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THE EFFECTS OF INTER-TARGET-INTERVAL CERTAINTY AND LENGTH ON AUTONOMIC AND CORTICAL REACTIVITY IN TYPE A AND TYPE B MALES

The University of North Carolina at Greensboro PH.D. 1983

THE EFFECTS OF INTERTARGET-INTERVAL CERTAINTY AND LENGTH ON AUTONOMIC AND CORTICAL REACTIVITY IN TYPE A AND TYPE B MALES

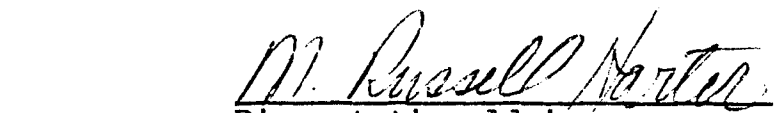
by

Norman Bruce Anderson

A Dissertation Submitted to
the Faculty of the Graduate School at
The University of North Carolina at Greensboro
in Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

Greensboro
1983

Approved by



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8/13/83
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ANDERSON, NORMAN BRUCE, Ph.D. The Effects of Intertarget-Interval Certainty and Length on Autonomic and Cortical Reactivity in Type A and Type B Males. (1983) Directed by Dr. M. Russell Harter. Pp. 139.

The purpose of this study was to examine the psychophysiological effects of waiting and uncertainty in young Type A and Type B males. It was hypothesized that Type subjects would exhibit greater sympathetically medicated cardiovascular changes (as measured by pulse transit time) than Type B subjects on a reaction time task where the intertarget interval (ITI) was relatively long as opposed to short, and when the ITI was uncertain or unpredictable. The prediction on ITI length was based on the notion that Type A subjects have a preference for a more rapid pacing of activities. It was also hypothesized that Type A subjects would evidence greater cortical reactivity when target stimulus occurrence was uncertain or unpredictable. The combination of relatively long and uncertain ITIs was also expected to enhance cardiovascular and cortical responses in Type As relative to Bs.

Ten Type A and 10 Type B subjects performed a reaction time task which involved either a relatively short (6 sec) or long (18 sec) average intertarget interval (ITI) and either with high certainty or low certainty as to the length of the ITI. Physiological measures included tonic and phasic heart rate, tonic and phasic pulse transit time, systolic and diastolic blood pressure, and event-related brain potentials (ERP).

All cardiovascular measures were analyzed using an analysis of covariance with baseline serving as covariate. ERPs were analyzed using an ANOVA. The findings were as follows: (1) Concerning ITI Length, support for the hypothesis was found in tonic heart rate, which was faster in Type As than Type Bs during the 18-sec condition. Type As had shorter phasic PTTs than Type Bs during the 6-sec ITI condition with Bs having shorter phasic PTTs in the 18-sec condition. (2) Concerning ITI Certainty, support for the experimental hypothesis was found on phasic PTT and P295 (P3) ERP latency. Type As had shorter phasic PTTs and P295 ERP latencies than Type Bs during the low certainty condition, while Type Bs had shorter phasic PTTs and P295 latencies under the high certainty condition. (3) Concerning the combined influence of the ITI Length and Certainty, the predicted effect was found only on N201 (N200) ERP latency, which was significantly shorter in Type As than in Type Bs during the 18-sec low certainty condition.

ACKNOWLEDGMENTS

I would like to acknowledge the support and guidance of my adviser, Dr. M. Russell Harter, at every stage of this project, and of my committee members, Drs. Robert Eason, Scott Lawrence, Rosemery Nelson, and Frank Wood for their valuable suggestions. Most of all, I would like to express my gratitude for the encouragement and love given by my parents, Charles and Lois Anderson, who provided the kind of role models that enabled me to stride toward my intellectual potential.

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CHAPTER I

INTRODUCTION

Over the last 30 years, coronary heart disease (CHD) has been the single greatest cause of death in the United States (Stallones, 1980), affecting both men and women, and blacks and whites. Statistics show that the mortality due to CHD reached its peak in the 1950's, at which time about 300 per 100,000 died from the disease. There has more recently been a decline in mortality, such that the death rate from CHD between 1950 and 1980 decreased by about 25%. The optimism induced by this downward trend in mortality must be tempered by the reality that CHD still claims close to a million lives a year and remains America's number-one health problem.

Coronary heart disease denotes a condition whereby there is an inability of the coronary arteries to supply the heart muscle with sufficient oxygen and nourishment to perform all of its functions given the demands made upon it (Friedman & Rosenman, 1974). Actually, CHD is a result of an earlier degenerative process, coronary artery disease or atherosclerosis, which is characterized by a thickening or occluding of the walls of blood vessels supplying the heart. This arterial thickening may be attributed to tiny lesions produced by trauma to a coronary artery as blood is carried up

to the heart. The artery engages in a self-healing procedure by which newly formed cells containing fatty substances or plaques cover the lesion with accompanying blood clots (thrombi), thereby producing arterial thickening (Glass, 1977). Figure 1 shows the progression of coronary occlusion that typically occurs.

This occlusion of the coronary arteries may result in any of several complications. One of the most common and serious complications is known as myocardial infarction or heart attack. Myocardial infarction is basically the necrosis of any part of the heart muscle due to oxygen deprivation over a prolonged time period (Friedman & Rosenman, 1974). This oxygen deprivation in a portion of the heart muscle is caused by the obstruction of blood supply to it by occluded arteries. While other forms of CHD are also prevalent, such as angina pectoris and congestive heart failure, all result from the development of plaques on the arterial wall leading to the obstruction of blood supply to the heart.

Epidemiological and experimental research has delineated several factors that may increase the likelihood that a person who possesses one or more of them will develop CHD. The major risk factors include high levels of serum cholesterol and serum lipoproteins, high systolic and diastolic blood pressure, increasing age, sex (being male), diabetes, and cigarette smoking. Although the above factors have clearly been linked to the incidence of CHD, the combination of them

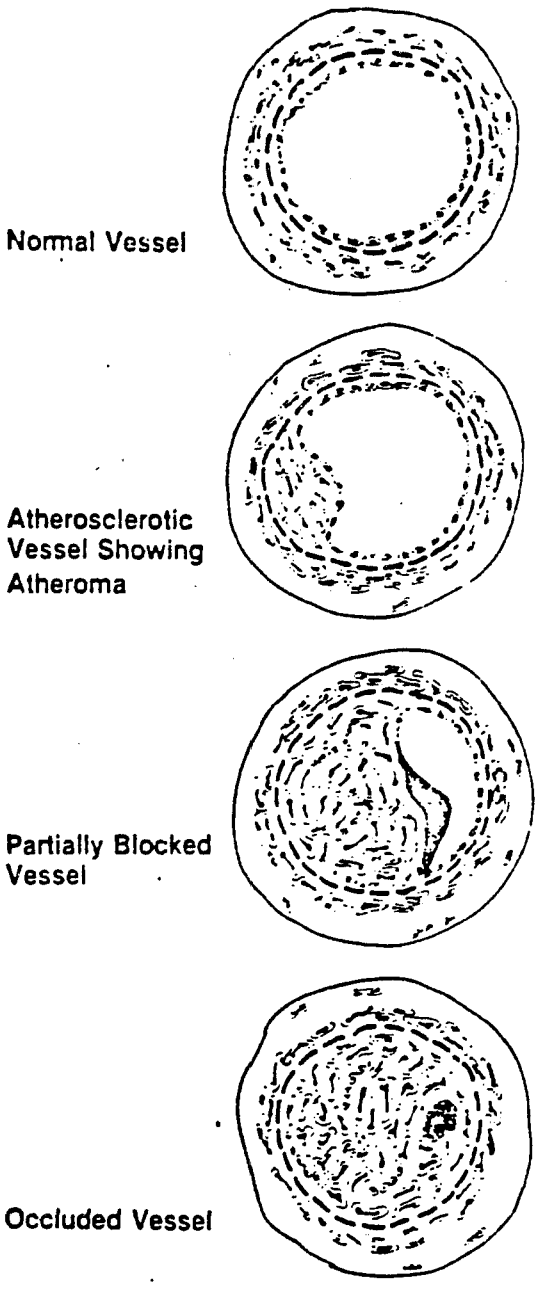

Figure 1. Course of atherosclerotic coronary artery disease, terminating in occlusion of the vessel. Normal vessel (top) and progressive occlusion of the vessel by scarred thickened plaque and thrombus. (From Arteriosclerosis Report of the Working Group on Arteriosclerosis, National Heart, Lung and Blood Institute, 1971.)

of them can account for less than half of the reported cases of heart disease each year (Jenkins, 1971).

As a result of the low explanatory power of the traditional risk factors in accounting for most of the new cases of CHD, researchers have begun to search for other risk factors. It has long been assumed that a person's life style or behavior could affect the well-being of his or her cardiovascular system (e.g., Osler, 1897). While there existed a casual acknowledgement of the possible role behavior might play in the development of CHD, until recently there was little empirical support for this notion. In the 1950's, however, two cardiologists, Meyer Friedman and Ray Rosenman (1959), observed consistent characteristic behavioral features, such as time consciousness and competitiveness, of many heart disease patients. These behavioral features, they speculated, might predispose patients to heart disease. To gain some idea of the thinking of lay people and medical professionals on this issue, Friedman and Rosenman sent out several hundred questionnaires to San Francisco business executives and internists, asking them to check which one of several items on a list they thought preceded a heart attack in a friend or patient. The vast majority in both groups believed that indulgence in "excessive competitive drive and meeting deadlines" was the primary precipitator (Friedman & Rosenman, 1974). The responses were provocative given the

current thought on the importance of cigarette smoking, cholesterol, and lack of exercise in bringing about a heart attack.

Subsequent to these early observations, several features have been added to the complex of behavior that is characteristic of many people who suffer from CHD (see Jenkins, 1975). This behavioral complex is known as the Type A or coronary-prone behavior pattern. The following is a discussion of the essential characteristics of the Type A behavior pattern and its assessment.

Characteristics of the Type A Behavior Pattern

In general, individuals manifesting the Type A behavior pattern are characterized by excessive time urgency, hard-driving competitive behavior, aggressiveness, hostility, a persistent desire for recognition and advancement, all of which are readily evoked by a variety of stimuli in the social and physical environment (Matthews, 1982; Rosenman & Friedman, 1974). The following is a discussion of some of the behavioral features of the Type A behavior pattern. It should be pointed out, however, that the following discussion of the characteristics of the Type A behavior pattern will be a descriptive and general one, concentrating on aspects of the behavior pattern which are reflected by the most commonly used Type A assessment procedures.

Motor Behavior

Persons evidencing the Type A behavior pattern often exhibit a rapid pacing of their motor functioning. For example, they tend to eat very rapidly, walk fast, and show rhythmic or repetitive movements when sitting or standing at rest, such as tapping their feet or fingers and fidgeting. Type As may use exaggerated gestures during normal conversations, sometimes pounding on a surface for emphasis. Their handshakes are usually quite firm and vigorous.

Verbal Behavior

One of the most striking characteristics of many Type A individuals is their speech topography. As they talk, many of their words have an "explosive" quality (Scherwitz, Berton, & Leventhal, 1977). That is, some words are said much louder than others and tend to be the first words in sentences or key words (Schucker & Jacobs, 1977). Similarly, the last words in a sentence are spoken more rapidly than the first. When interrupted, Type As may "talk-over" the speech of the other person in order to finish a thought, and frequently interrupt or otherwise attempt to hurry the speech of another by using anticipatory nods and "ahems" or "right, right" while listening (Jenkins, 1975). When they are asked a question, there is typically a very short latency between the end of the question and the onset of their response. Relatedly, when a questioner appears to be having difficulty

completing an obvious question (i.e., appears "stuck"), Type As without much hesitation will provide the right words.

Professional and Social Life

Type A individuals tend to be very achievement- and work-oriented. They put great value on recognition and power and draw much of their self-worth from their productivity. They have often been described as "possessing an intense drive toward self-selected but poorly defined goals" (Suinn, 1977). Because they are work-oriented, Type A persons have difficulty "finding the time" to relax, and, when they do find time, will spend much of it thinking about professional matters, or simply cut short the relaxation period to get back to doing "something useful" (Jenkins, 1975). The professional life of the Type A person is closely allied to the issue of "time urgency". Type As seem constantly to be under pressure to do things fast. They loathe waiting, whether at a traffic light, in the doctor's office, or in a grocery store checkout line, and become visibly irritated when made to do so. Professionally, they tend to constantly be under deadline pressure. This deadline pressure usually results from the setting of self-imposed and unrealistic deadlines for activities that require more time, and the scheduling of more and more activities in less and less time with fewer allowances made for unforeseen contingencies (Friedman & Rosenman, 1974). It is therefore not unusual

for a Type A individual to attempt to carry out several activities simultaneously (e.g., dictating letters while driving), and to prefer to read only summaries or abstracts of articles (Suinn, 1977). Type As variously describe themselves as being more aggressive, angry, achievement-oriented, shrewd, active, quick, dominant, sociable, lacking in self-control, and hard-working than Type Bs report (Caffrey, 1968; Chesney, Black, Chadwick & Rosenman, 1981; Matthews, 1982). Some studies have found that although Type As state that they are more self-confident and have achieved high-status occupations, they also report having symptoms that are indicative of stress and being dissatisfied with work, life achievements, and marriage (Howard, Cunningham, & Recknitzer, 1976, 1977; Waldron, 1978).

Another hallmark of the Type A behavior pattern is competitiveness. Type As seem to enjoy competitive situations -- especially when they win. This competitive quality is not restricted to the more obvious forms of competition such as, say, racquetball, but can also be evidenced professionally (Waldron, 1978). For example, a Type A person might boast about his/her company's profits over the year as compared to those of other companies, or the number of publications or amount of grant money accumulated by their laboratory as compared to others. Most Type As loathe losing,

and if it is apparent that they will lose, many become apathetic and simply give up.

The antithesis of Type A is the Type B behavior pattern. The simplest way to describe the Type B pattern is to say that it is characterized by a relative absence of the Type A features (Dembroski, MacDougall, & Shields, 1977). Essentially, if Type As are viewed as lying on the extremes of several behavioral continua (e.g., competitiveness, impatience, etc.), then Type Bs may be viewed as lying behaviorally on the opposite extremes. This is not to suggest that Type B individuals are not <u>at times</u> competitive or impatient, but the number of environmental stimuli which evoke these attributes are fewer and do so to a lesser degree than in Type As.

This latter point is important and deserves some elaboration. Neither the Type A nor Type B behavior pattern is viewed as a "trait". Rather, both are viewed as learned behavioral responses to specific environmental conditions. For example, research has demonstrated that only under sufficiently challenging conditions will people labeled as Type A display any of the behaviors that uniquely identify them (Dembroski et al., 1977; Goldband, 1980; Manuck & Garland, 1979). Suinn (1977) has presented a model describing how Type A behavior might be acquired and maintained. In

stressful or challenging situations, such as writing a paper, a person may impose a stringent deadline on himself (Type A behavior) for completion of the paper. Setting this deadline may result in the reinforcing consequences of completion of the paper and therefore stress reduction (see Figure 2).

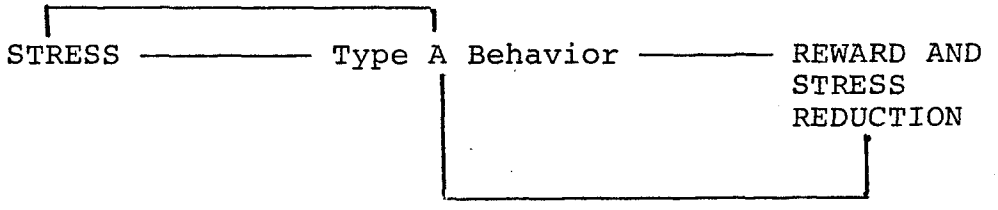

Figure 2. The cycle of stress. (From Suinn, 1977.)

Because of these favorable consequences, this person might in the future impose stringent deadlines on other work activities. Having to meet these deadlines, however, is in itself stress-producing, and may lead to other Type A behaviors such as longer working hours and reduced relaxation time. These activities in turn help the person accomplish his/her goals and are thus strengthened. In other words, Type A individuals are often caught in a vicious cycle of stress-involving environmental and behavioral interactions. While many individuals may exhibit Type A behaviors in a generalized fashion across a variety of situations, the behavior pattern is still viewed as learned and malleable by environmental factors.

Assessment of the Type A Behavior Pattern

The most commonly used devices for assessing Type A behavior are the Structured Interview (SI) and the Jenkins Activity Survey (JAS). The SI was developed by Rosenman, Friedman and associates (Rosenman, Friedman, Straus, Wurm, Kositchek, Hahn, & Werthessen, 1964) and consists of 25 questions dealing with the intensity of ambitions, competitiveness, sense of time urgency, and the nature and magnitude of hostile feelings. All interviews are tape-recorded and later reviewed independently by a trained auditor. The interview assessment technique takes into account not only the specific answers to questions but also the behavioral style of the subjects in making classifications. For example, some questions are designed to yield fairly direct diagnostic information, such as "When you play games with people your own age, do you play for the fun of it, or are you really in there to win?" Or "When you are in your automobile, and there is a car in your lane going far too slowly for you, what would you do about it? Would you mutter and complain to yourself? Would anyone riding with you know that you were annoyed?" Other questions, however, are designed to assess Type A characteristics more indirectly. For instance, on some questions the interviewer pretends to get "hung up" on a word; that is, appears not to be able to think

of the appropriate word to use in a sentence. Type A subjects are much more likely to provide an appropriate word than Type Bs. Also rated are voice stylistics such as accentuations and inflections, and number of "ahems" used to hurry the interview along.

