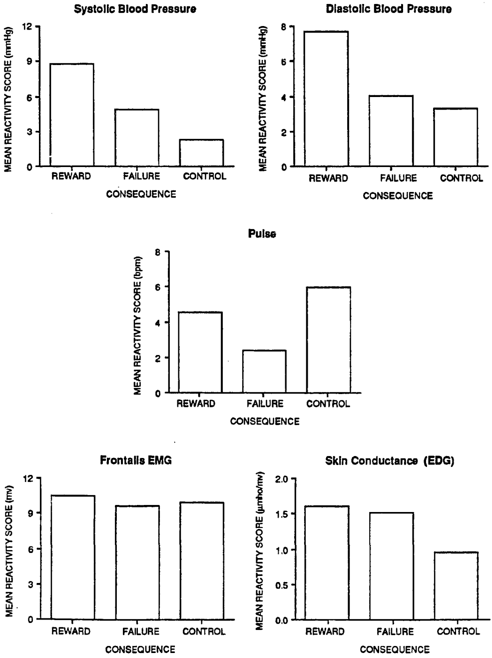


Figure 4. The Effects of Three Task Consequences, Reward, Forced Failure, and Control on Adjusted Mean Reactivity Scores for Systolic Blood Pressure, Diastolic Blood Pressure, Pulse, Frontalis EMG, and Skin Conductance (EDG) Measures.

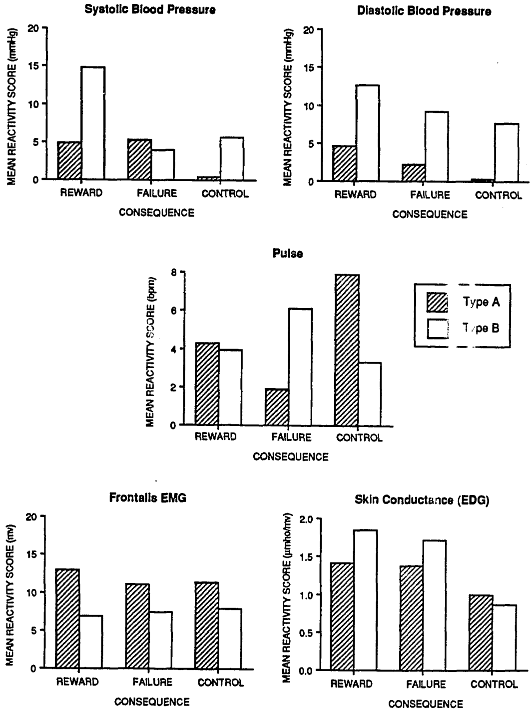


Figure 5. Comparison of Type A and Type B Group Differences for the Effects of Three Task Consequences, Reward, Forced Failure, and Control on Adjusted Mean Reactivity Scores for Systolic Blood Pressure, Diastolic Blood Pressure, Pulse, Frontalis EMG, and Skin Conductance (EDG) Measures.

Table 5

ANOVA Summary for Percent Correct on Task Performance

	df	F	P values for Percent Correct
Group	1	.242	p < .636
Session	2	9.66	p < .002
G x S	2	.040	p < .961

suggests that Type B subjects performed slightly better than Type A's across all three sessions there were no statistically significant differences in performance for the two groups.

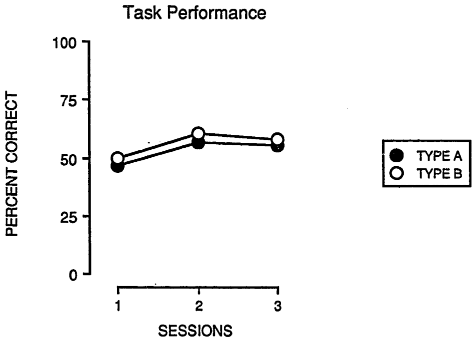


Figure 6. Comparison of Type A and Type B Group Differences for Task Performance (percent correct) Across Sessions 1-3.

Discussion

The findings of Experiment 2 demonstrated differential effects of task consequence on physiological reactivity for only one of the five measures (i.e., systolic blood pressure). For this cardiovascular measure, subjects responded during reward conditions with levels of reactivity that were greater than those that occurred for either the forced failure condition or the no consequence control condition. That this difference was not manifested in diastolic blood pressure, pulse rate, frontalis EMG, or skin conductance is not surprising. According to Holmes (1983) there is a high degree of response specificity in reactivity research and reliable differences are often limited primarily to systolic blood pressure, particularly in studies comparing Type A and Type B classified individuals.

A second finding showed that there were no significant interactions between Type A classification and the effects of either consequence or difficulty. Graphic trends, particularly for blood pressure reactivity, suggest the potiental for an interaction between Type A classification and consequence which warrents some speculation. Based on the observation of Glass (1977) that Type A's display a pattern indicative of learned helplessness when confronted with a task involving continued failure, it was hypothesized that Type A subjects would be more reactive than Type B's to the forced failure condition. Based on the trends that were depicted in the group data, it appeared that Type B subjects were more blood pressure reactive during reward conditions while Type A's responded similarly during reward and forced failure. These trends support the findings of Blumenthal et al. (1983) that Type B subjects show higher reactivity in conditions where incentives are offered (i.e., reward) than they do in conditions where no incentives are offered (control). The present findings for pulse rate reactivity also supported previous findings of Blumenthal et al. (1983) that Type A's respond similarly to Type B's during incentive conditions and evince increased reactivity to nonincentive conditions (i.e., control). Perhaps Type A's are more reactive than Type B's when performing a task without performance feedback whereas Type B's may evince increased reactivity during forced failure when incentives are present but not consistently tied to performance. This explanation is inconsistent with the learned helplessness position proposed by Glass (1977) regarding the Type A tendency to respond more to tasks involving continued failure. It is possible that Type A's were giving up during the control condition while Type B's were continuing to work and respond physiologically. The overall higher reactivity of Type B's for SBP, DBP, and EDG relative to Type A's supports this hypothesis. In any case, the present findings provide some evidence supporting the position that the task consequence in effect during presentation of psychological stressors is an important variable that may effect the patterns of reactivity under study. Failure to hold consequence effects constant in reactivity research may result in confounding of the effects of an independent variable when consequence is not held constant.