Using the structured interview, it is possible to rate behavior patterns on a 5-point scale: fully developed As (A_1); incompletely developed As (A_2) (absence of explosive speech patterns); incompletely developed Bs (B_3); fully developed Bs (B_4); and Type X, possessing characteristics of both A_2 and B_3 Types.

The second most frequently used Type A assessment device is the Jenkins Activity Survey for Health Prediction (JAS), a self-administered questionnaire. There are special versions of the JAS, one for working adults (Form B) and one for college students (Form T). Typical JAS questions are:

1) "Has your spouse or friend ever told you that you eat too fast?" A Type A response is "Yes, often," and Type B responses are "Yes, once or twice," or "No, no one has told me this."

2) How would your spouse (or closest friend) rate you?" Pattern A responses are, "Definitely hard-driving and competitive," and "Probably hard-driving and competitive," and B responses are "Probably relaxed and easy-going" and "Definitely relaxed and easy-going."

3) "Do you ever set deadlines or quotas for yourself at work or at home?" An A response is "Yes, once per week or more often," and B responses are "No" and "Yes, but only occasionally."

In using the JAS, subjects can be assigned a score along

a continuous Type A-B dimension. Typically, subjects who score in the upper half are classified as Type A and, in the lower half, as Type B. In addition, the JAS can also yield scores on independent subscales of "Hard Driving", "Speed and Impatience", "Job Involvement".

Both assessment instruments are of demonstrated reliability and validity. One-year test-retest reliability for the SI is .80 and between .65-.75 for the JAS (Dembroski, Caffrey, Jenkins, Rosenman, Spielberger, & Tasto, 1978; Jenkins, Rosenman, Friedman, 1968). Inter-rater reliability on the SI tape recordings has been found to be between .75 and .84 (Caffrey, 1968; Jenkins, Rosenman, & Friedman, 1967). Both instruments also appear to have some concurrent and construct validity. Research has shown the SI and JAS to be significantly correlated with measures of aggression, speed, and activity level (Dembroski et al., 1978; Glass, 1977), demonstrating to a certain degree their concurrent validity. The construct validity of the instruments has been shown in a variety of ways. When compared to Type Bs, Type A subjects 1) show a greater tendency to develop coronary heart disease; 2) show greater autonomic arousal in stressful conditions; 3) are more concerned with maintaining control over their environment; and 4) show more psychomotor activity in response to environmental challenges. In addition, there tends to be a low correlation between the two measures of Type A

behavior and more traditional measures of personality (Glass, 1977).

Type A Behavior and the Atherosclerotic Process

While epidemiological research has clearly documented the association between Type A behavior and CHD (see review of this research in a later section), it is not discernible from these studies how Type A behavior might cause CHD. That is, it is not clear how behavioral characteristics such as competitiveness, impatience, aggressiveness, and achievement-striving translate into occluded coronary arteries (just as it is unclear how smoking, hypertension, and other risk factors lead to CHD). Currently, however, there is strong support for the hypothesis that Type A behavior translates into CHD through the chronic activation of sympathetically mediated autonomic nervous system arousal and its associated cardiovascular and neuroendocrine phenomena (Williams, Friedman, Glass, Herd, & Schneiderman, 1978). On a molar physiological level, sympathetic autonomic arousal is best exemplified by the fight-or-flight concept, which states that humans and animals respond physiologically to stressful situations in predictable ways. The body prepares itself physiologically to escape from the stressor (flight) or to confront or fight the stressful stimulus. This physiological preparation is characterized by increases in heart rate, blood pressure,

respiratory rate, and blood flow to peripheral muscles. This pattern of responding is viewed as quite adaptive in lower animals and in more primitive cultures, where stressful stimuli are often of a life-threatening nature and rapid mobilization of bodily resources is necessary. The "stressors" facing modern man are predominantly not of the life-threatening nature, but involve more subtle psychosocial stimuli. Yet, for many people, especially those labeled Type A, the fight-flight response is initiated by these psychosocial stimuli when it need not be.

To understand the importance of frequent sympathetic autonomic arousal in the development of CHD, it is essential to understand certain biochemical and hormonal consequences of sympathetic activity. Specifically, it is the secretion of sympathetic catecholamines which is implicated as the primary pathogenic mechanism. A brief review of the relationship between the catecholamines and CHD follows.

Catecholamines and CHD

Activation of the sympathetic branch of the autonomic nervous system (see Table 1) leads to the discharge of the two primary catecholamines, epinephrine and norepinephrine (sometimes called adrenaline and noradrenaline, respectively) from sympathetic terminals and from the adrenal medulla (Steptoe, 1981). Pharmacological research has demonstrated

Table 1

Action of the Autonomic Nervous System

Structure	Function	Parasympathetic Nervous System	Sympathetic Nervous System
Eyes Iris	Constriction	+	-
Eyes Lens	Accommodation	+	-
Lacrymal glands	Tears	+	-(?)
Nasal mucosa	Secretion, dilation	+	-
Salivary glands	Salivation	+	-(?)
Gastrointestinal tract	Peristalsis	+	-
Stomach glands	HCL, pepsin, & mucus	+	0
Pancreas (islet cells)	Insulin	+	0
Heart (rate)	Acceleration	-	+
Lungs (bronchia)	Dilation	-	+
Adrenal medulla	Epinephrine	0	+
Sympathetic Terminals	Norepinephrine	0	+
Peripheral blood vessels	Vasoconstriction	?	+
Sweat glands	Sweating	0	+
Pilomotor cells	Piloerection	0	+
Internal sphincters Bladder Intestine	Contraction	-	+
Bladder wall Lower bowel	Contraction	+	-
Genitalia	Erection	+	-
Genitalia	Ejaculation	-	+

Note: In the table (+) indicates a facilitative effect and (-) an inhibitory effect. Note that the upper portion of the table emphasizes facilitative effects of the cranial parasympathetics, the bottom separates the sacral parasympathetic effects, and the central portion emphasizes sympathetic facilitative effects. (Adapted from Stern et al., 1980)

that these hormones may act as neurotransmitters at either of the two groups of sympathetic nervous system receptors, alpha and beta receptors. Stimulation of the alpha-sympathetic receptors promotes more peripheral vasoconstriction and blood pressure increase, while beta-receptor stimulation increases heart rate and force of cardiac contraction (Steptoe, 1981). There is now extensive evidence implicating the catecholamines as a major element at each stage of the atherosclerotic process.

First of all, catecholamines have been shown to initiate the atherosclerotic process via the development of myocardial lesions (Hueper, 1944; Raab, Stark, MacMillian et al., 1961; Waters & de Suto-Nagy, 1950). In animals, the severity and extent of these lesions varies directly with the amount and rate of catecholamines infused (Haft, 1974) and damage may occur in all chambers of the heart (Schenk & Moss, 1966).

Another way by which the catecholamines lead to cardiovascular pathology is through the facilitation of platelet aggregation. Platelets are disk-shaped blood elements which average about 250,000 per cubic millimeter of blood (Miller & Keane, 1978). The aggregation, or clumping together, of these platelets represents an early stage in blood clotting (sometimes called coagulation or thrombosis), which might impede circulation (see again Figure 1). Research has shown that epinephrine and norepinephrine facilitate platelet

aggregation and blood clotting (Ardlie, Glen, & Schwartz, 1966; Haft, 1974; Haft, Kranz, Albert et al., 1976; O'Brien, 1963).

Catecholamines might also affect pathogenic cardiovascular processes indirectly through the production of very low-density lipoproteins (VLDL), a form of cholesterol which is known to damage the arterial wall and contribute to the process of occlusion. Basically, three stages are involved here. In the first stage, secretion of the catecholamines facilitates the release of free fatty acids (FFA) from triglycerides, which are used by skeletal muscles and myocardium (heart muscle) in the production of energy (Herd, 1978; Ziesler, Maseii, Klassen, Rabinowitz, & Burgess, 1968). The amount of FFAs used is dependent on the energy demands of the body. For example, more FFAs are used during exercise and in cold environments than when an organism is at rest and warm (Ziesler et al., 1968). The second stage involves liver uptake of unused FFAs. At the liver, these FFAs are then used to produce VLDL which are secreted into plasma (Stage 3). Once in the blood stream, the lipoproteins may act to damage coronary arteries by the production of lesions (Selye & Bajusz, 1959) or, following lesion development, may accumulate over the lesion to cause arterial thickening.

Therefore, it is clear that activity of the sympathetic

nervous system, and the concomitant release of catecholamines, contributes significantly to the pathogenesis of of CHD. To summarize, empirical evidence has determined that sympathetic catecholamines may (1) initiate arterial lesions; (2) facilitate platelet aggregation and thrombosis; and (3) facilitate the production of very low-density lipoproteins. It is also clear that the degree and severity of these phenomena are related directly to the amount of catecholamine secretion -- the more catecholamines released, the more marked these phenomena.

Before discussing how Type A behavior relates to the catecholamines, it is important to state that sympathetic nervous system activity is not always pathological. For example, physical exercise and exertion lead to sympathetically mediated cardiovascular changes (Dimsdale & Moss, 1980; Frankenhaeuser, 1971)that are not considered hazardous to one's health. In fact, health is probably promoted by this type of activity. The primary difference between sympathetic activity elicited during exercise and nonphysical activity is that, when one exercises, the body's energy demands increase, and the catecholamines and FFAs are extracted from plasma and utilized as energy (Ziesler et al., 1968). Conversely, when one is late for a meeting and gets caught in a traffic jam, or is preparing to give a talk in front of a

large and critical audience, similar hormonal increases occur, but without the high rates of extraction and utilization (Herd, 1978). In other words, the catecholamines continue circulating, increasing the probability of arterial lesions, platelet aggregation, and production of VLDL. Therefore, "pathogenic" sympathetic arousal is due to the mobilization of energy substances without their subsequent utilization.

It may be concluded, therefore, that if environmental situations frequently and chronically elicit sympathetic hyperactivity in the absence of physical demands, the probability of CHD development increases. Additionally, experiments using both invasive and noninvasive measures of sympathetic arousal suggest that the greater the sympathetic activity, the greater the probability of serious arterial damage (Haft, 1974; Manuck & Kaplan, Note 2). For example, Manuck and Kaplan (Note 2) recently demonstrated that monkeys who exhibited heart rate hyperresponsivity under repeated exposure to stressful conditions developed significantly greater coronary atherosclerosis in their primary arteries, relative to monkeys low in responsivity.

The results of the research demonstrating atherosclerosis development through sympathetic nervous system activity provide the framework for the hypothesis that the Type A behavior pattern leads to CHD via the chronic elicitation of sympa-

thetic hyperreactivity (Williams et al., 1978). If this hypothesis is correct, then Type A individuals might be expected to exhibit significantly greater sympathetic reactivity to environmental challenges relative to Type B persons. There are now a number of studies demonstrating this greater reactivity in Type As. On a biochemical level, Type As have been shown to have significantly higher levels of serum norepinephrine than Type Bs during their working hours and when exposed to experimental challenges such as insoluble mental puzzles (Friedman, Byers, Diament, & Rosenman, 1975; Friedman, St. George, & Byers, 1960; Williams et al., 1978). Most studies, however, have used noninvasive psychophysiological procedures to detect sympathetic nervous system arousal. These studies will be reviewed shortly.

It may be concluded that there is strong evidence supporting the notion that excessive sympathetic activity is a pathogenic mechanism for CHD development, and that individuals labeled Type A often exhibit heightened levels of sympathetic activity under environmental challenges. The chain of events illustrating how environmental factors operate through sympathetic hyperreactivity which leads ultimately to CHD in persons labeled Type A is shown in Figure 3.

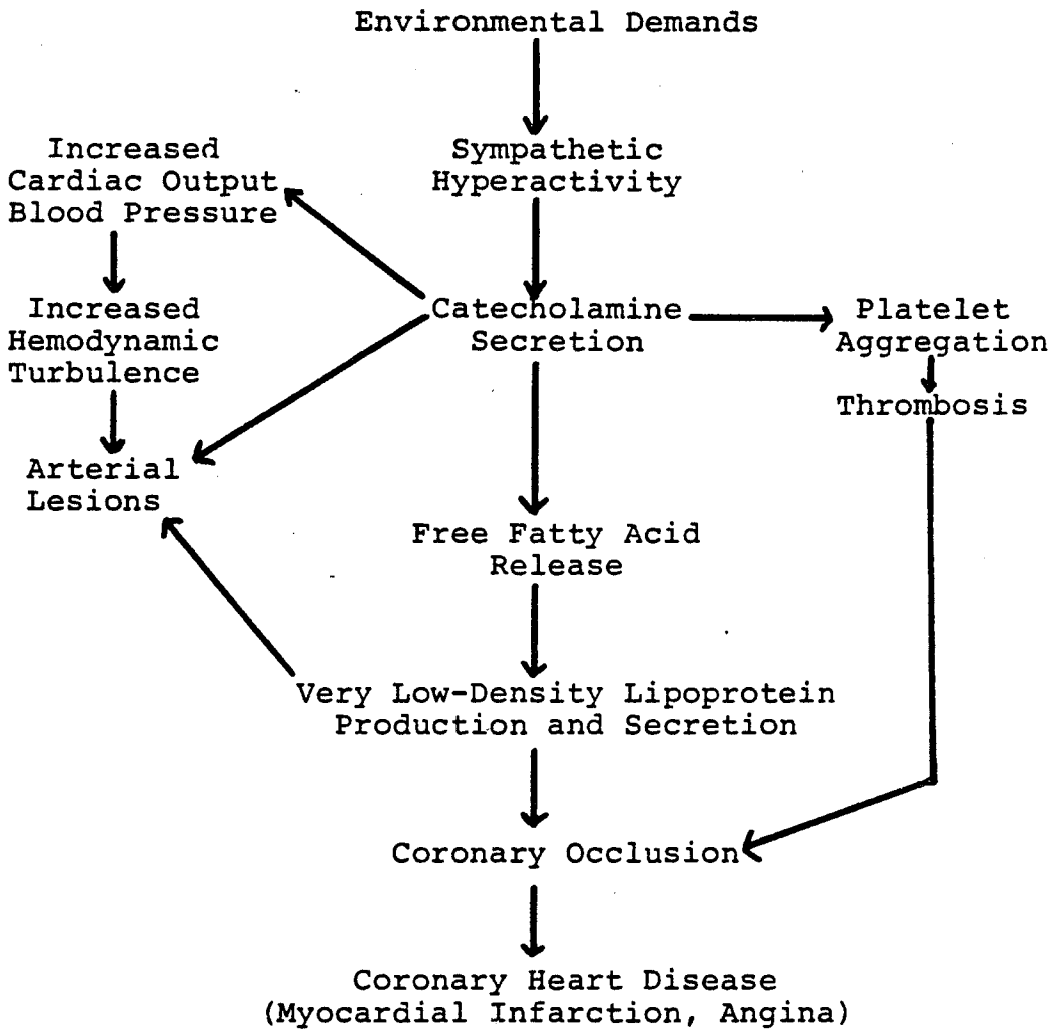

Figure 3. Processes by which environmental demands act to increase the probability of coronary heart disease in persons exhibiting the Type A behavior.

Association of Type A Behavior and Coronary Heart Disease

Epidemiological Research

The association between the Type A behavior pattern and CHD has now been demonstrated in both retrospective and prospective epidemiological studies. In the initial retrospective studies (Friedman & Rosenman, 1959; Rosenman & Friedman, 1961) it was discovered that Type A men and women have roughly four to seven times the rate of CHD of their Type B counterparts. For example, Friedman & Rosenman (1959) studied 83 Type A men, 83 Type B men, and 46 unemployed blind men selected because they manifested a chronic state of insecurity and anxiety. The Type A and B groups were selected from various corporations in California. Upon physical examination, it was found that Type A men evidenced seven times the frequency of clinical coronary artery disease (i.e., heart attacks) and significantly higher serum cholesterol levels than the Type B and blind men. Importantly, detailed analysis of the data suggested that the higher incidence of artery disease could not be attributed to differences in age, exercise, calorie or fat intake, alcohol, or cigarettes. Similar results were obtained with a female population (Rosenman & Friedman, 1961).

These initial studies were retrospective in nature -- that is, they were conducted using subjects who already evi-

denced some artery disease. Therefore, to ascertain the role of Type A behavior in predicting future occurrence of the disease, the Western Collaborative Group Study was started in 1960 as a prospective epidemiological project. The study involved 3,524 men, aged 39 to 59 years, who were employed in 11 participating corporations in northern and southern California. All were given thorough medical examinations which assessed subject's lipids and lipoproteins (i.e., fatty material), blood pressure and coagulation. In addition, each subject was given an electrocardiogram and other heart disease assessments. Additionally, extensive data were collected concerning their family medical history and smoking, diet, and exercise habits. During the screening examinations 113 of the 3,524 men were found to already have CHD. Of this group of 113, 80 (70.9%) were judged, using blind raters, to be Type A (Rosenman et al., 1964). The remaining, disease-free subjects (3,154) were followed subsequently for 8½ years. Upon re-examination, it was found that 257 had developed CHD (as evidenced by occurrence of heart attack and angina pectoris); of those, 178 (or roughly 70%) were classified as Type A; only 79 (or 30%) of the 257 heart disease cases were judged to be Type B. Thus, Type A subjects experienced over twice the incidence of CHD as the Type B subjects. Importantly, even after statistical adjustment

procedures which controlled for differences in traditional risk factors such as age, smoking, cholesterol, and systolic blood pressure, the Type A subjects were still 1.97 times, or doubly, at risk (Rosenman, Brand, Jenkins, Friedman, Straus, & Wurm, 1975).

Although the Structured Interview (SI) was used initially to classify subjects as Type A vs. Type B, the Jenkins Activity Survey (JAS) was later given to 92% of the original sample and was also shown to be useful in determining which subjects would subsequently develop CHD (Jenkins, Rosenman, & Zyzanski, 1974). Further, JAS scores are also predictive of recurrent heart attacks (Jenkins, Rosenman, & Zyzanski, 1974; Jenkins, Zyzanski, Rosenman, & Cleveland, 1971).

Finally, epidemiological research has further established the association of Type A behavior and CHD in different regions of the United States (Kenigsberg, Zyzanski, Jenkins, Wardwell, & Licciardello, 1974; Shekelle, Schoenberger, & Stamler, 1976) and in several European countries (Heller, 1979; Zyzanski, Wryesniewski, & Jenkins, 1979). Therefore, it now appears that the Type A behavior pattern represents a risk factor of the development of CHD "over and above those imposed by age, systolic blood pressure, serum cholesterol and smoking and appears to be of the same magnitude as the relative risks associated with any of these other factors" (Panel on Coronary Prone Behavior, 1978).

Arteriographic Evidence

Several reports have been published demonstrating the relationship between Type A behavior and atherosclerosis (arterial occlusion) as determined by arteriographic x-rays or angiograms. Blumenthal, Williams, Kong et al. (1975), at Duke University Medical Center, studied 142 patients undergoing coronary angiography and found Type A behavior as measured by the SI to be systematically related to the degree of arterial thickening. Under blind classification procedures, 44% of those patients evidencing mild atherosclerosis (see Figure 1, second picture) were judged to be Type A; of those patients with moderate atherosclerosis (Figure 1, third picture), 69% were Type A; and finally, of those patients with severe arterial occlusion (Figure 1, fourth picture), 95% were judged to be Type A. These same researchers obtained a comparable result when a larger sample consisting of a sizable proportion of female subjects was used. Type A female patients were as likely to have developed coronary atherosclerosis as the males (Blumenthal, Williams, Kong, et al., 1978). The findings of Blumenthal and associates have been replicated by researchers at Boston University School of Medicine (Zyzanski, Jenkins, Ryan, Flessas, Everist, 1976) and at Columbia University (Frank, Heller, Kornfeld, Sporn, & Weiss, 1978), and are consistent with autopsy data (Friedman, Rosenman, Straus, Wurm, & Kostichek, 1968).

In summary, the Type A behavior pattern has been shown to predict the occurrence of myocardial infarction, recurring myocardial infarction, and angina pectoris. Additionally, it has been associated with the degree of angiographically documented coronary atherosclerosis. What follows is a discussion of the psychophysiological literature on Type A behavior and autonomic reactivity.

Type A Behavior and Autonomic Reactivity

Cognitive Tasks

Manuck, Craft, and Gold (1978) conducted one of the initial investigations testing the hypothesis that the Type A behavior pattern represents a risk factor affecting the cardiovascular system via sympathetically mediated autonomic nervous system activity. These investigators studied the effects of a difficult cognitive task on three measures of cardiovascular response -- heart rate, systolic blood pressure, and diastolic blood pressure. The task involved presenting A and B subjects with cards containing four related objects (from Feldman & Drasgnio, 1959, Visual-Verbal Test). The objects differed along the dimensions of size, form, color, etc., but contained two dimensions that were common to three of the stimulus designs. The subject's task was to identify the stimulus objects on each card that belonged together, on the basis of these two salient attributes.

Subjects were given 6 seconds to respond, even though intervals of up to 3 minutes are typically allowed for responding to items on this test. Obviously, this time constraint significantly increased the difficulty of the task. It was found that Type A males evidenced significantly greater increases in systolic blood pressure than Type B males under these conditions. No differences were noted between groups in diastolic blood pressure or heart rate, nor between A and B females on any of the measures.

Manuck and Garland (1979) expanded on the previous study by adding a monetary incentive to the cognitive task. All subjects, Type A and Type B, received the cognitive task manipulation described previously. Half of each group, however, was paid 10¢ for every correct response up to a balance of either 80¢ or $1.60, depending on the condition. When a subject's balance reached either of these amounts, he/she received 15¢ and 20¢ respectively for each correct response. Throughout this condition, subjects lost 10¢ for each incorrect response, regardless of balance. Analyses revealed that Type A subjects showed significantly greater elevations in systolic blood pressure and pulse pressure than Type Bs, but the data showed no reliable interaction of the Type and Incentive factors. Also, no significant increases in heart rate or diastolic blood pressure were evident.

Another cognitive manipulation often used involves having Type A and Type B subjects attempt to answer difficult questions from an American history quiz (Dembroski, MacDougall, & Lushene, 1977; MacDougall, Dembroski, & Krantz, 1981). Subjects are told that they will be asked questions about "well-known facts in early American history." Most of the questions, except the first one, were in fact extremely difficult, and the probability of answering more than one or two correctly was small. MacDougall et al. (1981), studying Type A and Type B women, found significant systolic blood pressure increases for both groups during this challenging history quiz; however, the Type A women responded with somewhat greater increments in blood pressure than their Type B counterparts.

A recent study by Williams, Lane, White, et al. (Note 1) utilized a mental arithmetic task to study the Type A - cardiovascular relationship. Thirteen undergraduate males engaged in difficult arithmetic tasks requiring them to serially subtract 13 from a four-digit number. They were asked to subtract as fast as possible and informed that their answers would be recorded and that the best performer would receive a small prize. Measures of heart rate (HR), systolic and diastolic blood pressure (SBP, DBP), forearm blood flow (FBF), and forearm vascular resistance (FVR) were

taken during a 20-minute baseline period, during the 20-minute task period, and again during a 20-minute recovery period. The authors discovered that HR, SBP, DBP, and FBF all increased significantly from baseline during the arithmetic task. FVR decreased from baseline but not significantly. A high correlation was found between Type A behavior (as assessed via Jenkins Activity Survey and Structured Interview) and physiological response magnitude. Higher Type A scores on the Jenkins Activity Survey (JAS) were related to the greater increases in HR and FBF. In addition, there were significant differences between Type A and Type B subjects, identified via Structured Interview, on measures on FBF and SBP.

The study of Williams et al. (Note 1) is important because it was one of the first studies to find significant HR increases during a cognitive task with Type A subjects. One possible reason for this latter finding is that, in previous research, the cognitive task was not sufficiently challenging for the Type A subjects. The basis for this assumption lies in current research suggesting that the physiological effects of Type A behavior are evidenced only under sufficiently challenging or "relevant" conditions (Goldband, 1980). In the Williams et al. study, when subjects were told that the best performer would receive a prize, a direct competitive

situation was established. Since competition has previously been shown to be one of the central components of the Type A pattern (Dembroski, MacDougall, Shields, Petitto, & Lushene, 1978), increases in heart rate when subjects are competing might be expected. Another possible explanation is that the cognitive tasks used in previous studies might be characterized as "sensory intake" tasks, while Williams et al. (1981) utilized a "sensory rejection" task. The sensory intake-sensory rejection distinction is the result of theorizing by the Laceys (1974), and research by Williams, Bittker, Buchsbaum, and Wynne (1975). Sensory intake tasks are those requiring the detection of environmental stimuli in order to make the correct response (e.g., choice reaction time tasks), while sensory rejection requires no such attention to incoming stimuli (e.g. mental arithmetic tasks). These tasks have been found to elicit different patterns of autonomic response, with heart rate decreases typically occurring during sensory intake tasks, while sensory rejection tasks elicit heart rate increases. Recently, Williams, Lane, Kuhn, et al. (1982) found that Type A males showed significantly greater increases in forearm blood flow, epinephrine, norepinephrine, and cortisol, along with greater decreases in forearm vascular resistance, than Type B males on a sensory intake task (mental arithmetic), while no differences were discovered on the

sensory rejection task (reaction time). Therefore, the differences in cardiovascular and neuroendocrine reactivity in Type As and Type Bs on cognitive tasks appear to be dependent upon both the level of challenge and the nature of the task (i.e., intake vs. rejection).

A cognitive task involving elements of both sensory intake and sensory rejection was used recently in a study by Blumenthal, Lane, Williams, et al. (1983). The task was the Word Finding Test (WFT) which involves presentation of a series of sentences in which one word is missing. Each sentence provides a clue as to what the missing word is. A nonsense word is usually used in place of the correct one. For example, a series of items may be as follows: (a) A Grobnick really isn't worth very much; (b) However, Grobnicks are still more than a dime a dozen; (c) Many people won't bother to pick up a Grobnick if they find one; (d) Grobnicks are often given to little children who sometimes swallow them; (e) You can still get weighed for a Grobnick. The Grobnick is correctly identified as a penny (Blumental et al., 1983). Type A and B subjects performed this task either with or without the addition of a monetary incentive, whereby subjects could win up to five dollars. The amount a subject won was dependent on the number of clues needed to answer correctly. Subjects received 25¢ for a correct response to the first

clue; 10¢ for a correct response to the second clue, etc. Type A subjects were found to show significant increases in heart rate, systolic blood pressure, and forearm blood flow, and decreases in forearm vascular resistance across both incentive and nonincentive conditions. Type B subjects, in contrast, showed increases in heart rate and systolic blood pressure only under the incentive condition. Therefore, it appears that with the WFT, Type A subjects experienced enhanced physiological arousal in the absence of explicit performance demand, suggesting a possible difference in attributional characteristics of Type As and Type Bs. That is, Type A persons may more readily attribute to or perceive a challenge in a situation than Type B persons, leading to augmented physiological reactivity in Type As (Schlegel, Wellwood, Copps, Gruchow, & Shurratt, 1980; Smith & Anderson, Note 3).

Psychomotor - Physical Performance Tasks

Type A subjects, when exposed to tasks involving motor performance, tend to react with greater sympathetic arousal than their Type B counterparts (e.g., Dembroski, MacDougall, Herd, & Shields, 1979; Dembroski, MacDougall, & Shields, 1977; Goldband, 1980; Lovallo & Pichkin, 1980; MacDougall, Dembroski, & Krantz, 1981). Several of these studies will now be reviewed.

Dembroski et al. (1979) designed an experiment to determine whether Type A male subjects were more likely than Type B subjects to perceive a performance challenge on a given task, regardless of the nature of that task. It was assumed that if Type As always perceived a performance challenge on a task, they should evidence heart rate (HR) and blood pressure (BP) increases even when no direct challenge was made. To test this hypothesis, those cardiovascular measures were taken on A and B subjects while they were given a standard cold pressor test. Subjects were presented with either high-challenge instructions ("This is a test of will power and endurance. Try to keep your hand immersed in the water as long as you can") or low-challenge instructions ("Remove your hand when it gets too cold"). The results showed that under high-challenge instructions Type A subjects responded to the cold stress with significantly greater increases in HR and SBP than Type B subjects. Under low-challenge instructions, however, there were no differences between As and Bs in HR or BP. The authors concluded that "since the blood pressure difference between Type A and Type B subjects was paralleled by a significant difference in HR change during the high-challenge condition, these data are indicative of greater sympathetic nervous system arousal in Type A subjects" (Dembroski et al., 1979). The findings from the cold pressor test were partially replicated in the same study using a reaction time

task. Under high-challenge instructions, Type As again showed significantly greater increases in SBP than Type Bs.

A similar study was conducted by MacDougall et al. (1981), comparing the physiologic responses of Type A and Type B women. Subjects were presented with cold pressor and reaction time tasks that emphasized only the challenging nature of the tasks. The authors discovered that, unlike males in the Dembroski et al. study, Type A women in the present experiment did not prove to be more physiologically reactive than their Type B counterparts. One explanation posited by the authors was that these particular tasks were not perceived by the women as being highly relevant to their personal definition of success and achievement, and thus did not provide a sufficiently challenging situation for them. This reasoning was somewhat supported in a second experiment when Type A and Type B women were placed in a situation involving interpersonal and verbal challenge from another woman during a difficult U.S. history quiz. As reported earlier in the section on cognitive tasks, Type A women evidenced significantly greater increases in SBP under those conditions than did Type B women.

One of the most important studies on Type A and physiologic responses to date was conducted by Goldband (1980). In the first of two experiments, Type A and Type B subjects performed a reaction time task which either included manipu-

lations involving competition, time urgency, and loss of control (task-relevant condition) or which did not include those manipulations (neutral condition). Competition was manipulated in the relevant condition by informing the subjects that their performance would be compared with that of the "average subject" and that it was crucial for each subject to do his very best. In the neutral condition, subjects were told that the purpose of the experiment was to study some basic physiological processes, and that the reaction time task was merely a convenient way to keep the subject busy while measures were being taken. Time urgency was heightened in the relevant condition by introducing a deadline in the reaction time task. That is, subjects were given a preset criterion as to how fast they should respond on each trial. This criterion was set in such a way that, it was assumed, subjects would miss the deadline on approximately half of the trials. In the neutral condition, the deadline was set so that subjects met it 90% of the time. If the subject failed to meat the deadline, a tone sounded and a computer flashed the message, "You failed to meet the deadline". Loss of control was arranged by either allowing subjects to control whether or not they received feedback as to their reaction time after each trial (neutral condition) or, as in the relevant condition, having each subject receive

feedback on a yoked basis, according to whether a neutral subject received feedback. That is, relevant condition subjects had no control over when they received feedback.

Results indicated that Type A subjects in the relevant condition showed significant decreases in pulse transit time (representing increased sympathetic influence) compared to Type A subjects in the neutral condition. Pulse transit time (PTT) responses in Type B subjects were similar in both conditions. Actually, Type A subjects in the neutral condition evidenced significantly less of a decrease in PTT from baseline than did Type Bs in this condition. This finding suggests that under conditions that are not sufficiently challenging or "relevant" to the Type A behavior pattern, Type A subjects may actually be hyporesponders.

In the second experiment of the Goldband (1980) study, A and B subjects performed two physically stressful tasks (a cold pressor task and a task involving inflating 4 balloons until they burst) that did not involve specific manipulations relevant to the Type A behavior pattern, while HR and PTT were measured. Although both Type As and Type Bs showed some reactivity in physiological responses, there were no group differences. Taken together, the findings from the two Goldband experiments strongly support the notion that

the physiological hyperreactivity of Type A subjects is highly situation-specific, occurring only in response to certain types of environmental stressors relevant to the Type A pattern.

The notion that Type A persons have an enhanced need to control their environments has been given some attention in the psychophysiological literature. Previous reaction time studies have demonstrated that when subjected to uncontrollable stress (e.g. loud uncontrollable noise, noncontingent electric shock), Type A subjects exert increased effort to escape these stressors (e.g. faster reaction times), while Type Bs decrease their efforts in the face of uncontrollability (Brunson & Matthews, 1981; Glass, 1977).

For example, in a study reported in Glass (1977), Type A and Type B subjects were presented with a choice reaction time task on which a certain response either terminated (ESCAPE) or had no control over 100 dB noise bursts (NO ESCAPE). In comparison with Type B subjects, Type As showed slower reaction times when their responses were effective in terminating the noise, but significantly faster responses when there was no relationship between their behavior and the noise bursts. In fact, while Type As' reaction time speeds increased in the NO ESCAPE condition, those of the Type B subjects decreased. Glass interpreted this finding as being congruent with his

conception of Type A behavior as a response style elicited by conditions that threaten an individual's sense of environmental control. When initially confronted by each event, Type As, compared to their Type B counterparts, exert greater efforts to master the threatening stimulus.

This enhanced effort to exert control on the part of Type As is similar to Obrist's notion of "effortful active coping" (Obrist, 1976), which is characterized by a subject's attempt to perform well on highly difficult tasks. In a series of studies, Obrist, Light and their associates have demonstrated an enhancement of beta-andrenergic activity in subjects performing a shock avoidance reaction time test (Light, 1981; Light & Obrist, 1980(a); Light & Obrist, 1980(b); Obrist, Gaebelin, Teller, Langer, Grignola, Light, & McCubbin, 1978).

To investigate the cardiovascular and neuroendocrine effects of effortful active coping on Type A persons, Contrada, Glass, Krakoff, Krantz et al. (1982) measured circulating catecholamines as well as heart rate and blood pressure while Type A and B subjects performed a choice reaction time test. In one condition, subjects could avoid aversive stimulation (loud noise bursts and/or electric shocks) by attaining a predetermined reaction time speed (Contingency), while subjects in the other condition were informed that

their responses had no control over aversive stimulation (No Contingency). Within each condition, half the subjects received a high frequency of aversive stimulation (High FAS) while the other half received a low frequency (Low FAS). Within the Contingency condition, High and Low FAS signified failure or success at meeting the reaction time criterion. It was predicted that Type As would be more reactive in FAS since this condition would elicit increased attempts to master the task. As predicted, Type A subjects evidenced greater physiologic (increases in norepinephrine) and behavioral (faster reaction time) hyperresponsivity under High FAS but only with the Contingency manipulation. It was found that when exposed to uncontrollable noise, Type As evidenced greater decreases in pulse transit time and faster reaction times than Type Bs.

Although there is considerable evidence from the psychophysiological literature suggesting that Type A individuals exhibit heightened autonomic responsivity to stress when compared with Type Bs, it should be noted that some studies have reported no differences between As and Bs (Diamond & Carver, 1980; Lott & Gatchel, 1978; Krantz et al., 1982). As Matthews (1982) pointed out, however, these studies were more likely to have included women (Lott & Gatchel, 1978) or cardiac patients (Krantz et al., 1982)

as subjects, classified subjects according to JAS scores, or imposed tasks which failed to elicit large responses from any subject (Diamond & Carver, 1980).