Contrary to expectation, the present findings revealed that increasing the range of difference between low difficulty and high difficulty conditions did not result in a clearer demonstration of differential reactivity as a function of task difficulty. In fact, reactivity levels during the extremely easy condition were nearly equivalent to levels during the extremely hard condition. Thus even when efforts are made to control for the effects of task consequence, task difficulty as defined in the present procedures did not appear to differentially affect reactivity. Once again it may be that the manipulations in the problem series did not result in a real or experienced increment in task difficulty. This outcome for both Experiment 1 and 2 suggests that an alternative definition of task difficulty should be used in future research. Efforts should be made in future research to validate conditions of task difficulty prior to their implementation in reactivity studies by collecting data such as percent correct and subjective ratings of difficulty to confirm that the levels that are constructed (a) produce substantially different levels of performance and (b) are experienced by subjects as comparively different in degree of challenge. Different types of difficulty should be included in this validation process because it is likely that tasks are difficult across a ı. .oer of dimensions (i.e., behavioral requirement, time requirement, degree of coordination required, degree of vigilance) and the degree of reactivity each will produce. Unfortunately, because this validation effort was not made in the present studies, we do not know whether the lack of differential reactivity across task difficulty resulted form a failure to arrange tasks of substantially different levels of difficulty or the lack of a causal relationship between reactivity and task difficulty.

The percent correct data revealed improved performance across sessions 1-3. The degree of improvement was approximately 10% and showed a trend toward stability. Practice effects can not be eliminated as a possible confound and may be responsible for the reduction in overall levels of reactivity seen in the later sessions. Previous research generally has not reported performance data and thus no comparisons can be made to the present data. Although the difference was very slight, Type B subjects' task performance was consistently better than A's across sessions. Methodological consideration of practice effects to stressor task performance over time should be included in future research to allow for analyses and comparisons of the effects of different stressor tasks on reactivity while accounting for changes in performance over time. Efforts should be made to collect performance data, to examine changes in performance over time, and to account for the presence or absence of changes in reactivity which may result from correlated changes in performance.

In summary, the results for Experiment 2 provided no dramatic or decisive demonstration of the specific effects of either task difficulty or consequence. Instead, there was widely varying effects across measures and the differences in reactivity produced by the various consequence conditions were quite modest. With regard to Type A and B group differences, the present study revealed no significant effects for either consequence or difficulty. Although the differences between the Type A and B groups were not as dramatic as expected, the nonsignificant trends for differential responsivity to the different consequence conditions were noted.

EXPERIMENT 3

The third experiment was a study of the effects of social stress situations which incorporate hostility, a factor that has been designated as a primary link in the relationship between Type A behavior pattern and coronary artery disease (CAD), on reactivity. According to Dembroski et al. (1984) attempts to determine which psychological aspects of the global Type A construct are the most "toxic" have pointed to the components of anger and hostility as most highly correlated with CAD. The Duke study conducted by Dembroski et al. (1984) evaluated hostility using the Ho Scale of 50 MMPI items as a measure of hostility. In Experiment 3 of the present studies, the role of laboratory tasks which incorporate an element of social interactions that commonly elicit hostility was assessed. The research question was whether Type A and B subjects would show a more dramatic difference in reactivity to tasks that involve this theoretically relevant Type A behavior pattern feature and whether this difference would be less evident during exposure to standard laboratory stressors with no specific theoretical relevance to Type A behavior pattern.

In a series of two experiments, Goldband (1980) attempted to identify a number of possible Type A behavior relevant task features that might be related to the differences in cardiovascular reactivity observed between Type A and Type B individuals. In the first experiment, male undergraduates performed a standard reaction time task with and without stress relevant to the Type A behavior pattern. The conditions constructed to include features theoretically relevant to Type A behavior were Competition, Time Urgency, and Loss of Control. A neutral or nonrelevant task consisted of the reaction time task performed according to standard procedures. Competition consisted of the reaction time task with the delivery of additional instructions that emphasized that the subject" with the assumption that subjects would compete with this standard. During Time Urgency, time pressure was added with the introduction of a performance based deadline such that no matter how fast the subject responded, the deadline was missed on approximately half the trials. In the Loss of Control condition subjects had no control over presentation of performance feedback. The results showed that the physiological responses of Type A subjects were greater in the relevant stress compared to the neutral task condition while the responses of the Type B subjects did not differ across the two conditions. In the second experiment, Type A and B subjects performed a task that was not theoretically relevant to the Type A behavior pattern. No differences in physiological responding were observed between Type A and Type B subjects under these conditions.

Attempting to analyze the relevance of a task to Type A behavior as a task variable is wrought with procedural and conceptual problems. Like task difficulty, theoretical relevance is not easily objectified. Rosenman and Friedman characterized their "coronary prone" patients as aggressive, hard-driving, competitive, impatient, and time urgent. It is apparent that Goldband (1980) was attempting to incorporate these early subjectively defined elements into his experimental conditions. Research subsequent to the early Type A definition has worked to objectify the Type A construct through component analyses and direct behavioral observation (Dembroski et al., 1978; Matthews et al., 1984). In more recent years, Type A has been regarded as a behavior pattern with focus on specific behavioral differences such as speech, choice of tasks, and response to hostility (Houston, 1986). Thus we do not know what task features are more or less relevant to Type A. Furthermore, these features may not be easily isolated for manipulation in experimental procedures.