Type A Subcomponents, Environmental Demands, and Sympathetic Nervous System Activity

One relatively new line of investigation has been to examine separately the subcomponents of the Type A behavior pattern. It has frequently been noted that while the global Type A behavior pattern predicts the occurrence of CHD, it has not been determined which of the subcomponents of the behavior pattern (e.g. potential for hostility, competitive drive, impatience, and vigorous voice stylistics) is actually placing one at risk for heart disease, or whether all the subcomponents are equal in this respect (Glass, Krakoff, Contrada, Hilton, et al., 1980; Scherwitz, Leventhal, Cleary, & Laman, 1978). It is conceivable that some of the subcomponents, for example competitive drive and/or impatience, may elicit cardiovascular activity disposing Type A persons to CHD, while others, such as potential for hostility and vigorous voice stylistics, may not. Identifying the subcomponents of the Type A pattern that result in elevated levels of sympathetic autonomic activity would not only enhance our understanding of Type A-CHD relationship, but, from a clinical perspective, would begin to delineate those aspects of

the behavior pattern in greatest need of modification. There have been only a few studies which have explored the relationship between the subcomponents of the Type A pattern and arousal of the sympathetic nervous system.

In their study with Type A women, MacDougall, Dembroski, and Krantz (1981) found that the Potential for Hostility subcomponent of the SI was the only component that significantly predicted physiologic response in a reaction time task. Dembroski, MacDougall, Shields et al. (1978) also found the potential for hostility to be substantially correlated with elevations in both SBP and HR. Subjects who scored higher on this scale tended to show greater increase in systolic blood pressure and heart rate than subjects who scored lower. Attributes such as verbal competitiveness, rapid/accelerated speech, and speed and impatience (as measured by the Structured Interview) correlated strongly with only one physiological measure (either heart rate or systolic blood pressure).

The importance of these first subcomponent studies by Dembroski, MacDougall, and associates is that they began to more clearly delineate the relative contribution of each of the separate Type A dimensions. High scores on certain Type A subcomponents correlated significantly with physiological arousal. While a correlational approach might provide important information concerning the subcomponent-sympathetic

arousal interaction, a more convincing and potentially more clinically useful approach would involve placing Type A subjects in particular situations which might evoke certain Type A characteristics while physiological arousal is assessed. For example, to determine if competition is a Type A subcomponent which might lead to pathogenic autonomic arousal, Type A subjects could be placed in challenging competitive situations, while the appropriate dependent measures are taken. This experimental approach is more useful than the correlational approach in at least two respects. First of all, since the Type A subcomponents are perceived as responses to specific environmental stimuli, it seems more appropriate to measure physiological arousal while subjects are in conditions which approximate such environmental stimuli. Secondly, from a more applied perspective, the ability to discern the environmental circumstances under which pathogenic autonomic arousal occurs would be valuable in the development of intervention programs aimed at decreasing Type A behavior. That is, by elucidating experimentally the relevant environmental factors, clinicians can be more efficient in helping Type As alter their pattern of responding to these factors.

One might argue, however, that demonstrating heightened sympathetic autonomic arousal in Type A persons under

conditions of, say, competition, is of little importance, inasmuch as their JAS scores or structured interview responses already suggest that this would occur. That is, if subjects say they are very competitive, it is expected that they would show hyperarousal in competitive situations. This, however, may not be the case. Glass et al. (1980) conducted the only study to date in which the subcomponents of the behavior pattern were experimentally manipulated while psychophysiological measures were taken. These researchers examined the physiological effects of competition and hostility in Type A and B men. Subjects competed at an electronic board game with a competitor who said nothing, and also with a hostile competitor who continuously harassed the subject (e.g. "Come on, you aren't even trying"). It was found that Type As exhibited more autonomic arousal than Type Bs only under the hostile competition conditions. This study by Glass and associates suggests that the relationship between situational factors, Type A behavior, and physiological arousal may not be a simple one, since competition alone did not differentiate between groups, while the interaction of competition and hostility did. Simply placing Type A and Type B persons in situations which mimic the Type A subcomponents, therefore, may not yield group differences. It is necessary to look also at the interaction of subcomponents and situation

demands, and at the exact nature and parameters of the environmental challenges. That is, emphasis should be placed on the interaction and specificity of situational demands.

One subcomponent of the Type A pattern which may be critical to the development of later CHD is time urgency or impatience. Previous research comparing As and Bs on this dimension has revealed that Type As tend to arrive earlier for appointments (Gastorf, 1980), estimate the end of a time interval sooner (Brunan et al., 1975), exhibit greater behavioral hyperactivity when forced to wait (Glass, Snyder, & Hollis, 1974), show longer response latencies on choice reaction time tasks involving long preparatory intervals (Glass, 1977), and evidence irritation when forced to slow down the rapid pacing of their activities (Glass et al., 1974). In fact, in many of the psychophysiological studies conducted using Type As, the experimental tasks contained challenges which placed subjects under time pressure. For example, in studies using reaction time (e.g. Dembroski, MacDougall, Shields, Petitto, & Lushene, 1978) and cognitive tasks (e.g. Manuck, Craft, & Gold, 1978; Manuck & Garland, 1979), subjects are typically instructed to "respond as rapidly as possible". In essence, then, there appears to be strong behavioral evidence and, to a lesser degree, psychophysiological support for the notion that Type A individuals

exhibit a unique response to time pressures. No study to date, however, has systematically investigated the possible role time urgency might play in the elicitation of pathogenic sympathetic arousal. That is, a question remains concerning whether Type A individuals evidence sympathetically mediated cardiovascular hyperactivity in situations involving extended waiting periods. For example, do Type A individuals show a systematic increase in cardiovascular hyperarousal corresponding with increments in the time they must wait?

As noted earlier, it may not be sufficient simply to place subjects in challenging situations which mimic Type A subcomponents, since this approach does not always yield A-B differences (e.g., Glass et al., 1980). Rather, it may be more important to explore how different situations interact to augment autonomic arousal in Type A persons. In fact, it is conceivable that many of the challenging situations to which individuals are exposed outside of the laboratory are multi-dimensional, comprising a number of simultaneously occurring demands. With regard to the time urgency component of the Type A behavior pattern, conditions which require extended waiting periods may interact with other situational demands to enhance autonomic reactivity in Type As -- over and above that elicited by waiting alone. One factor which

might interact with waiting to enhance autonomic reactivity is the uncertainty or unpredictability of the waiting period. Given the often-reported need for environmental control attributed to Type A persons, it might be that unpredictable or uncertain conditions decrease their sense of control, and consequently increase their efforts to gain control -- processes which might ultimately lead to enhanced autonomic responsivity.

Type A Behavior Pattern and Cortical Activity (Event-Related Potentials)

Event-related potentials (ERP) are scalp-recorded manifestations of neural events associated with the processing of environmental stimuli. Their waveforms depend on the location of the electrode and voltage source, and on the nature of the experimental manipulations. While ERPs have been used by some researchers to study differences between selected groups of subjects (e.g., Calloway, 1975), they have yet to be used in the study of the Type A behavior pattern. Two ERP components are particularly relevant to the purposes of this experiment, the $\overline{\text{N200}}$ and $\overline{\text{P300}}$ components. The $\overline{\text{N200}}$ component is a negative-going shift which occurs around 200 msec after presentation of an attended or task-relevant stimulus or a stimulus with features common to the relevant stimulus, and is presumably reflective of modality-specific

selection processes (Harter & Guido, 1980). The $\overline{P300}$ component is a positive voltage change with a latency of approximately 300 msec following a task-relevant event (Duncan-Johnson, 1981) and is maximal when recorded over the parietal region. Among other things, $\overline{P300}$ amplitude and latency are sensitive to the subjective probability of the target stimulus (Donchin, 1980; Duncan-Johnson, 1978; Squires, Wickers, Squires, & Donchin, 1976), while its latency also varies with the difficulty of a discimination (Duncan-Johnson, 1981; Johnson, Pfefferbaum, & Kopell, 1981; Kutas, McCarthy, & Donchin, 1977). These ERP components might serve as important indices of increased effort on the part of Type A subjects -- which, as noted above, might be elicited by conditions of uncertainty or unpredictability. It could be that Type A individuals are not only autonomically hyperreactive to challenging situations, but may exhibit augmented neural information processing as well. In addition, an accelerated rate of information processing in Type As would provide neural evidence for their aforementioned tendency toward and preference for rapid pacing of their motor activities.

Purpose and Predictions

The purpose of the present study was to explore the hypothesis that extended waiting and uncertainty play a

significant role in eliciting heightened levels of sympathetic and cortical arousal in Type A as compared to Type B individuals. Tonic (sustained) and phasic (transient) autonomic activity and ERPs of Type A and Type B subjects were measured while they performed a reaction time task emphasizing speed, with either a relatively short or long intertarget interval (ITI) and with either low or high certainty as to length of the ITI. The study determines whether greater sympathetic autonomic arousal in Type As depends on 1) how long they must wait and 2) how certain the interval length is. It also determines if Type As respond to conditions which might engender effortful active coping (e.g., uncertainty) with enhanced cortical responsivity.

It was predicted that Type A subjects would evidence more sympathetically mediated autonomic reactivity (as indexed principally by pulse transit time) than Type B subjects during the longer ITIs. Long waiting intervals prior to responding were expected to affect the impatient or time-urgent component of the Type A pattern, leading to enhanced sympathetic responsivity. The length of ITI was not expected to differentially affect cortical activity in Type As and Type Bs. It was also predicted that Type As would exhibit greater sympathetic autonomic reactivity than Type Bs under conditions of uncertainty. This prediction stemmed primarily from the Laceys' (Lacey & Lacey, 1970, 1974) research which

showed that on fixed foreperiod reaction time tasks, heart rate actually decelerates as the time of the target stimulus approaches, and immediately accelerates following stimulus occurrence, and that these phasic heart rate changes are under parasympathetic control (Obrist, Langer, Light, Grignolo, & McCubbin, 1979). Type A individuals have been shown to evidence these stimulus-bound cardiac changes (Goldband, 1980), and under some conditions show greater prestimulus heart rate decreases than Type Bs (Jennings & Choi, 1981). Therefore, Type A subjects were expected to show less sympathetically mediated autonomic changes under conditions where the ITI is relatively certain. Conversely, when the waiting interval is less certain, the stimulus-locked cardiac deceleration should not occur since stimulus occurrence is unpredictable. Instead, it is expected that Type As would exhibit greater sympathetic autonomic arousal than Type Bs under these conditions. This prediction comes from the idea that Type A persons are more concerned than Type Bs with controlling their social and physical environment (Glass, 1977; Williams et al., 1978), and the finding that Type As will work harder to gain control over uncontrollable stressors than Type Bs. Therefore, following Obrist's notion of effortful active coping, it was expected that the pattern of autonomic arousal would suggest greater sympathetic innervation, and that Type As would exhibit greater

arousal than Type Bs. It is also during this low certainty condition that, due to increased effort and sustained attention, Type A subjects will exhibit a greater enhancement of cortical activity than Type B subjects. Finally, it was predicted that the combined effects of long waiting periods and low certainty would also lead to greater cardiac and cortical responses in Type As as compared to Type Bs.

CHAPTER II

METHOD

Subjects

Subjects were 20 male undergraduate volunteers selected from the pool of introductory psychology students (see Behavior Pattern Assessment below). All subjects were paid $10.00 for their participation in the study. All subjects were nonsmokers and each was asked to abstain from drinking coffee for four hours prior to participation. The average height of the Type A subjects was 5 ft., 10 inches and was 5 ft., 11 inches for Type B subjects. The average weight for the Type A subjects was 168 lbs. as compared to 171 lbs. for the Type Bs.

Behavior Pattern Assessment

Subjects were selected for participation in this experiment based on their scores on the JAS - Form T (Krantz, Glass, & Snyder, 1974). The JAS were given to male introductory psychology students and later scored along the A-B dimension (see Appendix A for copy of JAS).

Of the 54 items on the JAS, 21 are used to classify subjects as Type A or Type B (see circled items on JAS). Typically, these 21 items are scored by assigning a weighting of 1 to responses designated to be Type A, and 0 to Type B responses. The exact score at which subjects are labeled

Type A or Type B has varied across previous studies. For example, subjects scoring 8 or below have been labeled Type B and those scoring 10 or above, Type A (Goldband, 1980); or subjects scoring above the median of all scores labeled Type A and those below as Type B (Krantz et al., 1974). For the purposes of the present study, an attempt was made to choose subjects with extreme scores. Therefore, of the 80 students who completed the JAS, the 10 with the highest scores and the 10 with the lowest scores were selected as subjects. This method resulted in a mean Type A score of 14.2 and a mean Type B score of 5.2.

Apparatus

Heart rate was measured using a Glass (Model 7P4) cardiotachograph in conjunction with a low-level DC amplifier (Model 7P3). Pulse transit time was measured using an AC amplifier (electrocardiogram) Glass Photoelectric Transducer (Model PTTN) (finger pulse) and two Schmitt triggers. Blood pressure was measured manually using a sphygomanometer and stethoscope. A Fabritek 1062 Signal Averager was used for measuring phasic heart rate and phasic pulse transit time. Electroencephalograms for measuring evoked potentials were amplified with Grass 785 wide-band AC preamplifiers in a Grass 7WC polygraph with half-amplitude high and low frequency filter set at 35 and 1 Hz, respectively. Cortical activity was averaged with a Fabritek 1062 Signal Averager

for 500 msec following the stimulus flash. Each average evoked potential was based on 34 trials.

Stimuli

The stimuli in this experiment were a white circle and a white square. The figures were presented in a 1.92°-diameter black display surrounded by a white field. Stimuli were presented at a rate of 1/500 msec, and each was displayed for a duration of 40 msec. The white circles served as nontarget while the white squares were target stimuli. The frequency of occurrence of the squares was dependent upon the experimental condition (see Conditions below).

Task

Subjects were instructed to respond to the white square by lifting their left index finger from a button mounted on a wooden board and then returning the finger as quickly as possible. The lifting of the finger constituted a response. Subjects were signaled by a "click" if their response times were longer than 350 msec.

Experimental Conditions

The target stimuli were presented on four schedules varying in the certainty of the intertarget interval (high certainty vs. low certainty) and the length of the ITI (6 sec vs. 18 sec). In the high certainty conditions, subjects were aware of the time interval between the

occurrence of successive square stimuli (targets). Two interval lengths were used: a 6-sec and an 18-sec interval.

In the low certainty condition, subjects were unaware of the interval between the occurrence of the successive squares. The interval varied randomly from 1 to 30 seconds. Two average interval lengths were used: a 6-sec and an 18-sec interval.

Design

The experiment was designed to examine the cardiovascular and cortical effects of waiting different lengths of time (6 and 18 sec on the average) under different conditions of certainty (low vs. high) before responding on a reaction time task in Type A and Type B males. To examine these issues, a three-factor mixed design was used, with repeated measures on two factors (see Figure 4). The two within-subject factors were ITI Certainty (high vs. low) and ITI Length (6 vs. 18 sec), and the between-subjects factor was Subject Type (A vs. B).

Dependent Measures

Reaction time. The latency of reaction time (RT) on each trial was measured. Mean reaction time across 34 trials per condition was derived for each subject. Other behavioral measures were percentage of "hits" or "false alarms," represent-

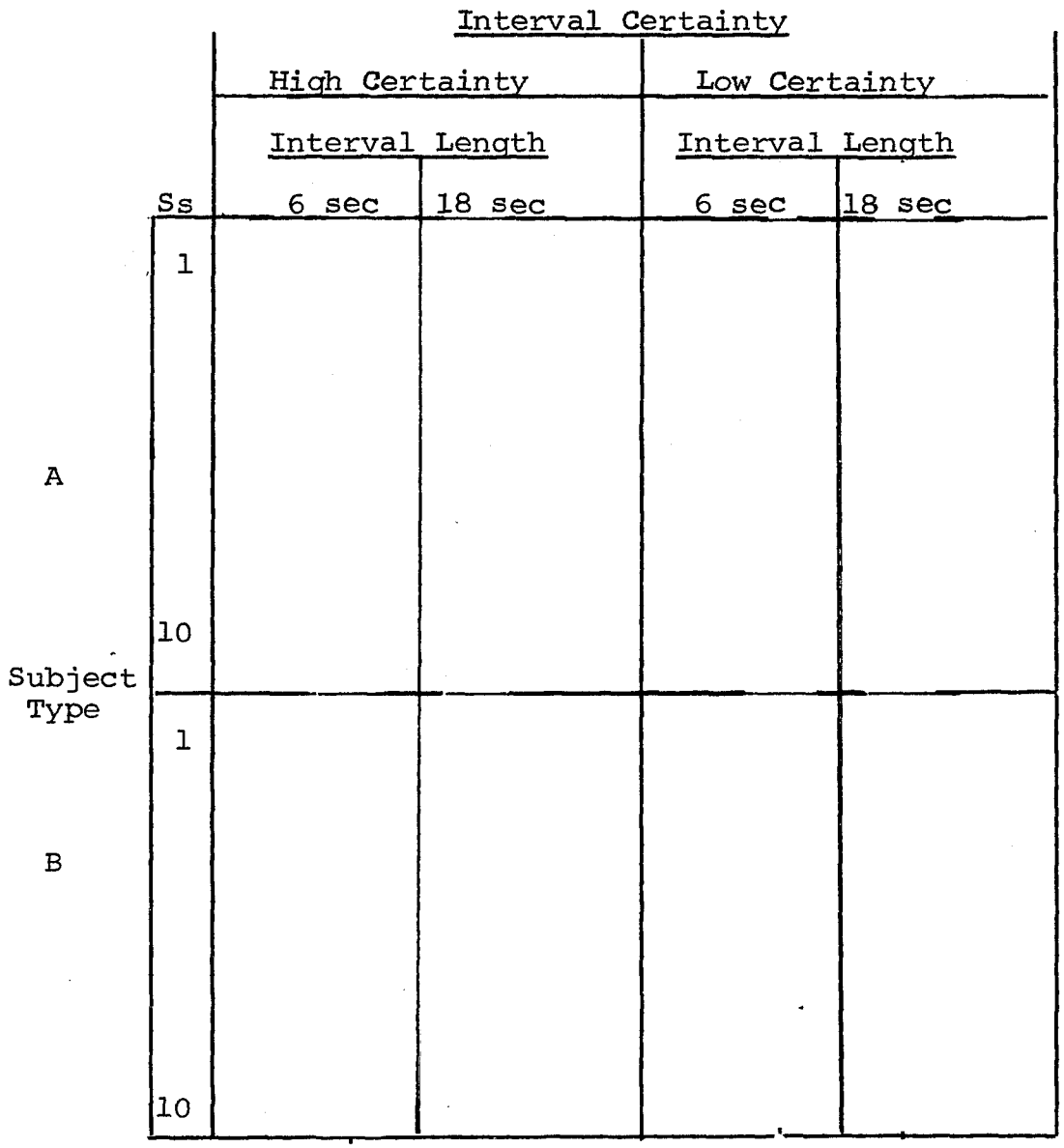

Figure 4. Design matrix for experimental conditions, showing repeated measurements on interval certainty (high and low), and interval length (6 and 18 seconds) for Type A and B subjects. The presentation of conditions was counterbalanced across subjects.

ing responses occurring within 350 msec. of target and non-target stimuli, respectively.