In the Goldband (1980) study these problems of definition are apparent in that nonrelevant and relevant conditions differed on a number of overlooked dimensions. The time-urgency condition incorporated changes in instructions, task difficulty, and forced failure. The authors failed to mention that reaction time tasks generally have a built in time-based criteria that would have been in effect across all conditions. During competition no efforts were made to verify whether subjects were in fact competing with the performance of the average subject described in the instructions they received. Subjects in the loss of control condition were never actually exposed to a condition where they did have control of feedback so that the two control conditions actually differed in type of feedback rather than loss of control of feedback. Thus the results obtained in this study may be due to any one of the "nonrelevant" and uncontrolled factors.

Experiment 3 assessed the effects of TAB relevant tasks that are more objectively defined and incorporate the more recent formulation of Type A behavior pattern as closely tied to situations of anger and hostility (Dembroski et al., 1984). Specifically, the hypothesis is that the differences obtained by Goldband (1980) between TAB relevant and nonrelevant tasks may have been due to the degree to which they elicited physiological responses associated with anger (e.g., impatience, competition, and hostility). These elements are most commonly encountered in social situations. Thus the primary differences between TAB relevant and nonrelevant tasks in this study was the presence or absence of a requirement to interact with another person. Nonsocial tasks were defined as mental arithmetic and a computer math task with a rapid time-limit (similar to reaction time tasks). The comparative effects of these tasks were to be examined first as general stressors and then for possible differential effects on subjects classified as Type A or B according to the FTAS assessment. It was hypothesized that (a) the two nonsocial tasks would produce similar patterns of reactivity, (b) that the social interaction tasks would produce similar reactivity patterns, and (c) that Type A subjects would evidence greater reactivity to the social interaction tasks than to the nonsocial tasks.

Method

Subjects and Setting

Nine of the participants in Experiment 1 served as subjects. Subject WS did not participate. According to the Framingham Type A Scale, five subjects were assessed as Type A and four as Type B. The experiment was conducted in the same location as Experiment 1.

Procedure

Experiment 3 was conducted to compare physiological reactivity of Type A and Type B subjects during performance of tasks requiring social and nonsocial interaction. Subjects attended one assessment session in which they were exposed to five stressor tasks following the same session format and measurement procedures used in Experiment 1 and 2 except that there was one additional rest period and one additional stress period. The social interaction conditions were Impatience, Competition, and Hostility. Nonsocial task conditions were Mental Arithmetic and Computer Arithmetic.

Nonsocial Task Conditions

The Mental Arithmetic (MA) condition required that the subjects start at 100 and count backwards by 7's as quickly as possible within the 5 minute stress period. If they finished before the allotted time they were instructed to start over at 100 again. If an error was made, the correct answer was given with the instruction to continue. Points were earned for correct answers and were exchangeable for money at the end of the session.

The Computer Arithmetic (CA) task required that subjects indicate whether or not a number that was flashed on the monitor was divisible by three. This task resembled a reaction time task in the sense that subjects responded by pressing one of two keys on the keyboard within a 3 second time limit. The computer emitted a high tone for correct answers and a low tone for incorrect or late answers. Points were earned for correct answers and were exchanged for money at the end of the session. No correction procedure was available for this task because of the rapid timing.

Social Interaction Conditions

During the Impatience (IMP) task, the subject performed a modified game of Perfection with a confederate. The game was divided into four 1 minute segments, two completed by the subject and two by the confederate. The game board consisted of 20 plastic pieces with numbers on them. The pieces were placed on the board with the numbers down. The players were to turn over the pieces and place them on pegs in correct numerical sequence starting at one and progressing to 20. Only the next piece in the sequence could be placed on the pegs. Subjects could turn over pegs to see numbers but had to turn them back over if it was not the next peg in the sequence. The order of players was always confederate, subject, confederate, subject. The element of impatience was constructed by having the confederate fumble on his turns, wasting time and making frequent mistakes. The goal of the task was for all the pegs to be placed in numerical before the time-limit. If this was accomplished the players could split a five dollar prize. However, if they failed, no money was earned.

During the Competition (COM) condition subjects competed with a confederate for a five-dollar prize. Each player was given 25 scrabble pieces which could be arranged to make five, five-letter words. The first player to create all five words within the three minute time-limit was the winner. In this case the confederate who already had knowledge of the correct answers was to obnoxiously brag about the ease of the task (e.g.,"Oh, this is a piece of cake") criticize the words being formed by the subject (e.g.,"Is that a word?") and keep the odds very close by completing his words only one ahead of the subject.

In the Hostility (HOS) stress condition subjects role-played a social situation which they had described previously as one involving another person who made them angry. The confederate played the part of the other person and was instructed to exaggerate the characteristics that the subject had described as irritating. Table 6 summaries the experimental conditions for Experiment 3.

Data Reduction and Analysis

Data were subjected to analysis of covariance (ANCOVA) as in Experiment 1 and adjusted means were summarized graphically for visual analysis of trends for condition effects and condition by group interactions.

Table 6

Experimental Conditions Experiment 3

Condition		Task
Nonsocial	1	Mental Arithmetic
	2	Computer Arithmetic
Social	3	Impatience
	4	Competition
	5	Hostility

RESULTS

A mixed-model analysis of covariance (ANCOVA) was performed to analyze the effects of tasks involving social interaction on p[illegible]ogical reactivity. The grouping factor in the analysis was Type A as assessed by the Framingham Type A Scale and the repeated measures factor was task Condition. The covariate, as in Experiment 1 and 2, was the mean resting value following the session baseline. According to the ANCOVA there were no significant main effects for any task condition. The analysis did reveal a significant Group by Condition effect for PUL, $F(2,27)= 3.39$, $p < .036$ such that pulse rate reactivity levels produced for Type A subjects' during Competition were substantially greater than those produced by Type B subjects.