Subjective tension rating. Each subject rated their subjective level of tension before and after each condition using a 100-mm visual analogue scale. The scale consisted of a 100-mm vertical line with the bottom end labeled "NO TENSION" and the top labeled "MOST TENSION POSSIBLE". To rate their tension, subjects simply placed a horizontal slash at any point across this line. Tension ratings then were quantified in terms of millimeters from the NO TENSION endpoint.

Event-related potentials (ERP). Evoked potentials were recorded with Grass gold-cup scalp electrodes located over the parietal (Pz) and frontal (Fz) regions of the scalp with the earlobes serving as references. Electrode placement followed the International 10-20 system (Jasper, 1958). Resistance was reduced by rubbing these locations briskly with a Q-tip and electrode cream. The ERP was quantified by using the average voltage of the first 50 msec of each ERP waveform as a baseline. The amplitude and latency of specific points along the ERP waveform were used as dependent measures: the most negative deflection between 150 and 300 msec and between 300 and 450 msec, and the most positive deflections between 250 and 350 msec and between 350 and 500 msec.

Both the latency and amplitude of each deflection were measured and subjected to statistical analysis.

Tonic heart rate (THR). Heart rate was measured using standard gold cup electrodes. Before electrodes were positioned, the placement areas on the skin were cleaned. Electrode cream was placed inside the electrode cup, which was held on the surface of the subject's skin using adhesive tape. Placement of the electrode followed the standard limb lead II method (Stern, Ray, & Davis, 1980) which involves placing one electrode on the right arm and another on the left leg. This right arm - left leg placement was chosen since subjects responded with their left hand, and thus movement artifact was reduced. Heart rate was obtained by having the EKG spike trigger a counter. Tonic heart rate was quantified in terms of number of beats per minute.

Phasic heart rate (PHR). In order to ascertain variations in heart activity which were temporally related to stimulus presentation, PHR changes occurring before (at times -1.0 and -0.5 sec), following (at times +0.5 and +1.0 sec) and at the time of target stimulus (time 0.0) were measured. Phasic heart rate was measured by computer sampling of heart rate occurring during the aforementioned 2-second interval, which was printed on graph paper as a horizontal line representing the average phasic heart rate across the

34 trials. Using time 0.0 as a baseline, the difference in the slope of the horizontal line between this point and the four other points was measured in terms of millimeters. The computer was calibrated such that one millimeter represented two beats per minute. A counter recorded the inter-beat-interval (IBI) occurring at the time of the target stimulus (time 0.0) which served as reference for the quantification of heart rate at the other sampling times. All values (including IBI at time 0.0) were converted into heart rate in terms of beats per minute.

Tonic pulse transit time. Pulse transit time (PTT) is defined as the interval between the onset of ventricular electrical activity and the arrival of the pulse at a peripheral site (Weiss, Note 4). Stated differently, it is the time required for the pulse wave to travel from the heart (after contraction) to a distant location, such as an arm or finger, or from one point to another point (Stern et al., 1980). The importance of pulse transit time as a dependent measure in this study rests on its sensitivity to sympathetic nervous system effects on the myocardium, and therefore catecholamine secretion. Several studies using the beta-blocking agent propranolol have demonstrated a consistent relation between beta-adrenergic (sympathetic) inhibition and increases in pulse transit time (decreases in PTT reflect beta-adrenergic excitation) (Obrist, Light, McCubbin,

Hutcheson, & Hoffer, 1979; Weiss, Del Bo, Reichek, & Engelman, 1980). Therefore, PTT was chosen as the best noninvasive indicator of sympathetic nervous system activity.

Pulse transit time was measured by recording the time delay from the onset of the R-wave of the EKG to the onset of a vasomotor pulse at the distal end of the radial artery. The occurrence of the R-wave activated a Schmitt trigger which in turn started a timer; the timer was terminated by a second Schmitt trigger which was activated by the arrival of the pulse wave at the distal end of the radial artery. The sum of every three R-wave-to-pulse-wave cycles was printed by a print-counter and later divided by three to get mean pulse transit time for three heart beats.

Phasic pulse transit time. As with HR, PTT was meas- used just prior to, just after, and at the time of the target stimuli. In this case, phasic PTT was sampled at times -1.0 sec, -0.5 sec, 0.0 (time of the target stimulus), and +0.5 sec. To measure phasic PTT, the first EKG R-wave-to-pulse-wave interval following each occurrence of the four stimuli (occurring at the times above) was computer-sampled. This produced four frequency histograms reflecting the distribution of PTTs relative to each stimulus across 34 trials. Mean PTT for each histogram was derived by measuring half

of its amplitude (mode) at the midpoint of the distribution; then the distance between the point of stimulus onset and the mode of the frequency histogram was measured, whereby 1 cm equaled 100 msec.

Blood pressure. Both systolic and diastolic blood pressure were measured manually before and after each condition. In all, then, a total of 8 blood pressure readings were taken. Blood pressure was measured using a sphygmonanometer and stethoscope.

Procedure

Upon their arrival at the Psychophysiology Laboratory, each subject was told that the purpose of the experiment was to study the interaction of behavior and physiological processes. They were informed that their task would be to respond by releasing a lever whenever a certain stimulus appears, and that their cardiovascular responses would be recorded as they worked on the tasks. At this time, subjects were asked to sign a consent form if they still wished to participate.

Subjects were then taken into the experimental chamber where electrodes were put in place. A 5-minute baseline was taken on the recordings, after which the experimenter played a recording of the following instructions:

> On the screen in front of you is a black display panel on which a series of circular lights will appear. Occasionally, a square light within the series of circular lights will appear. Your task is to respond as rapidly as possible to the square light by lifting your finger from the button on the board in front of you. Please observe the screen carefully since the lights will be flashing very rapidly, and it is important that you make a response to each square light. In the event that you fail to respond to a square light, or your response is too slow, you will hear a click, and thereafter should try to improve your speed. If your reaction times are on the average faster than those of any other subject in the study, you will receive a $10.00 cash prize in addition to your payment for participation.

At this time, when subjects were in the high certainty conditions, they were told that the square figure would appear at fixed-time intervals (either 6 or 18 seconds apart depending on condition). When subjects were in the low certainty conditions, they were told that the square stimulus would appear at unpredictable intervals, but that it would occur between 1 and 30 seconds and, on the average, would occur either every 6 or 18 seconds.

Following each condition, the subject was asked to relax while another 5-minute baseline was taken on the dependent measures. Subjects were then told what the next condition would be and were reminded to respond as rapidly as possible. For each experimental condition there were 34 reaction time trials.

Data analysis. All behavioral measures (reaction times, hits, and false alarms) and ERPs were subjected to a repeated measure analysis of variance (SAS program). Then tension and autonomic data (THR, TPTT, SBP, DBP, PHR, PPTT) were analyzed using an analysis of covariance with the precondition baseline serving as the covariate. All data will be graphically presented as change scores. This procedure was used since research has shown that even nonsignificant baseline differences between groups or individuals can influence responsiveness to experimental manipulations (Benjamin, 1967; Kinsman & Staudeumayer, 1978). Since there were extremely large variance differences between Type As and Type Bs in pulse transit time, a log transformation procedure (Winer, 1971) was performed on these scores to make the variances more homogeneous (SAS program). Multiple comparisons of between-individual means were carried out using the Neuman-Kuel's Multiple Range Test.

CHAPTER III

RESULTS

Behavioral and Subjective Measures

The independent variables used in the analysis of the behavioral and subjective measures were subject Type (A vs. B), ITI Certainty (high vs. low), and ITI Length (6 sec vs. 18 sec), creating a 2 x 2 x 2 mixed design. All figures and tables which follow can be found in Appendix B.

Reaction time, false alarms, and hits. The data for reaction time are shown in Figure 5. Reaction times were significantly shorter during the high as compared to low certainty condition (ITI Certainty effect, F (1, 18) = 22.68, $p < .001$). Reaction times were also faster during the 6-sec than 18-sec ITI Length condition (ITI Length effect, F (1, 18) = 76.45, $p < .001$). Figure 6 shows that there was a greater percentage of hits during the 6-sec as compared to the 18-sec ITI condition (F (1, 18) = 12.10, $p < .002$). Other main effects or interactions reaction time, false alarms, and hits did not approach statistical significance.

Tension. No significant main effect or interactions were found for subjective tension ratings.

Tonic Autonomic Measures

All data on autonomic measures unless otherwise stated are presented in terms of change scores from baseline. For blood pressure, change scores were calculated by subtracting the baseline mean from the Post-condition score (Post-Pre). For heart rate and PTT the baseline mean was subtracted from the mean of the condition (Condition $\bar{x}$-baseline $\bar{x}$).

No significant main effects or interactions were found for tonic PTT or systolic blood pressure (see Figures 7 and 8). The apparent main effect of Type on topic PTT was due primarily to extremely large change scores for one Type A subject which dramatically influenced the group mean.

Tonic heart rate. The data for tonic heart rate are shown in Figures 9 and 10. Under both ITI Length manipulations, Type Bs evidenced a decrease in heart rate from baseline (Figure 10). However, as seen in Figure 9 (bottom graph), the overall decrease in heart rate for Type B subjects during the 6-sec condition was due primarily to their large decrease in heart rate during the low certainty 6-sec condition. In contrast, Type As showed only a slight decrease during the 6-sec condition and an increase in heart rate during the 18-sec condition. The ITI Length x Type interaction was statistically significant F, $(1,18) = 59.1$, $p < .04$. Other main effects on tonic heart rate were not statistically significant.

Diastolic Blood pressure. The data for diastolic blood pressure are shown in Figure 11. Higher blood pressures occurred during the low certainty condition (Certainty, F,(1,18) = 4.97, $p < .03$). However, inspection of the figure shows that higher mean diastolic blood pressure during the low certainty condition tended to be due to Type As subject's response rather than Type Bs, although this interaction did not approach statistical significance.

Phasic Autonomic Measures

Phasic heart rate. Heart rate was sampled at 5 points in time in reference to the target stimulus: 1.0 sec before (-1.0 sec), 0.5 msec before (-0.5 sec), at the time of the target stimulus (0.0), 0.5 msec after (0.5 sec), and 1.0 sec (1.0 sec) after the target stimulus. These sampling points introduced into the analysis a new independent variable labeled Time. Therefore, the phasic heart rate analysis consisted of the factors Type (A vs. B) by ITI Certainty (high vs. low) x ITI Length (6 sec vs. 18 sec) x Time (-1.0, -0.5, 0.0, +0.5 and +1.0). The results for phasic heart rate are shown in Figure 12.

As can be seen in Figure 12 there was a greater deceleration in heart rate occurring across the time intervals during the high certainty than low certainty condition (Certainty x Time interaction, F (9, 180) = 24.7, $p < .001$). During the high certainty conditions heart rate at time -1.0 was significantly greater than that occurring at Time 0.0 ($p < .01$) and

at Time +1.0 (p .01). Figure 12 also suggests a greater decline in heart rate during the 6-sec ITI condition, particularly with high certainty. There was in fact a significant ITI Length x Time interaction, F (9, 180) = 24.7, $p < .0001$, with a greater deceleration in phasic heart rate occurring during the 6- as compared to the 18-sec ITI condition. There was also a significant Time effect, F (4, 90) = 37.15, $p < .0001$, with heart rate decreasing from time 1.0 sec before to 1.0 sec after the target stimulus.

Phasic pulse transit time. Figure 13 shows the data for phasic PTT. Type A subjects evidenced shorter phasic PTT than Type Bs during the low certainty condition for all time periods except -0.5, whereas Type Bs showed shorter PTTs during the high certainty condition across the same time periods (Type x Certainty x Time interaction, F (10, 174) = 2.46, $p < .009$). Both Type As and Type Bs exhibited decreases in phasic PTT from their baselines (corresponding to increase in sympathetic activity) for all time periods. Type As, in addition, showed shorter phasic PTTs than Type Bs during the 6-sec ITI condition (Type x Length x Time interaction, F (18, 174) = 2.34, $p < .01$) at all time periods except -0.5. (See Figure 14.) However, this interaction was due primarily to the shorter PTTs in Type As during the low certainty condition, since As and Bs were similar in PTT during the high certainty

6-sec condition. Finally, Type As showed overall greater shorter PTTs than Bs particularly at time +0.5 msec, but not at time -0.5 (Type x Time interaction, $\underline{F}$ (5, 89) = 2.23, $\underline{p} < .05$). Again, the effect can be attributed primarily to the effects of the low certainty condition (Figure 13).

Correlations. Overall, there was a significant negative correlation (-.23) between heart rate and PTT ($\underline{p} < .04$). As heart rate increased PTT decreased, suggesting sympathetic nervous system activation. Separate correlations x Type revealed a significant negative correlation between heart rate and PTT for the Type A group only ($\underline{p} < .05$).

Event-Related Potentials (ERP).

All ERP measures were subjected to separate analyses of variance. The factors in each analysis were Type (A,B), ITI Certainty (high vs. low), ITI Length (6 vs. 18 sec), Time (-1.0, -0.5, 0.0), and Electrode Location (Fz, Pz). Multiple comparisons were made using Newman-Kuel's Multiple Range Test. Analyses were performed on four components of the ERP: the most negative wave between 150 and 300 msec, and between 300 and 450 msec, and the most positive wave between 250 and 350 msec, and between 350 and 500 msec. Hereafter, these four peaks will be labeled according to their mean latencies: N201, N371, P295, and P436. Raw ERP waveforms for Type A and Type B subjects for each condition are shown in Figure 15. These waveforms represent subject responses to target stimuli at the parietal location. Evoked potential

data were analyzed using nine Type A subjects and nine Type B subjects, since the ERPs from one Type A and one Type B subject were omitted due to equipment difficulties.

N201. The data for N201 latency are graphed in Figure 16. Graphed are the Type A/B N201 latencies in response to the target (0.0 sec) and preceding nontarget stimuli (-1.0, -0.5 sec). The most striking feature of the graph is the consistently shorter latencies for Type As as compared to Type Bs for all conditions and particularly at time 0.0, with the exception of the high certainty 18-sec condition (Type effect, F (1, 16) = 5.22, $p < .03$; Type x Time, F (3, 48) = 3.5, p .02). The A/B differences on N201 latency was greatest during the low certainty 18-sec condition and primarily at time 0.0 (interaction of Type x Certainty x Length x Time, F (3, 48) = 3.53, $p < .02$). This interaction indicates that A's N201 latencies were much shorter than B's during the low certainty 18-sec condition, and primarily at time 0.0 sec in response to the target stimulus.

Other effects were also significant. N201 occurred earlier in response to the target stimuli as compared to preceding nontarget stimuli (Time effect, F (2, 48) = 27.44, $p < .0001$). Shorter N201 latencies to nontarget stimuli (-1.0 and -0.5 sec) were found at the frontal location, while target stimuli (0.0 sec) produced shorter N201 latencies at the parietal location (Electrode Location x Time, F (5, 96) = 12.68, $p < .0001$). No main effects or interactions were significant on N201 amplitude.

P295. The data on P295 latency are shown in Figures 17 and 18. Type As generally had shorter P295 latencies during the low certainty condition while Type Bs generally had shorter latencies during the high certainty condition (Type x Certainty, F (1, 16) = 6.09, $p < .02$) (Figure 18). Figure 17 shows that during the high certainty condition, Type B's P295s were shorter primarily in response to the target stimuli (time 0.0); however, under conditions of low certainty, Type As had a consistently earlier P295 to both targets (0.0) as well as nontargets (-1.0, -0.5) (Type x Certainty x Time, F (3, 48) = 3.12, $p < .03$).

Other main effects and interactions were also significant. P295 occurred earliest when the ITI was 6-sec as compared to 18-sec (290 vs. 300 msec) (ITI Length, F (1, 16) = 6.2, $p < .02$). Figure 19 illustrates that P295 latencies decreased across time during the 6-sec condition, whereas P295 latencies increased across time during the 18-sec condition (Length x Time interaction, F (3, 48) = 2.73, $p < .05$). P295 latencies decreased between times -1.0 and 0.0 during the high certainty 6-sec condition, but increased between -1.0 and 0.0 sec during the high certainty 18-sec condition. P295 occurred consistently earlier during low certainty 6-sec condition as compared to low certainty 18-sec condition for all time points, and occurred earlier at time 0.0 (in response to the target stimuli) than at times -1.0 and 0.5 (Certainty x Length x Time interaction, F (3, 48) = 4.0, $p < .01$).