Figure 7 summarizes the effects of the five task conditions on reactivity summarized across Type A and Type B sujbects for all five dependent measures. Although the differences were not significant, the figures show a general trend for greater levels of reactivity during Impatience, Competition, and Hostility conditions across all physiological measures with the exception of EDG. The Impatience task condition appeared to produce the highest elevation in frontalis EMG and SBP. The Computer Arithmetic task produced the lowest levels of reactivity in DBP, SBP, PUL, and EDG. Social stress conditions appeared to produce slightly higher elevations than nonsocial tasks for DBP, PUL, and EMG. Skin conductance data indicate no differential effects for any condition.

Figure 8 displays the effects of social versus nonsocial tasks on reactivity scores for Type A and B groups assessed according to the FTAS. The pulse datashow that Type A subjects were more reactive than Type B subjects for Competition. Type B's actually showed a mean decrease in pulse reactivity score during the Competition condition. In contrast, Type B subjects evidenced much greater pulse reactivity to Hostility than did Type A subjects. Diastolic data depict widely differing group reactivity patterns across tasks. Type B subjects' DBP reactivity scores were greater than those for Type A subjects during Computer Math, Competition, and Hostility conditions while Type A subjects responded with higher DBP during Mental Arithmetic and Impatience. Type B subjects showed much higher SBP reactivity during the Hostility condition. Type B subjects showed higher EDG reactivity relative to Type A subjects for Mental Arithmetic, Computer Math, and Impatience while Type A subjects had higher mean EDG reactivity scores during Competition and Hostility tasks. Type B's were consistently more EMG reactive than Type A's across all task

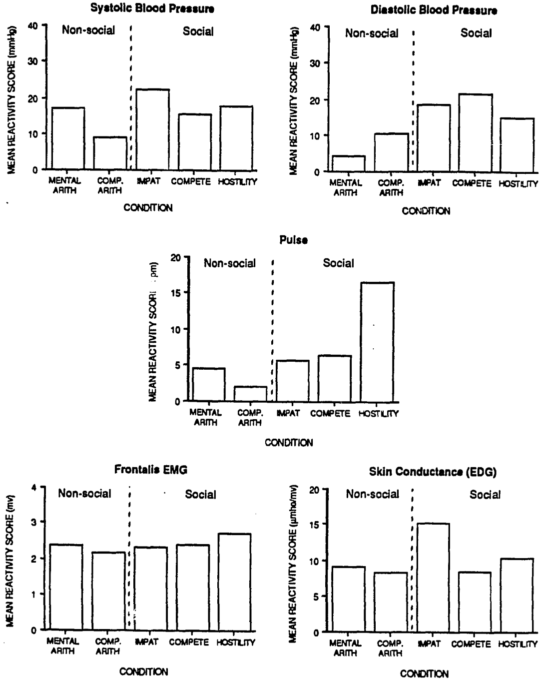


Figure 7. The Effects of Nonsocial Tasks (Mental Arithmetic and Computer Arithmetic)and Social Tasks (Impatience, Competition, and Hostility) on Adjusted Mean Reactivity Scores for Systolic Blood Pressure, Diastolic Blood Pressure, Pulse, Frontalis EMG, and Skin Conductance (EDG) Measures.

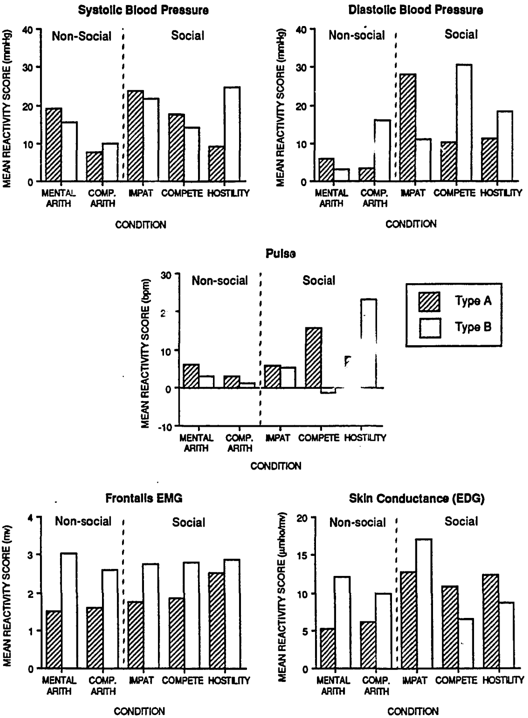


Figure 8. Comparison of Type A and Type B Group Differences for the Effects of Non-social (Mental Arithmetic and Computer Arithmetic) and Social Tasks (Impatience, Competition, and Hostility) on Adjusted Mean Reactivity Scores for Systolic Blood Pressure, Diastolic Blood Pressure, Pulse, Frontalis EMG, and Skin Conductance (EDG) Measures.

conditions.

Discussion

The present findings indicate that the degree of reactivity produced by the five experimental tasks did not differ according to the presence of a social stress component with theoretical relevance to Type A behavior pattern as expected. Tasks that contained a social stress component did produce greater levels of physiological reactivity than did nonsocial tasks but the differences were moderate and inconsistent. While similar patterns of reactivity occurred for SBP and EDG in response to the experimental conditions, it appeared that, overall, the five tasks produced different patterns of reactivity for each physiological measure.