Larger amplitude P295 occurred in response to the target stimuli (Time, F (2, 48) = 13.0, $p < .001$), particularly over the parietal location and at time 0.0 (Electrode Location x Time, F (4, 80) = 7.92, $p < .0001$).

N371. The effects of the experimental variables on N371 latency are shown in Figure 20. As with P295, N371 latencies in Type As were shorter than Type Bs during the low certainty 18-sec condition. N371 occurred earlier in response to target stimuli (Time, F (2,48) = 34.76, $p < .001$), and earlier at the frontal location to non-target stimuli (at times -1.0 and -0.5) and earlier at parietal location in response to the target stimuli (Electrode Location x Time F (5,96) = 20.04, $p < .0001$).

Figure 21 shows that N371 became more positive between times -1.0 and 0.0 during the high certainty condition but became more negative between these times during the low certainty condition (Certainty x Time, F (3, 48) = 9.40, $p < .0001$). From Figure 21 it also can be seen that N371 was generally more negative during the low certainty condition as compared to the high certainty condition (Certainty, F (1, 16) = 13.50, $p < .002$). At both frontal and parietal locations, N371 was more positive than baseline during the high certainty condition. In the low certainty condition, however, N371 at parietal was more negative than baseline while the frontal location continued to evidence a positive voltage (Certainty x Location, F (1, 16) = 9.91, $p < .006$).

P436. The effects of the experimental variables on P436 latency are shown in Figure 23. Type Bs had shorter latencies than As, F (1, 16) = 6.05, $p < .02$. Type B subjects also showed a trend toward shorter P436 latencies in response to targets (at Time 0.0) as opposed to nontargets (at time -1.0, -0.5), while As showed an increase (Type x Time, F (3, 48) = 3.07, $p < .03$).

For P436 amplitude a larger response occurred at the frontal location at times -1.0 and -0.5 (nontarget stimuli), and over parietal location at time 0.0 (target stimulus) (Electrode Location x Time, F (4, 80) = 7.29, $p < .0001$). Overall, there was a larger P436 at time 0.0 in response to the target stimulus (Time, F (2, 48) = 13.46, $p < .0001$).

Summary of Results

The results are summarized in Tables B1, B2, and B3. Table B1 shows the main effects of the independent measures on each of the primary physiological dependent measures. Under each column are the levels of the dependent (e.g. Type A vs. Type B) with either a plus or minus sign, denoting whether the dependent measure listed on the far left of the table showed a change which suggested an increase (+), a decrease (-), or no change (0) in autonomic arousal from baseline. In the case of phasic HR, however, the signs denote the direction of the trend in absolute heart rate. Less than (<), greater than (>) or approximately equal

to (≈) signs compare relative influence of the two levels of the independent variable. For the ERPs, the tables illustrate which level of the independent measures produced the shorter latency. As an example, Table B1 shows an increase in tonic heart rate for Type As and a decrease for Type Bs, but the difference was not significant (NS). Likewise, the table shows that P295 latency was shorter during the 6-sec condition and this difference was statistically significant (.02). Tables B2 and B3 are designed to show the interactions of the Type variable with one (Table B2) or both (Table B3) manipulations (Length and/or Certainty). To illustrate, Table B2 shows that tonic heart rate decreased in the 6-sec condition for both As and Bs, and increased in the 18-sec for As but not for Bs. The table shows that this Type x Length interaction was significant at .04. Table B3 indicates that the only significant interaction involving Type, Length and Certainty was on N201 (at .02). It should be noted that the significant effects found with phasic HR in Table B1 and with phasic PTT in Table B2 also included the variable Time (see asterisk *).

CHAPTER IV

DISCUSSION

The purpose of this experiment was to explore the effects of ITI Certainty (high vs. low) and ITI Length (6-sec vs. 18-sec) on cardiovascular and cortical reactivity in Type A and Type B individuals. It was predicted that Type A subjects would evidence greater sympathetic autonomic reactivity when the ITI was relatively long (18-sec), unpredictable (low certainty), and when these conditions were combined. Type As were expected to exhibit greater cortical reactivity than Bs when the ITIs were relatively unpredictable, especially when combined with longer ITIs. The results relevant to these predictions showed the following:

(1) Concerning ITI Length, support for the hypothesis was found in tonic heart rate, which was faster in Type As than Type Bs during the 18-sec condition. Tonic heart rate was also faster in Type As during the 6-sec condition, but the A/B difference in heart rate was greater during the 18-sec condition (Figure 10 and Table B2). Conversely, Type As had shorter phasic PTTs than Type Bs during the 6-sec ITI condition with Bs having shorter phasic PTTs in the 18-sec condition (Figure 14 and Table B2), producing the opposite effect than was predicted. Other interactions of the Type variable with ITI Length on other autonomic

measures were not statistically significant (see Table 3). However, there were faster reaction times and a greater percentage of hits during the 6-sec as compared to the 18-sec condition.

(2) Concerning ITI Certainty, support for the experimental hypothesis was found on phasic PTT and P295 ERP latency. Type As had shorter phasic PTTs and P295 ERP latencies than Type Bs during the low certainty condition (Figure 13 and Table B2). Type Bs had shorter phasic PTTs and P295 latencies under the high certainty condition. Additionally, there were faster reaction times during the high as opposed to low activity condition.

(3) Concerning the combined influence of the ITI Length and Certainty, the predicted effect was found on N201 ERP latency, which was significantly shorter in Type As than in Type Bs during the 18-sec low certainty condition (Figure 16 and Table B3). In addition, the data showed trends in the direction of greater cortical arousal in Type As than Type Bs during the 18-sec low certainty condition on P295 and N371 ERP latencies (Figures 17 and 20).

(4) Other effects occurred which did not involve the Type variable, but which nonetheless demonstrate the differential effects of the experimental manipulations on the dependent variables. There was an overall greater decline

in phasic HR from times -1.0 to 1.0, and a greater decline at these times under the 6-sec as opposed to the 18-sec condition, and during the low compared to high certainty condition. Diastolic blood pressure was higher during the low compared to high certainty condition. The manipulations also produced a large number of ERP amplitude changes which indicate a greater response to target as compared to nontarget stimuli (P295, P436), and higher amplitude response during the low as opposed to high certainty condition (N371). Latency data showed that some ERP components occurred earlier in response to target stimuli compared to nontargets (N201, N371), and when the ITI was 6-sec as opposed to 18-sec (P295).

In summary, although the ITI Length hypothesis was supported by tonic heart rate, it failed to find support in phasic PTT, phasic HR, tonic PTT, and blood pressure. The ITI Certainty hypothesis was supported by phasic PTT and P295 ERP latency, yet failed to be supported by tonic and phasic HR, tonic PTT, blood pressure, and N201, N371, and P436 ERP latencies. The combined effects of ITI Length and Certainty was supported only by N201 latency.

Effect of ITI Length

Of the autonomic measures (tonic heart rate, tonic PTT) and blood pressure, significant effects involving subject type were found only on heart rate. With heart rate, Type B subjects decreased significantly from baseline in both ITI

Length conditions, while Type As' heart rate either maintained (as in the 6-sec condition) or increased (as in the 18-sec condition). This finding supports the hypothesis that longer ITIs might affect the impatient or time-urgent component of the Type A behavior pattern and thus lead to increased arousal. This finding, however, is tempered by the absence of significant changes in blood pressure and particularly PTT. Therefore, it is doubtful that this increase in heart rate for As during the 18-sec condition was under sympathetic nervous system control. While a heart rate increase may be induced by sympathetic excitation, it may also reflect vagal as well as mixed vagal and sympathetic excitation (Obrist et al., 1979). Therefore, in light of the lack of significant changes in the other autonomic measures, particularly PTT, it does not appear that the 18-sec as compared to 6-sec ITI condition produced a higher level of sympathetic activation. It should be kept in mind that blood pressure was measured before and after each condition rather than during, and thus changes occurring while subjects were actually performing the task may have been lost.

This finding of an increase in heart rate for Type As in the absence of significant PTT changes suggests that although the 18-sec ITI condition did not elicit the overall enhanced sympathetic effects on Type As as expected, there was <u>less</u> of a vagal influence in As than Bs. That is, in a task that frequently elicits heart rate deceleration,

Type A subjects were less susceptible to this phenomenon than were Type Bs, particularly when forced to wait relatively long intervals prior to responding. Research from other areas suggest that individuals who are the most sympathetically reactive on tasks evoking sympathetic activity (such as mental arithmetic) are also the least vagally reactive in tasks evoking vagal activity (such as reaction time) (Bunnell, 1982). Thus, Bunnell (1982) suggested that high reactive individuals, as Type As are assumed to be, may be both sympathetically overreactive and have a diminished capacity for vagal restraint. Therefore, Type As may have been more "resistant" to the bradycardial effects of the task. Relatedly, it should be noted that overall there was a significant negative correlation between tonic heart rate and PTT in Type As but not Type Bs, suggesting greater sympathetic activation in Type A subjects.

Unexpectedly, phasic PTT and P295 ERP latency were shorter for Type As than Type Bs during the 6-sec ITI condition while the opposite was true for the 18-sec condition, countering the original prediction. These findings on the effects of the 6-sec condition have implications for how Type As might encounter more frequently those environments which elicit heightened physiological arousal. It was originally hypothesized that the 18-sec condition would enhance reactivity in Type As due to their preference for fast-moving activities and general impatience with

delays, and that when placed in situations which required extended waiting they would be maximally aroused. The current findings suggest that the type of situations which Type As prefer (i.e., faster-paced) may be precisely the kinds which are most arousing for them. This finding is congruent with a recently proposed model of Type A behavior and physiological reactivity (Smith and Anderson, Note 3) which holds that Type As construct stressful (or at least reactivity-engendering) environments in part through their choice of situations. That is, given the opportunity to choose between a faster or slower pacing of activities, Type As will choose the faster pace and exhibit greater autonomic arousal as a function of their choice--the latter being suggested by the current findings.

An alternative although not incompatible explanation for these findings on ITI Length is that the relationship between waiting and sympathetic reactivity in Type As may not be a linear one. That is, increases in sympathetic activity in Type As may in fact coincide with increases in waiting interval up to a certain point, at which time there begins to be a decline in sympathetic activity. If this is true, then the relationship between sympathetic activity and waiting length in Type As might best be represented as an inverted U (see Figure 24). In this experiment, the 6-sec ITI condition might have produced sympathetic activity in Type As which approached the apex of the distribution shown in

Figure 24, whereas the 18-sec ITI condition produced activity similar to that on the declining side. It is therefore possible that if a shorter ITI than 6 seconds had been used, sympathetic activity in Type As might have been more similar to that found for 18-sec condition, in which case the autonomic-waiting interval relationship might have suggested increases in arousal with increases in waiting.

Effects of ITI Certainty

The low certainty ITI condition produced greater phasic PTT decreases and shorter P295 latencies in Type A subjects than in Type B subjects, as predicted. Type Bs had shorter phasic PTTs and P295s under the high certainty condition. Comparisons of the mean phasic PTT changes across conditions for each group separately showed that Type As had only small decrease from their baseline in PTT under the high certainty ITI condition ($\bar{X}$ = -4.1 msec), but evidenced larger decrease in the low certainty ITI condition ($\bar{X}$ = -18.3 msec). In contrast, the high certainty and low certainty manipulations did not produce as great a differential effect in PTT for Type Bs ($\bar{X}$ = -8 and -14.3 msec, respectively). This finding with phasic PTT is consistent with that found by Goldband (1980) where Type As were shown to be more reactive in PTT compared to Type Bs on more challenging reaction-time tasks. On a less challenging reaction-time task (involving little time pressure and competition), Type As

were less reactive than Type Bs, with Type As in fact exhibiting minimal PTT changes from baseline. Goldband hypothesized that under conditions not sufficiently challenging or "relevant" to the Type A behavior pattern, Type A subjects may actually be hyporesponders. Indeed, the current findings are congruent with this notion and suggest something analogous to an "all-or-none" phenomenon of PTT reactivity in Type As.

The notion of an all-or-none pattern of reactivity is also in line with Glass' assertion that Type As have an enhanced need to control their environments, and that threats to this control elicit increased efforts to control --what Obrist (1976) terms "effortful active coping." The low certainty ITI condition might be conceptualized as an uncontrolable or unpredictable situation for Type As, thus enhancing their efforts to control. The evoked potentials data lend support to this idea, since Type As had an earlier onset of the P295 component during the low certainty ITI condition (possibly suggesting greater involvement in or concentration on the task) as compared to the high certainty condition. The opposite was true for Type Bs -- P295 was earliest during the high certainty ITI condition.

It appears then that phasic PTT (occurring across a 1500-msec period) was quite sensitive to the certainty manipulation -- particularly for the Type A group. As noted in the Introduction, chronic or sustained activation of the

sympathetic nervous system has been implicated as a factor leading to atherosclerosis. It has also been proposed that phasic increases in sympathetic nervous system activity -- of which PTT is a function -- may be involved in endothelial injury via increased hemodynamic turbulence (Ross & Glomset, 1976) and other metabolic processes, and may be linked specifically to the development of clinical manifestations of heart disease, such as myocardial infarction and/or angina pectoris (Herd, 1978; Krantz, Glass, Schaffer, & Davia, 1982). The finding in the present and in a previous study (Goldband, 1980), that under some circumstances Type A persons may be hyporesponsive and that appropriately challenging conditions (e.g., uncertainty) "trigger" cardiovascular reactivity, is quite interesting in light of speculation on the causal role of abrupt shifts between vagal and sympathetic activity in cardiovascular disorders, including sudden death (Engel, 1970; Richter, 1957).

It is apparent from the results on ITI Length as well as those in ITI Certainty that there was a clear lack of uniformity in the level of influence on the autonomic indices exerted by the manipulations. That is, significant effects were found on only some dependent measures, particularly PTT. This lack of uniformity possibly suggests that the tasks did not evoke a singular sympathetic nervous system

response, but rather evoked mixed parasympathetic and sympathetic excitation. As noted, the value of PTT rests in its sensitivity to sympathetic influences, even in light of vagal excitation. Heart rate, on the other hand, is more susceptible to vagal influences. In fact, it has been demonstrated that even mild or moderate levels of parasympathetic excitation can predominate over sympathetic influences on the heart, and can actually block the effects of even high levels of sympathetic stimulation (Levy & Zieske, 1969). Indeed, in the most comprehensive study to date on physiological reactivity in Type As, Williams et al. (1982) found no significant A/B differences during a mental arithmetic task on measures of heart rate or systolic and diastolic blood pressure. Significant A/B differences were found, however, on norepinephrine, epinephrine, and cortisol, as well as on forearm blood flow and forearm vascular resistance. Thus, heart rate as well as blood pressure may be relatively insensitive to the presence of sympathetic stimulation, and could lead to inappropriate conclusions. Therefore, the presence of significant changes in PTT in the absence of heart rate or blood pressure changes in the present study is not viewed as arguing against sympathetic stimulation, but as demonstrating complex autonomic nervous system interactions.

It is interesting that although significant sympathetic excitation was shown to occur under some conditions (e.g., uncertainty), no significant changes in subjects' self-rating of tension were evident. This finding suggests that situations which might produce potentially pathogenic physiological arousal in Type A persons are not necessarily those which may be perceived as "stressors." In addition, it also indicates that very subtle environmental conditions can lead to potentially dangerous physiological adjustments. One clinical implication of this is that if there is little correspondence between physiological reactivity and aversive subjective experiences, there may be little motivation for individuals to change. In fact, some individuals, the high level of autonomic arousal which might be elicited by things such as video games (Glass et al., 1980) may even be pleasurable. In other words, Type As may enjoy being Type A and enjoy their tendency toward heightened autonomic reactivity. Indeed, anecdotal observations suggest that healthy Type A subjects generally resist lifestyle change (e.g., reduced work load), while post-heart-attack As are much more malleable.

Combined Effects of ITI Certainty and Length

As noted, the evaluation of the effects of multi-dimensional situational demands might yield differences between Type As and Type Bs beyond those elicited by one variable singly. The present data from N201 supports

this idea. Maximal A-B differences on N201 were found when the ITI was uncertain and relatively long. This combination of challenges, while evoking trends toward greater autonomic arousal in Type As, did not, however, lead to significant changes in autonomic reactivity.

One possible explanation for this is related to how subjects were selected. Matthews (1982) has noted that Type As assessed by the Jenkins Activity Survey exhibit different behavioral characteristics than SI-assessed Type As. The latter group seems to be characterized by a tendency toward heightened behavioral reactivity under challenging circumstances, while JAS-assessed Type As are characterized more as vigorous achievement strivers, who may be aggressive and competitive (Matthews, 1982). Also, in studies in which no A-B differences were found in cardiovascular reactivity, subjects tended to be selected based on JAS scores. In some of these studies (e.g., MacDougall et al., 1981), when subjects were reclassified using the SI, significant A-B differences were found (with As being more reactive). In contrast, when SI-assessed subjects were reclassified using the JAS, significant cardiovascular differences between As and Bs were diminished (e.g., Blumenthal et al., 1983). This is in some respects not surprising, since Type A scores on the SI were more predictive of incidence of coronary heart disease in the Western Collaborative Group Study (Brand, Rosenman, Jenkins, Sholtz, and Zyzanski, Note 5).

It might therefore be assumed that the differences in reactivity that were discovered in the present study were obtained using an assessment procedure less likely to produce A-B differences.