The effects of Mental Arithmetic (a nonsocial task) were in most cases comparable to those produced by the social stress conditions. The consistently lower levels of reactivity during Computer Math might best be explained as due to familiarity, since subjects had extensive practice in computer math in the previous experiment. The three social stress conditions did produce comparable reactivity patterns with the exception of two cases. Pulse reactivity in the Hostility condition was markedly higher than in all other conditions, as was EDG reactivity during the Impatience condition. The results are in agreement with the research findings of Krantz, et a 986), showing that sometimes the distribution of reactivity responses for one task is retained for a second or third task even when the tasks are conceptually different while at other times, there is less consistency across tasks or the observed consistency occurs on some response parameters but not others. These authors further point out that in studies where cardiovascular responses are reliably correlated across task conditions, the strength of these associations tends to be only moderate. That the correlations do exist may be due to inflated generalization across tasks because of common setting conditions A second explanation is that the added reactivity observed during the social tasks is in fact, due to the presence of another person and thus the social nature of the stressors. Finally, it may be that the social interaction tasks more closely approximate natural-life stressors and produced higher levels of reactivity than would a standard laboratory task. While any of these explanations are plausible, the different patterns across measures make it difficulty to discount one or the other. One added point of interest is that Mental Arithmetic, a commonly used laboratory stressor in previous research, did not evoke changes as high as those observed for the relevant tasks for measures of DBP or PUL. This strongly suggests that although this task is simple to use in reactivity procedures, future research efforts might opt for other tasks that evoke larger physiological responses, perhaps social stressors.

Group data were highly inconsistent across experimental conditions but did reveal some interesting differences. One interesting finding was that there were markedly high levels of systolic blood pressure and pulse rate reactivity in Type B subjects during Hostility conditions. This result is consistent with findings of previous research that indicate that reliable differences between Type A individuals and Type B individuals most often are found for systolic blood pressure (Holmes & Zurawski, 1983).

Taken together these data suggest that the social aspects of the tasks may not be valid explanations for the group differences in reactivity observed here. Rather it appears that there is differences in physiological responses to various tasks independent of Type A as measured by the Framingham Type A Scale. The resu..ing differences might be due to unspecified and unprogramme. consequences (i.e., reward for Mental and Computer Arithmetic, forced failure for Impatience and Competition, and punishment for Hostility) or some other unidentified variable such as degree of difficulty or idiosyncratic subject histories that establish some of the tasks as more stressful than others. These alternative explanations await further research.

Generally, the present findings agree with the reports by Goldband (1980) in that the social tasks that were relevant to global Type A behavior pattern produced greater reactivity than nonsocial tasks. Contrary to the Goldband findings, the present experiment did not find the physiological responses of Type A subjects to be consistently greater during social tasks with relevance to TAB compared to the nonsocial tasks nor were the responses of Type B subjects similar during social and nonsocial conditions. These conflicting findings may stem from procedural differences. Goldband used one task and varied elements of competition, time urgency and loss of control within that single task. In this experiment, each condition involved an entirely different task. Additionally, the studies differed in the TAB assessments used, Goldband assigned groups according to the Jenkins Activity Survey while the present study utilized the Framingham Type A Scale. Thus the present findings may simply provide evidence of the limited value the the Framingham assessment as a predictor of reactivity differences between Type A and B individuals which may have been more evident when the Jenkins Activity Survey or Structured Interview are used.

There is another interpretation for the present findings that should be considered. In all phases of the present experiments, a great deal of individual variability in physiological responding was observed. Although some of the variability was controlled in terms of consideration of the law of initial values, the presence of this wide variability can not be ignored. Perhaps reactivity is determined not on the basis of specific or categorizable behavior patterns or physiological patterns to various levels of task difficulty or task consequence and so forth. Rather, reactivity may be reflected as the individual pattern of physiological responding that reflects ones unique physiology and learning history. If so, the conclusions we may draw based on the patterns observed from summaries of many individuals' physiological patterns may be less valuable than those we draw from studies focused on individual data and individual histories. Future research is suggested that will test this hypothesis by comparing the effects of various stressors on individual physiological reactivity patterns while attempting to account for individual histories.

A final point is worthy of consideration with regard to the small differences in physiological responding between Type A and Type B subjects. In their review of the reactivity literature, Holmes and Zurawski (1983) point out that in the limited studies that have reported reliable differences in reactivity between Type A and B subjects, the differences have been quite small (i.e., mean differences for heart rate of 5.7 bpm, 9.13 mmHg for systolic blood pressure, and 5.46 mmHg for diastolic blood pressure)and are of questionable clinical importance. It may be that the small degree of difference is due to the limited effectiveness of laboratory tasks to elicit the degree of reactivity that could be occurring in real-life settings. Another factor is that it may be that magnitude of reactivity is not the critical parameter and that instead, the link between reactivity and disease development is due to some other dimension of stress such as the frequency of exposure. Some evidence does exist that suggests that Type A individuals more often choose high challenge situations (Ortega & Pipal, 1984) that are Type B individuals.

METHODOLOGICAL CONSIDERATIONS

Several methodological weaknesses in the present studies should be addressed. First, if we evaluate the present findings within the scope of group research, the small number of subjects from a specialized population require that the findings be taken with caution since a larger number may have produced quite different patterns of reactivity. If we evaluate the results according to the previous individual variability argument, the results suggest that we have a weak and poorly understood variable, Type A behavior, that must be further refined before we may effectively study its relationship to other disease related variables. Second, the complexity of the design of Experiment 1 where difficulty and consequence variables were evaluated in combination may have obscured the effects of these variables had they been evaluated independently as they were in Experiment 2. On the other hand, since performance consequences are always in effect during any task, analysis of the interaction of these two variables may provide a new and valuable contribution to the reactivity literature.

Finally, the choice of a post-coronary population limits the generality of the findings since this group does not represent the typical Type A or Type B individual. At least three previous studies have examined reactivity in this subgroup (Corse et al., 1982; Dembroski et al., 1979b; and Krantz et al., 1981) suggesting that there is some value to examining reactivity in a group where the effects of pathogenic proces ave emerged. Such an examination provides information about the degree to which hyperresponsivity to environmental stress may persist despite the or. et of illness and associated lifestyle changes.

SUMMARY AND CONCLUSIONS

The present experiments examined the effects of three task variables on reactivity: task difficulty, task consequence, and social factors related to TAB. The results for the evaluation of task difficulty suggest that the objectively defined levels of difficulty used in the first two experiments were not effective in producing different levels of reactivity for any measure. Given the many definitions of difficulty and the inconsistent findings regarding the effects of task difficulty in the reactivity literature, there is growing evidence that this variable is multidimensional and thus not readily analyzed. Difficulty may be a variable that is not independent of consequence as in cases where increased difficulty requires the availability in additional incentives for performance or where exposure to aversive consequences follow poor performance on difficult tasks or vice versa on easy tasks. The problem may be resolved in part by attempts to independently validate the conditions of difficulty via the collection of subjective data in future research. Such a validation is needed in order to assess whether programmed levels of difficulty are in fact different from one another.

A second finding in the present research was that task consequences involving reward or forced failure produce higher degrees of systolic blood pressure reactivity as well as different patterns of reactivity across all measures compared to conditions with no programmed consequence. These findings are in agreement with previous research that has demonstrated that the addition of a performance incentive increases physiological responding during stress induction (Blumenthal et al., 1983) but produces no reliable interactions with Type A and Type B grouping (Manuck & Garland, 1979).

Although the present findings did not reveal consistent differences between Type A and Type B subjects, several interesting factors emerged. The finding that Type A subjects tended to show less reactivity to the task conditions involving higher levels of difficulty supports previous research findings of Glass et al. (1980). According to Glass et al. (1980) when tasks become increasingly impossible to complete successfully, as in the Seligman (1970) learned helplessness model, the physiological responses of Type A individuals are indicative of decreased reactivity. Finally, tasks involving social interaction were not found to produce reliably higher levels of reactivity as has been reported in previous research. No consistent differences were observed for the effects of social interaction tasks on Type A versus Type B subjects. The question of the usefulness of the focus on Type A behavior pattern in the reactivity literature is raised in light of the possibility that patterns of reactivity may be more a matter of individual differences in responsivity, history, and physiology than the result of a specific behavior pattern. Further research efforts should be directed at delineating the differential effects of these three task variables on physiological reactivity, in larger and more varied populations, and with special attention to task consequences.

APPENDIX

Instructions delivered to subjects during experimental sessions

Experiment 1

Rest period instructions

"For the next five minutes I would like you to rest quietly while your physiological responses are being monitored. Please minimize unnecessary movement. I will inform you when the rest period is over."

Stress period instructions

"For the next three minutes you will be asked to solve time-limited math and anagram problems using the computer. Please work as quickly as possible while trying to make as few mistakes as possible. If you make a mistake you may use the backspace key to make corrections. Don't forget to press the return key to enter your answer. Begin responding when the first problem appears on the screen."

Experiment 2

During Experiment 2 instructions were delivered verbally and displayed on colored cards.

Rest period instructions

The rest period card read simply "REST". Verbal instructions were as stated above.

Stress period instructions

The stress period instructions were presented on colored cards in front of the subjects. The information on each card is described below for each consequence. Verbal instructions were stated as follows; "During the next five minutes you will be asked to solve math and anagram computer problems. During this period you will work easy (hard) problems, the computer will accept all of your correct responses (reject some of your responses), you will (will not) earn money for correct responses, and will (will not) receive a low buzz from the computer for incorrect answers.

	Reward	Forced Failure	Control
1.	Easy (Hard)	Easy	Easy
2.	Accept	Reject	Accept
3.	Money	Money	No money
4.	Buzzer	Buzzer	No buzzer

Experiment 3

Rest period instructions: (same as above)

Mental Arithmetic:

"For the next three minutes I would like you to perform a mental arithmetic task. When I say begin, please start at 100 and count backwards by subtracting by seven's. Ready, begin."

Computer Arithmetic:

"For the next three minutes I would like you to perform a new computer arithmetic task. Numbers will flash on the TV monitor. If the number is divisible by 3, press the YES button. If the number is NOT divisible by 3, press the NO button. You do not have to press return. Ready begin.

Impatience:

"For the next four minutes you will work with your partner to complete a task that resembles concentration. If you are successful in completing the task before the time expires you will earn five dollars. You will take turns placing the yellow pegs in the correct sequence on the board. Turn over pegs to read their numbers. Pegs must be placed only one at a time and only the next peg in the sequence can be placed. If you pick the wrong peg you must turn it back over and try again. You will each have two turns of one minute each. Any questions? Okay begin."

Competition:

"For the next task, you will compete with each other in a word game. Each of you are provided with 25 scrabble pieces that will form five five-letter words. The first person to complete all five words will win five dollars. You will have three minutes. Ready, begin."

Hostility:

"For the next three minutes you and your partner will roleplay the situation you discussed previously as a situation where someone aggravated you or was hostile toward you. Please continue the role-play until you are told to stop." (The situation was reviewed and the confederate began the exchange).

BIBLIOGRAPHY

Benson, H., & Gutman, M. C. (1974). The relation of environmental factors to systemic arterial hypertension. In R. S. Eliot, (Ed.), Contemporary problems in Cardiology, Vol I: Stress and the heart, (p. 22). New York: Futura.

Blumenthal, J. A., Lane, J. D., Williams, R. B., McKee, D. C., Haney, T., & White, A. (1983). Effects of task incentive on cardiovascular response in type A and type B individuals. Psychophysiology, 20, 63-70.

Blumenthal, J. A., O'Toole, L. C. & Haney, T. (1984). Behavioral assessment of the type A behavior pattern. Psychosomatic Medicine, 46, 415-423.

Contrada, R. J., Glass, D. C., Krakoff, L, R., Krantz, D. S., Kehow, K., Isecke, W., Collins, C., & Elting, E. (1982). Effects of control over aversive stimulation and type A behavior on cardiovascular and plasma catecholamine response. Psychophysiology, 19, 408-419.