A unique feature of the present study was the use of ERPs as additional measures of physiological reactivity. Since these measures have not previously been used in the Type A research area, further discussion of their significance seems warranted. As discussed in the Introduction, ERP components might be useful in the study of information processing differences in Type As and Type Bs. Of the four peaks measured, two in particular seemed most responsive to the experimental manipulations and differentiated between As and Bs: N201 and P295. A third component, P436, also differentiated between Type As and Type Bs but was not as sensitive to the experimental manipulations. The N201 component of the present study appears similar to the $\overline{\text{N200}}$ wave reported by other investigators (e.g., Ritter, et al., 1979; Simson, Vaugh & Ritter, 1977) in both its mean latency and response to improbable task-relevant stimuli. The P295 wave resembles the $\overline{\text{P300}}$ component (see Duncan-Johnson, 1981) in its larger amplitude in response to target as opposed to nontarget stimuli, its earlier latency when the frequency of target stimulus occurrence was greater (as in the 6-sec ITI condition), and its onset immediately following what is thought to be $\overline{\text{N200}}$.

There exists some consensus that $\overline{N200}$ reflects initial target selection (Harter, Aine, & Schroeder, 1982; Ritter et al., 1979). The $\overline{P300}$ wave, on the other hand, may be indicative of further, possibly more complex processing. While the exact psychological "meaning" of ERP components is unclear at this time, they might be usefully viewed, as Callaway (1975) had noted, "as a history of the response to a stimulus". That is, the ERPs measured in this experiment represent sequential information processing, with earlier peaks indicating neural processes which occur before subsequent peaks. The present study suggests, then, that Type A and Type B subjects may be differentiated on the basis of the time course of information processing. Stimulus events appear to evoke generally earlier neural responses (N201) in Type As, although task demands appear to modulate the latency of this wave in both groups. "Middle" processing (P295) seems to be more a function of subject Type interacting with specific task demands (e.g., ITI Certainty). That is, task demands seem to determine whether Type As or Type Bs will exhibit the shorter latency. Later processing (P436) seems to occur earlier for Type B subjects regardless of task demands.

While the time course of information processing was shown to be different in As as compared to Bs, it is clear that this difference is very complex. It is not the case that

Type As simply exhibit faster processing, but that whether As or Bs process information faster depends on the nature of the task. Therefore, rather than representing markers for fixed biological or genetic differences between Type As and Type Bs, these ERP peaks might best be viewed as possible indicators of task-induced cognitive changes. Cognitive psychophysiologists such as Donchin, Ritter, and McCallum (1978) view certain "late" peaks (occurring after 200 msec), such as those measured in this study, as being associated with a subjects' "intentions and decisions, modulated by task parameters and experimental instructions" (p. 355). Of course, this does not preclude the possibility that in fact there may be neurological differences between Type As and Type Bs which might be evidenced in shorter or longer cortical responses occurring prior to 200 msec, such as the $\overline{N100}$ or $\overline{P200}$ waves, which are thought by some researchers to be less sensitive to psychological factors (Donchin et al., 1978). Indeed, a recent finding by Krantz, Arabian, Davi , and Parker (1982) that Type A individuals exhibit greater systolic blood pressure reactivity and more complications (arrhythmias) while under general anesthesia during coronary artery bypass surgery, led the authors to suggest that there may in fact be constitutional differences between Type As and Type Bs, of which their overt behavioral characteristics are merely an index. Evoked potentials (especially early

components) may serve to provide converging evidence as to the validity of this idea.

Ultimately, the value of ERP research in this area rests in its ability to enhance our understanding of Type A and Type B individuals. While ERP data, when combined with autonomic and/or behavioral data, may provide converging support for particular hypotheses, they might also impart information beyond that obtained with the more typically used measures. Indeed, although data from the present study show nonsignificant reaction time differences between groups, several of the ERP components (N201, P295, and P436) showed clear A-B differences. Therefore, the incorporation of ERPs may provide for a more complete appraisal of the effects of task demands which may otherwise be obscured.

In summary, the current findings support and extend the conceptualization that persons exhibiting the Type A behavior pattern evidence increased sympathetic arousal under specific environmental conditions. The present study increases our understanding of the situations which might facilitate increased physiological arousal in Type As. The findings suggest that circumstances which are characterized by a low degree of certainty may elicit in Type As enhanced sympathetically mediated phasic changes as well as augmented cortical arousal. While both short and long waiting intervals lead to greater tonic heart rate reactivity in As relative to Bs, the longer waiting period produced the greatest A/B

differences. In addition, the findings are suggestive of a pattern of autonomic enhancement in Type As when low certainty and extended waiting are combined. The present study is the first to demonstrate that physiologic reactivity in Type As is not limited to autonomic nervous system function, but is exhibited as well in cortical activity as measured by event-related brain potentials. The latency of the N201 and P295 peaks in many respects paralleled the autonomic measures in terms of the conditions which elicited the greatest reactivity in both Type A and Type B subjects. Event-related brain potentials may prove to be useful in the study of both physiological reactivity and information processing in Type As and Type Bs as a function of environmental demands.

Reference Notes

1. Williams, R., Lane, J., White, A., Kuhn, C., Schonberg, S., & Clavert, R. Type A behavior pattern and cardiovascular response during mental work. Dept. of Psychiatry, Duke University Medical Center. 1978.

2. Manuck, S., & Kaplan, J. Behaviorally-induced heart rate reactivity and coronary artery atherosclerosis in monkeys. Paper presented at the 21st annual meeting of the Society for Psychophysiological Research, Washington, D.C., 1981.

3. Smith, T.W., & Anderson, N.B. An interactional model of Type A behavior and physiological activity. Manuscript in preparation. Brown University Program in Medicine, Providence, R.I.

4. Weiss, T. Noninvasive measures of sympathetic myocardial influence. Paper presented at the 21st annual meeting of the Society for Psychophysiological Research, Washington, D.C., 1981.

5. Brand, R., Rosenman, K., Jenkins, L., Sholtz, R., & Zyzanski, S. Comparison of coronary heart disease prediction in the Western Collaborative Group Study using the Structured Interview and the Jenkins Activity Survey assessment of the coronary-prone Type A behavior pattern. Paper presented at the conference on cardiovascular disease epidemiology of the American Heart Association, Orlando, March, 1978.

6. Allen, M.I., & Lawler, K.A. Physiological responses of Type A and Type B college students to two conditions of inactivity. Paper presented at the 21st annual meeting of the Society for Psychophysiological Research, Washington, D.C., 1981.

7. Neilson, W., Neufeld, R.W., & Goldband, A. Controllability, cardiovascular responsivity and the Type A behavior pattern. Paper presented at the 22nd annual meeting of the Society for Psychophysiological Research, Minneapolis, MN, 1982.

BIBLIOGRAPHY

Andreassi, J.L. Psychophysiology: Human behavior and physiological response. New York: Oxford Press, 1980.

Ardlie, G., Glen, G, & Schwartz, C.J. Influence of catecholamines on nucleotide induced platelet aggregation. Nature, 1966, 212, 415-417.

Arteriosclerosis 1981. Report of the working group on arteriosclerosis of the National Heart, Lung and Blood Institute, 1981, 1, 3.

Benjamin, L. Facts and artifacts in using analysis of covariance to "undo" the law of initial values. Psychophysiology, 1967, 4, 187-206.

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

Blumenthal, J., Williams, R., Kong, Y., Schanberg, S., & Thompson, L. Type A behavior and angiographically documented coronary disease. Circulation, 1978, 58, 634-639.

Blumenthal, J., Williams, R., Kong, Yl, Thompson, L., Jenkinds, C.D., & Rosenman, R.H. Type A behavior and angiographically documented coronary disease. Psychosomatic Medicine, 1975, 37 (abs.).

Brookhuis, K., Mulder, G., Mulder, L., Gloerich, A., Van Dellen, H., Van Der Meese, J., & Ellermann, H. Late positive components and stimulus evaluation time. Biological Psychology, 1981, 13, 107-123.

Brunson, B., & Matthews, K. The Type A coronary-prone behavior pattern and reactions to uncontrollable events: An analysis of learned helplessness. Journal of Personality and Social Psychology, 1981, 40, 906-918.

Bunam, B.I., & Matthews, K.A. The Type A coronary-prone behavior pattern and reaction to uncontrollable events: An analysis of learned helplessness. Journal of Consulting and Clinical Psychology, 1981, 40, 906-918.

Bunnell, D.E. Autonomic myocardial influences as a factor determining inter-task consistency of heart rate reactivity. Psychophysiology, 1982, 19, 492-498.

Burnam, M., Pennebaker, J., & Glass, D.C. Time consciousness, achievement striving, and the Type A coronary-prone behavior pattern. Journal of Abnormal Psychology, 1975, 84, 76-79.

Caffrey, B. Reliability and validity of personality and behavioral measures in a study of coronary heart disease. Journal of Chronic Disease, 1968, 21, 191-209.

Callaway, E. Brain electrical potential and individual psychological differences. New York: Grune & Stratton, 1975.

Chesney, M., Black, G., Chadwick, J., & Rosenman, R. Psychological correlates of the coronary-prone behavior pattern. Journal of Behavioral Medicine, 1981, 4, 217-230.

Contrada, R., Glass, D., Krakoff, L., Krantz, D., Kehoe, K., Isecke, W., Collins, L., & Elting, E. Effects of control over aversive stimulation and Type A behavior on cardiovascular and plasma catecholamine responses. Psychophysiology, 1982, 19, 408-419.

Dembroski, T., Caffrey, B., Jenkins, C.D., Rosenman, R.H., Spielberger, C., & Tasto, D. Section summary: Assessment of coronary-prone behavior. In Dembroski, Weiss, Shields, Haynes, & Feinleib (Eds.,), Coronary-prone behavior. New York: Springer-Verlag, 1978.

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

Dembroski, T., MacDougall, J. & Lushene, R. Interpersonal interaction and cardiovascular response in Type A subjects and coronary patients. Journal of Human Stress, 1977, 3, 2-9.

Dembroski, T., MacDougall, J., & Shields, J. Physiologic reactions to social challenge in persons evidencing the Type A coronary-prone behavior pattern. Journal of Human Stress, 1977, 3, 2-10.

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

Diamond, E., & Carver, C. Sensory processing, cardiovascular reactivity, and the Type A coronary-prone behavior pattern. Biological Psychology, 1980, 10, 265-275.

Donchin, E. Surprise! ... Surprise? Psychophysiology, 1981, 18, 493-513.

Donchin, E., & Cohen, L. Averaged evoked potentials and intramodality-specific selective attention. Electroencephalography and Clinical Neurophysiology, 1967, 22, 537-546.

Donchin, M., Ritter, W., & McCallum, W. C. Cognitive psychophysiology: The endogenous components of the ERP. In E. Calloway, P. Tuiting, & S. Kaslow (Eds.), Event-related brain potential in man. New York: Academic Press, 1978.

Duncan-Johnson, C. C. The P300 component of the critical event-related potential as an index of subjective probability and processing duration (Doctoral dissertation, University of Illinois, 1978). Dissertation Abstracts International, 1978, 39, 6098B-6099B (University Microfilms No. 79-13504).

Duncan-Johnson, C. C. P300 latency: A new metric of information processing. Psychophysiology, 1981, 18, 207-215.

Eliot, R., Clayton, F., Puper, G., & Todd, G. Influence of environmental stress on pathogenesis of sudden cardiac death. Proceedings for the Federation of Experimental Biological Sciences, 1977, 36, 1719-1724.

Engel, G. L. Sudden death and the 'medical model' in psychiatry. Canadian Psychiatric Association Journal, 1970, 15, 527-538.

Feldman, M., & Drasgnio, J. The visual-verbal test. Los Angeles, CA: Western Psychological Services, 1959.

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

Frankenhaeuser, M. Behavior and circulating catecholamines. Brain Research, 1971, 31, 241-262.

Frankenhaeuser, M., Lundberg, V., & Forsman, L. 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, 1980, 10, 79-81.

Friedman, D., Vaughan, Jr., H., & Erlenmeyer-Kimling, R. Stimulus and response related components of the late positive complex in visual discrimination tasks. Electroencephalography and Clinical Neurophysiology, 1978, 45, 319-330.

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

Friedman, M., Byers, S., Rosenman, R., & Elevitch, J. Coronary-prone individuals (Type A behavior pattern): Some biochemical characteristics. Journal of the American Medical Association, 1970, 212, 1030-1037.

Friedman, M., St. George, S., Byers, S., & Rosenman, R. Excretion of catecholamines, 17-ketosteroids, 17-hydroxycortinoids, and 5-hydroxyindole in men exhibiting a particular behavior pattern (A) associated with high incidence of clinical coronary disease. Journal of Clinical Investigations, 1960, 39, 758-764.

Friedman, M., & Rosenman, R. Association of specific overt behavior pattern with blood and cardiovascular findings. Journal of the American Medical Association, 1959, 169, 1286-1296.

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

Friedman, M., Rosenman, R., Straus, R., Wurm, M., & Kositchek, R. The relationship of behavior pattern A to the state of the coronary vasculature: A study of fifty-one autopsy subjects. American Journal of Medicine, 1968, 44, 525-537.

Gastorf, J. Time urgency and the Type A behavior pattern. Journal of Consulting and Clinical Psychology, 1980, 48, 299.

Glass, D. Behavior patterns, stress and coronary disease. Hillsdale, N.J.: Lawrence Erlbaum Associates, 1977.

Glass, D.C. Pattern A behavior and uncontrollable stress. In Dembroski, Weiss, Sheilds, Haynes, & Feinleib (Eds.), Coronary-prone behavior. New York: Springer-Verlag, 1978.

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

Glass, D., Snyder, M., & Hollis, J. Time urgency and the Type A coronary-prone behavior pattern. Journal of Applied Social Psychology, 1974, 4, 125-140.

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

Goldband, S., Katkin, E.S., & Morell, M.A. Personality and cardiovascular disorder: Steps toward demystification. In C.D. Spielberger & I.G. Sarason (Eds.), Stress and anxiety. Washington, D.C.: Hemisphere, 1979.

Haft, J. Cardiovascular injury induced by sympathetic catecholamines. Progress in Cardiovascular Disorders, 1974, 17, 73-86.

Haft, J., Kranz, P., Albert, F., et al. Intravascular platelet aggregation in the heart induced by norepinephrine. Circulation, 1976, 46, 698-708.

Haft, J., Fani, K., Alcorta, C., et al. Effect of propanolol on stress-induced intravascular platelet aggregation in the heart. Circulation, 1973, 48 (Suppl. IV), 1973.

Harter, M.R., Aine, C., & Schroeder, C. Hemispheric differences in neural processing of stimulus location and type: Effects of selective attention on visual evoked potentials. Neuropsychologia, 1982, 20, 421-438.

Harter, M.R., & Guido, W. Attention to pattern orientation: Negative cortical potentials, reaction time, and the selection process. Electroencephalography and Clinical Neurophysiology, 1980, 49, 461-475.

Heller, R. Type A behavior in coronary heart disease. British Medical Journal, 1979, 2, 368.

Herd, J.A. Physiological correlates of coronary-prone behavior. In Dembroski, Weiss, Shields, Haynes, & Feinleib (Eds.), Coronary-prone behavior. New York: Springer-Verlag, 1978.

Howard, J., Cunningham, D., & Rechnitzer, P. Health patterns associated with Type A behavior: A managerial population. Journal of Human Stress, 1976, 2, 24-31.

Howard, J., Cunningham, D., & Rechnitzer, P. Work patterns associated with Type A behavior: A managerial population. Human Relations, 1977, 30, 825-836.

Hueper, W.C. Arteriosclerosis. Archives of Pathology, 1944, 38, 245-285.

Jenkins, C.D. Psychologic and social precursors of coronary disease. New England Journal of Medicine, 1971, 284, 244-255, 307-317.

Jenkins, C.D. The coronary-prone personality. In W.D. Gentry & R.B. Williams (Eds.), Psychological aspects of myocardial infarction and coronary care. St. Louis: C.V. Mosby Co., 1975.

Jenkins, C.D., Rosenman, R., & Zyzanski, S. Prediction of coronary heart disease by a test for the coronary-prone behavior pattern. New England Journal of Medicine, 1974, 290, 1271-1275.

Jenkins, C.D., Zyzanski, S., Rosenman, R., & Cleveland, G. Association of coronary-prone behavior scores with recurrence of coronary heart disease. Journal of Chronic Diseases, 1971, 24, 601-611.

Jennings, J.R., & Choi, S. Type A components and psychophysiological responses to an attention-demanding performance task. Psychosomatic Medicine, 1981, 43, 475-487.

Johnson, R., Jr., Pfefferbaum, A., & Kopell, B.S. P300 and reaction time in a long-term memory task: A dissociation. Psychophysiology, 1981, 18, 144-145 (abstract).

Kahn, J.P., Koenfeld, D., Frank, K., Heller, S., & Hoar, P. Type A behavior and blood pressure during coronary artery bypass surgery. Psychosomatic Medicine, 1980, 42, 407-414.

Keith, R., Lown, B., & Stor, F. Coronary heart disease and behavior patterns: An examination of method. Psychosomatic Medicine, 1965, 27, 424-434.

Kenigsberg, S., Zyzanski, S., Jenkins, C.D., Wardwell, W., & Licciardello, A.T. The coronary-prone behavior pattern in hospitalized patients with and without coronary heart disease. Psychosomatic Medicine, 1974, 36, 344-351.

Kinsman, R., & Staudenmayer, H. Baseline levels in muscle relaxation training. Biofeedback and Self-Regulation, 1978, 3, 97-104.