Contrada, R. J., Wright, R. A., & Glass, D. C. (1984). Task difficulty, type A behavior pattern, and cardiovascular response. Psychophysiology, 21, 638-646.

Corse, C. D., Manuck, S. B., Cantwell, J. D., Giordani, B., & Matthews, K. A. (1982). Coronary-prone behavior pattern and cardiovascular response in persons with and without coronary heart disease. Psychosomatic Medicine, 44, 449-459.

Darrow, C. W. (1929). Differences in psychological reactions to sensory and ideational stimuli. Psychological Bulletin, 26, 185-201.

Davis, J. F. (1952). A manual of surface electromyography. Montreal: Laboratory for Psychological Studies, Allen Memorial Institute of Psychiatry.

Davis, R. C., Buchwald, A. M., & Frankmann, R. W. (1955). Autonomic and muscular responses, and their relation to simple stimuli. Psychological Monographs: General and Applied, 69, 1-71.

DeBacker, G., Kornitzer, M., Kittel, F., Bogaert, M., VanDurme, J. P., Rustin, R. M., Degre, C., & De Schaepdrijver, A. (1979). Relation between coronary-prone behavior pattern, excretion of urinary catecholamines, heart rate, and heart rhythm. Preventive Medicine, 8, 14-22.

Dembroski, T. M., MacDougall, J. M., Herd, J. A., & Shields, J. L. (1979a). Effects of level of challenge on pressor and heart rate responses in type A and type B subjects. Journal of Applied Social Psychology, 9, 209-228.

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

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

Dembroski, T. M., MacDougall, J. M., Williams, R. B., Haney, T. L., & Blumenthal, J. A. (1984). Components of type A, hostility, and anger-in: Relationship to angiographic findings. Psychosomatic Medicine, 46, 109-114.

Eliot, R. S. (1979). Stress and the major cardiovascular disorders. Mount Kisko, New York: Futura.

Fey, S. G. & Lindholm, E. (1975). Systolic blood pressure and heart rate changes during three sessions involving biofeedback or no feedback. Psychophysiology, 12, 513-519.

Frankenhaeuser, M., Lundberg, U., & Foresman, L. (1980). Dissociation between sympathetic-adrenal and pituitary-adrenal responses to an achievement situation characterized by high controllability: Comparison between type A and type B males and females. Biological Psychology, 10, 79-91.

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

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

Gastorf, J. W. (1981). Physiologic reaction of type a's to objective and subjective challenge. Journal of Human Stress, 7, 16-27.

Glass, D. C. (1977). Behavior patterns, stress, and coronary disease. Hillsdale: New Jersey: Lawrence Erlbaum.

Glass, D. C., Krakoff, L. R., Contrada, R., Hilton, W. F., Kehoe, K., Mannucci, E. G., Collins, C., Snow, B., & Elting, E. (1980). Effect of harassment and competition upon cardiovascular and plasma catecholamine responses in type A and type B individuals. Psychophysiology, 17, 453-463.

Goldband, S. (1980). Stimulus specificity of physiological response to stress and the type A coronary-prone behavior pattern. Journal of Personality and Social Psychology, 19, 670-679.

Haynes, S. G., Feinleib, M. & Kannel, W. B. (1980). The relationship of psychosocial factors to coronary heart disease in the framingham study: III. Eight-year incidence of coronary heart disease. American Journal of Epidemiology, 111, 37-58.

Holmes, D. S. (1983). An alternative perspective concerning the differential psychological responsivity of persons with the type A and type B behavior patterns. Journal of Research in Personality, 17, 40-47.

Holmes, D. S., McGilley, D. C. & Houston, B. K. (1984). Task-related arousal of type A and type B persons: Level of challenge and response specificity. Journal of Personality and Social Psychology, 46, 1322-1327.

Holmes, D. S. & Zurawski, R. M. (1983). Cardiovascular responses to stressors by persons with type A and type B behavior patterns: A review of the laboratory research. Unpublished manuscript.

Houston, B. K. (1986). Psychological variables and cardiovascular and neuroendocrine reactivity. In K. A. Matthews, S. M Weiss, T. M. Dembroski, B. Falkner, S. B. Manuck, & B.W. Redford (Eds.), Handbook of stress, reactivity, & cardiovascular disease (pp. 207-230). New York: John Wiley.

Jorgensen, R. S., & Houston, B. K. (1981). Family history of hypertension, gender, and cardiovascular reactivity and stereotypy during stress. Journal of Behavioral Medicine, 4, 175-189.

Keys, A., Taylor, H. L., Blackburn, H., Brozed, J., Anderson, J. T., & Solmonson, E. (1971). Mortality and coronary heart disease among men studied for 23 years. Archives of Internal Medicine, 128, 201-214.

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

Krantz, D. S., Glass, D. C. & Snyder, M. L. (1974). Helplessness, stress level, and the coronary-prone behavior pattern. Journal of Experimental Social Psychology, 10, 284-300.

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

Krantz, D. S., Manuck, S. B. & Wing, R. R. (1986). Psychological stressors and task variables as elicitors of reactivity. In K. A. Matthews, S. M Weiss, T. M. Dembroski, B. Falkner, S. B. Manuck, & B.W. Redford (Eds.), Handbook of stress, reactivity, & cardiovascular disease (pp. 85-108). New York: John Wiley.

Lacey, J. (1956). The evaluation of autonomic responses: Toward a general solution. Annals of the New York Academy of Science, 67, 123-164.

Lane, J. D., White, A. D. & Williams, Jr. R. B. (1984). Cardiovascular effects of mental arithmetic in Type A and type B females. Psychophysiology, 21, 39-46.

Lippold, O. C. J. (1967). Electromyography. In P. H. Venables and I. Martin (Eds.), A manual of psychophysiological methods (pp. 126-141). Amsterdam: North-Holland.

Lovallo, W. R., & Pishkin, V. (1980). A psychophysiological comparison of type A and B men exposed to failure and uncontrollable noise. Psychophysiology, 17, 29-36.

Lundberg, U., & Foresman, L. (1979). Adrenal-medullary and adrenal-cortical responses to understimulation and overstimulation: Comparison between type A and type B persons. Biological Psychology, 9, 79-89.

MacDougall, J. M., Dembroski, T. M., & Krantz, D. S. (1981). Effects of types of challenge on pressor and heart rate responses in type A and B women. Psychophysiology, 18, 1-9.

Maldonado, A. J. (1982). The effects of progressive muscle relaxation training under stressful and nonstressful conditions on the management of high blood pressure. Unpublished masters thesis, Western Michigan University, Kalamazoo.

Manuck, S. B. & Garland, F. N. (1979). Coronary-prone behavior pattern, task incentive and cardiovascular response. Psychophysiolog 16, 136-142.

Manuck, S. B. Harvey, A. E., Lechleiter, S. L., & Neal, K. S. (1978). Effects of active coping on blood pressure responses to threat of aversive stimulation. Psychophysiology, 15, 544-549.

Manuck, S. B., Kaplan, J. R. L., & Clarkson, T. B. (1983). Behaviorally-induced heart rate reactivity and atherosclerosis in cynomolgus monkeys. Psychosomatic Medicine, 45, 95-108.

Manuck S. B., Kaplan, J. R. L., & Clarkson, T. B. (1985). Stress-induced heart rate reactivity and atherosclerosis in female macaques. Psychosomatic Medicine, 47, 90. (Abstract)

Matthews, K. A., Weis, S. M., & Detre, T. (1984). Status of and prospects for stress, reactivity, and cardiovascular disease: Themes from the NHLBI-University of Pittsburgh working conference. Behavioral Medicine Update, 6, 7-9.

Obrist, P. A., Gaebelein, C. J., Teller, E. S., Langer, A. W., Grignolo, A., Light, K. C., & McCubbin, J. A. (1978). The relationship among heart rate carotid dP/dt and blood pressure in humans as a function of the type of stress. Psychophysiology, 15, 102-115.

Ortega, D. F., & Pipal, J. E. (1984). Challenge seeking and the type A coronary-prone behavior pattern. Journal of Personality and Social Psychology, 46, 1328-1334.

Perkins, K. A. (1984). Heart rate change in type A and type B males as a function of response cost and task difficulty. Psychophysiology, 21, 14-21.

Price, K. P., & Clarke, L. K. (1978). Behavioral and psychological correlates of the coronary-prone personality: New data and unanswered questions. Journal of Psychosomatic Research, 22, 409-417.

Review Panel on Coronary-Prone Behavior and Coronary Heart Disease. (1981). Coronary-prone behavior and coronary heart disease: A critical review. Circulation, 63, 1199-1215.

Rosenman, R. H. (1978). The role of the type A behavior pattern in ischemic heart disease: Modification of its effects by beta-blocking agents. British Journal of Clinical Practice, 1, 58-65.

Ross, R., & Glomset, J. A. (1976). The pathogenesis of atherosclerosis. New England Journal of Medicine, 295, 369-377.

Seligman, M. E. P. (1970). On the generality of the laws of learning. Psychological Review, 77, 406-418.

Scherwitz, C., Berton, K., & Leventhal, H. (1978). Type A behavior, self-involvement, and cardiovascular response. Psychosomatic Medicine, 40, 593-609.

Schwartz, G. E., Shapiro, D., & Tursky, B. (1971). Learned control of cardiovascular integration in man through operant conditioning. Psychosomatic Medicine, 33, 57-62.

Simpson, M. T., Olewine, D. A., Jenkins, C. D., Ramsey, F., H., Zyzanski, S. J., Thomas, G. & Hames, C. G. (1974). Exercise-induced catecholamines and platelet aggregation in the coronary-prone behavior patten. Psychosomatic Medicine, 36, 476-487.

Smyth, K., Call, J., Hansell, S., Sparacino, J., & Strodtbeck, F.L. (1978). Type A behavior pattern and hypertension among inner-city black women. Nursing Research, 27, 30-35.

Steptoe, A., Melville, D. & Ross, A. (1984). Behavioral response demands, cardiovascular reactivity and essential hypertension. Psychosomatic Medicine, 46, 33-48.

Van Egeren, L. F. (1979). Cardiovascular change during social competition in a mixed-motive game. Journal of Personality and Social Psychology, 37, 858-864.

Weidner, G. & Matthews, K. A. (1978). Reported physical symptoms elicited by unpredictable events and the type A coronary-prone behavior pattern. Journal of Personality and Social Psychology, 36, 1212-1220.

Wilder, J. (1956). Basimetric approach (law of initial values) to biological rhythms. Annals of the New York Academy of Science, 67, 123-164.

Wilder, J. (1968). Stimulus and response: The law of initial values. Baltimore: Williams and Wilkens.

Williams, R. B. (1986). Patterns of reactivity and stress. In K. A. Matthews, S. M Weiss, T. M. Dembroski, B. Falkner, S. B. Manuck, & B.W. Redford (Eds.), Handbook of stress, reactivity, & cardiovascular disease (p. 109-126). New York: John Wiley.

Williams, R. B., Haney, T. L., Lee, K. L., Kong, Y. Blumenthal, J. A., & Whalen, R. E. (1980). Type a behavior pattern and coronary atherosclerosis. Circulation, 58, 634-639.

Zyzanski, S. J., Jenkins, C. D. & Ryan, T. J. (1976). Psychological correlates of coronary angiographic findings. Archives of Internal Medicine, 136, 1234-1237.