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

Krantz, D.S., Glass, D.C., Schaeffer, M.A., & Davia, J.E. Behavior patterns and coronary disease: A critical evaluation. In J.T. Cacioppo & R.E. Petty (Eds.), Focus on cardiovascular psychophysiology. New York: Guilford Press, 1982.

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

Kutas, M., McCarthy, G., & Donchin, E. Augmenting mental chronometry: The P300 as a measure of stimulus evaluation time. Science, 1977, 197, 792-795.

Lacey, J.I., & Lacey, B.C. Some autonomic-central nervous system interrelationships. In P. Block (Ed.), Physiological correlates of emotion. New York: Academic Press, 1970.

Lacey, J.I., & Lacey, B.C. On heart rate responses and behavior: A reply to Elliott. Journal of Personality and Social Psychology, 1974, 30, 1-18.

Levy, M. N., & Zieske, H. Autonomic control of cardiac pacemaker activity and atreoventricular transmission. Journal of Applied Physiology, 1969, 27, 465-470.

Light, K.C. Cardiovascular responses to effortful active coping: Implications for the role of stress in hypertension development. Psychophysiology, 1981, 18, 216-225.

Light, K.C., & Obrist, P.A. Cardiovascular response to stress: Effects of opportunity to avoid, shock experience, and performance feedback. Psychophysiology, 1980, 17, 243-252. (a)

Light, K.C., & Obrist, P.A. Cardiovascular reactivity to behavioral stress in young males with and without marginally elevated casual systolic pressures: A comparison of clinic, home, and laboratory measures. Hypertension, 1980, 2, 802-808. (b)

Lott, G., & Gatchel, R. A multi-response analysis of learned heart rate control. Psychophysiology, 1978, 15, 576-581.

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

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

Manuck, S., Craft, S., & Gold, K. Coronary-prone behavior pattern and cardiovascular response. Psychophysiology, 1978, 15. 403-411.

Manuck, S., & Garland, F.M. Coronary-prone behavior pattern, task incentive, and cardiovascular response. Psychophysiology, 1979, 16, 136-142.

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

Miller, B., & Keane, C. Encyclopedia and dictionary of medicine, nursing, and allied health. Philadelphia: W.B. Saunders Co., 1978.

Newlin, D. Relationships of pulse transmission times to pre-ejection period and blood pressure. Psychophysiology, 1981, 18, 316-321.

O'Brien, J. Variability in the aggregation of human platelets by adrenaline. Nature (London), 1963, 200, 763-764.

Obrist, P.A. The cardiovascular-behavioral interaction: As it appears today. Psychophysiology, 1976, 13, 95-107.

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

Obrist, P., Langer, A., Light, K., Grignolo, A., & McCubbin, J. Myocardial performance and stress: Implications for basic and clinical research. In Kimmel, Van Olst, & Orlebele (Eds.), The orienting reflex in humans. Hillsdale, N.J.: Lawrence Erlbaum & Associates, 1979.

Obrist, P., Light, K., McCubbin, J., Hutcheson, J., & Hoffer, J. Pulse transit time: Relationship to blood pressure and myocardial performance. Psychophysiology, 1979, 16, 292-301.

Oscar-Berman, M. Commentary. In E. Callaway, P. Tueting, & S. Koslow (Eds.), Event-related brain potentials in man. New York: Academic Press, 1978.

Osler, W., Lectures on angina pectoris and allied states. New York: D. Appleton & Co., 1897.

Panel on Coronary-Prone Behavior. Bethesda, MD, 1978.

Piston, T., Woods, D., Stuss, D., & Campbell, K. Methodology and meaning of human evoked potential scalp distribution studies. In D.A. Otto (Ed.), Multidisciplinary perspectives in event-related brain potential research. EPA: North Carolina, 515-522.

Raab, W., Stark, E., MacMillian, W., et al. Sympathogenic origin and anti-andrenergic prevention of stress-induced myocardial lesions. American Journal of Cardiology, 1961, 8, 203-211.

Renault, B., & Lesevic, N. A trial by trial study of the visual omission response in reaction time situations. In D. Lehrmann & E. Callaway (Eds.), Human evoked potentials: Applications and problems. New York: Plenum Press, 1979.

Richter, C.P. On the phenomenon of sudden death in animals and man. Psychosomatic Medicine, 1957, 19, 191-198.

Ritter, W., Simson, R., Vaughan, Jr., H.G., & Friedman, D. A brain potential related to the making of a sensory discrimination. Science, 1979, 203, 1358-1361.

Rosenman, R., Brand, R., Jenkins, C.D., Friedman, M., Straus, R., & Wurm, M. Coronary heart disease in the Western Collaborative Group Study: Final follow-up experience of 8½ years. Journal of the American Medical Association, 1975, 233, 872-877.

Rosenman, R., & Friedman, M. Association of specific behavior pattern in women with blood and cardiovascular findings. Circulation, 1961, 24, 1173-1184.

Rosenman, R., & Friedman, M. Neurogenic factors in pathogenesis of coronary heart disease. Medical Clinics of North America, 1974, 58, 269-279.

Rosenman, R., Friedman, M., Straus, R., Wurm, M., Kositchek, R., Hahn, W., & Werthessen, N. A predictive study of coronary heart disease: The Western Collaborative Group Study. Journal of the American Medical Association, 1964, 189, 15-22.

Ross, R., & Glomset, J.A. The pathogenesis of arteriosclerosis. New England Journal of Medicine, 1976, 295, 369-377, 420-425.

Schenk, E.A., & Moss, A.J. Cardiovascular effects of sustained norepinephrine infusions. II. Morphology. Circulation Research, 1966, 18, 605-615.

Scherwitz, L., Leventhal, H., Cleary, P., & Lamon, C. Type A behavior: Consideration for risk modification. Health Values: Achieving High Level Wellness, 1978, 2, 291-296.

Schlegel, R.P., Wellwood, J.K., Copps, B.E., Gruchow, W.H., & Shurratt, M.T. The relationship between perceived challenge and daily symptom reporting in Type A vs. Type B postinfarct subjects. Journal of Behavioral Medicine, 1980, 3, 191-209.

Schucker, B., & Jacobs, D. Assessment of behavioral risk for coronary disease by voice characteristics. Psychosomatic Medicine, 1977, 39, 219-228.

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

Selye, H., & Bajusz, E. Conditioning by corticoids for the production of cardiac lesions with noradrenaline. Acta Endocrinology, 1959, 30, 183-187.

Shekelle, R., Schoenberger, J., & Stamler, J. Correlates of the JAS Type A behavior pattern score. Journal of Chronic Diseases, 1976, 29, 183-187.

Simson, R., Vaughan, Jr., H., & Ritter, W. The scalp topography of potentials associated with missing visual and auditory stimuli. Electroencephalography and Clinical Neurophysiology, 1977, 42, 528-535.

Squires, K.C., Wickens, C., Squires, N.K., & Donchin, E. The effect of stimulus sequence on the waveform of the cortical event-related potential. Science, 1976, 193, 1142-1146.

Stallones, R. The rise and fall of ischemic heart disease. Scientific American, 1980, 243, 53-59.

Steptoe, A. Psychological factors in cardiovascular disorders. New York: Academic Press, 1981.

Steptoe, A., & Ross, A. Psychophysiological reactivity and the prediction of cardiovascular disorders. Journal of Psychosomatic Research, 1981, 25, 23-31.

Steptoe, A., Smulyan, G., & Gribbin, B. Pulse wave velocity and blood pressure change: Calibration and applications. Psychophysiology, 1976, 13, 488-493.

Stern, R., Ray, W., & Davis, C. Psychophysiological recording. New York: Oxford University Press, 1980.

Suinn, R. Type A behavior pattern. In R.B. Williams & W.D. Gentry (Eds.), Behavioral approaches to medical treatment. Cambridge, MA: Ballinger Publishing Co., 1977.

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

Van Egeren, L.F. Social interactions, communications, and the coronary-prone behavior pattern: A psychophysiological study. Psychosomatic Medicine, 1979, 41, 2-18. (b)

Waldron, I. The coronary-prone behavior pattern, blood pressure, employment and socio-economic status in women. Journal of Psychosomatic Research, 1978, 22, 79-87.

Waters, I., & de Suto-Nagy, G. Lesions of the coronary arteries and great vessels of the dog following the injection of adrenalin. Science, 1950, 111, 634-635.

Weiss, T., Del Bo, A., Reichek, N., & Engelman, K. Pulse transit time in the analysis of autonomic nervous system effects on the cardiovascular system. Psychophysiology, 1980, 17, 202-207.

Williams, R.B., Bittker, J.E., Buchsbaum, M.S., & Wynne, L.C. Cardiovascular and neurophysiologic correlates of sensory intake and rejection : 1. Effect of cognitive task. Psychophysiology, 1975, 12, 427-433.

Williams, R., Friedman, M., Glass, D., Herd, J., & Schneiderman, N. Section summary: Mechanisms linking behavioral and pathophysiological processes. In

Dembroski, Weiss, Shields, Haynes, & Feinleib (Eds.), Coronary-prone behavior. New York: Springer-Verlag, 1978.

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

Winer, B.J. Statistical principles in experimental design (2d ed.). New York: McGraw-Hill, 1971.

Ziesler, K., Maseii, A., Klassen, D., Rabinowitz, D., & Burgess, J. Muscle metabolism during exercise in man. Transcontinental Association of American Physicians, 1968, 81, 266-273.

Zyzanski, S., Jenkins, C., Ragan, T., Flessas, A., & Everist, M. Psychological correlates of coronary angiographic findings. Archives of Internal Medicine, 1976, 136, 1234-1237.

Zyzanski, S., Wryesniewski, K., & Jenkins, C.D. Cross-cultural validation of the coronary-prone behavior pattern. Social Science and Medicine, 1979, 13, 405-412.

APPENDIX A

JAS SCALE

APPENDIX B

FIGURES AND TABLES

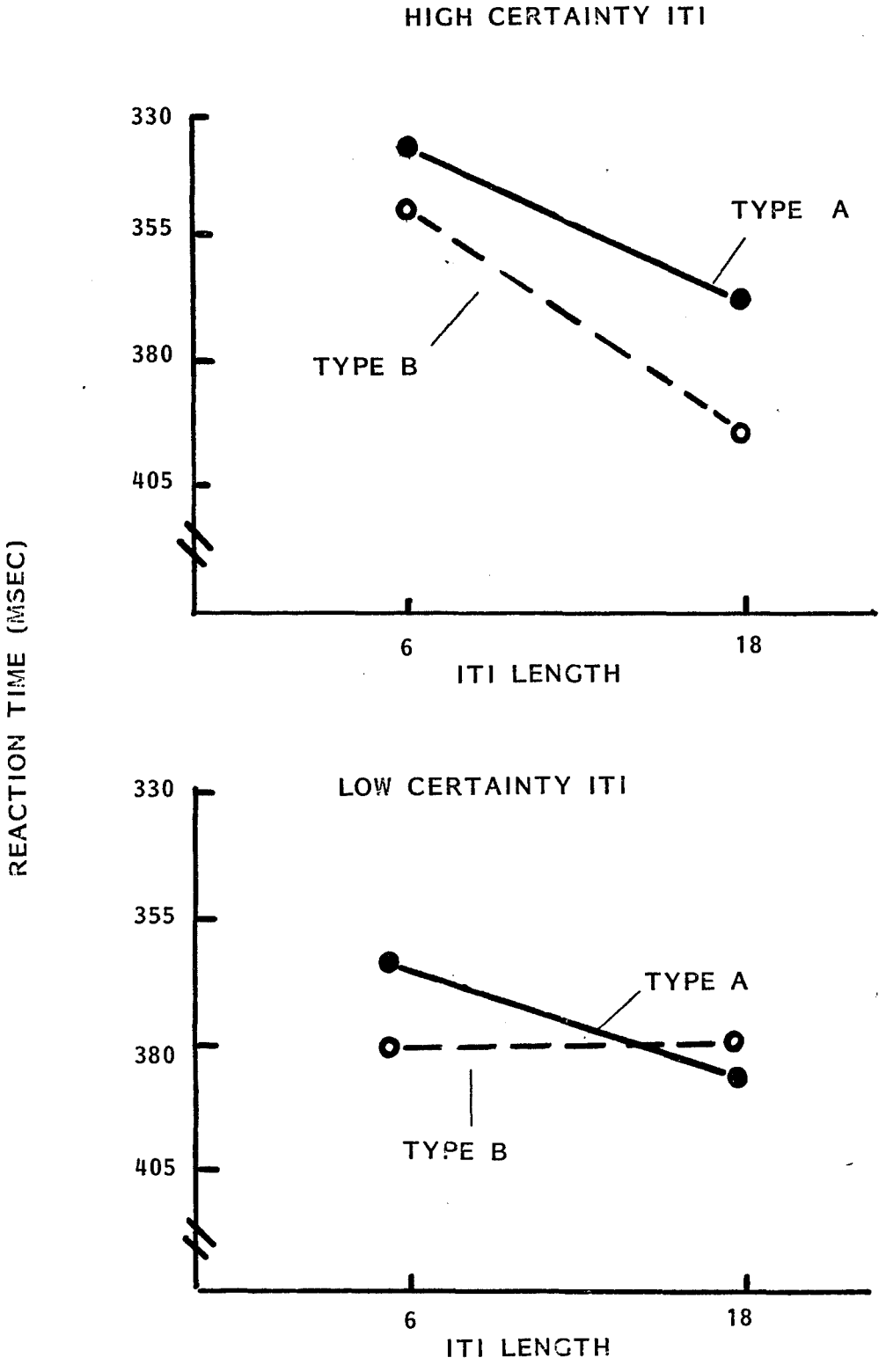

Figure 5. Reaction times for Type A and Type B subjects during the high certainty (top graph) and low certainty (bottom graph) conditions with either 6- or 18-sec ITIs.

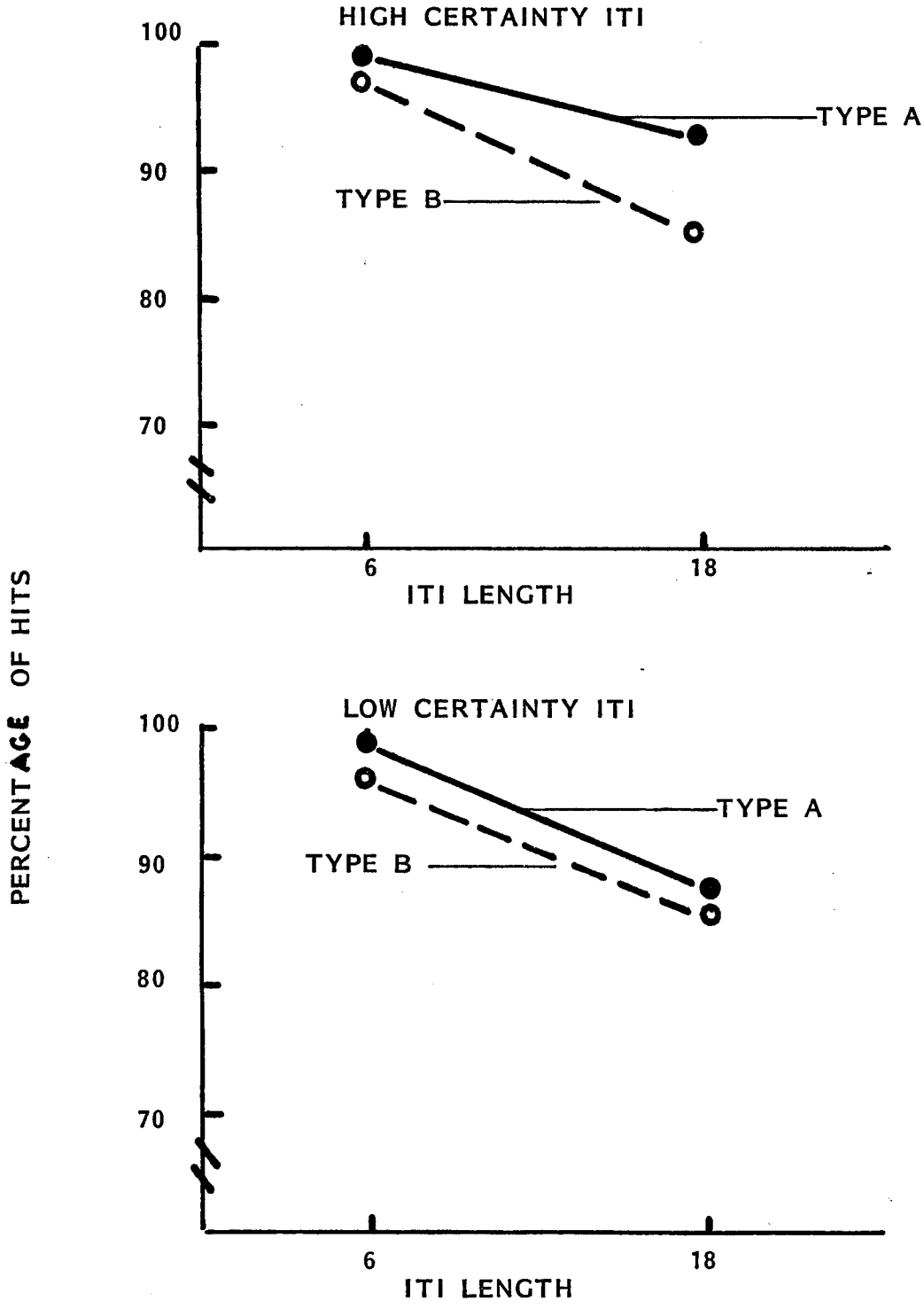

Figure 6. Percentage of hits for Type A and Type B subjects during the high certainty (top) and low certainty (bottom) conditions with either 6- or 18-sec ITIs.

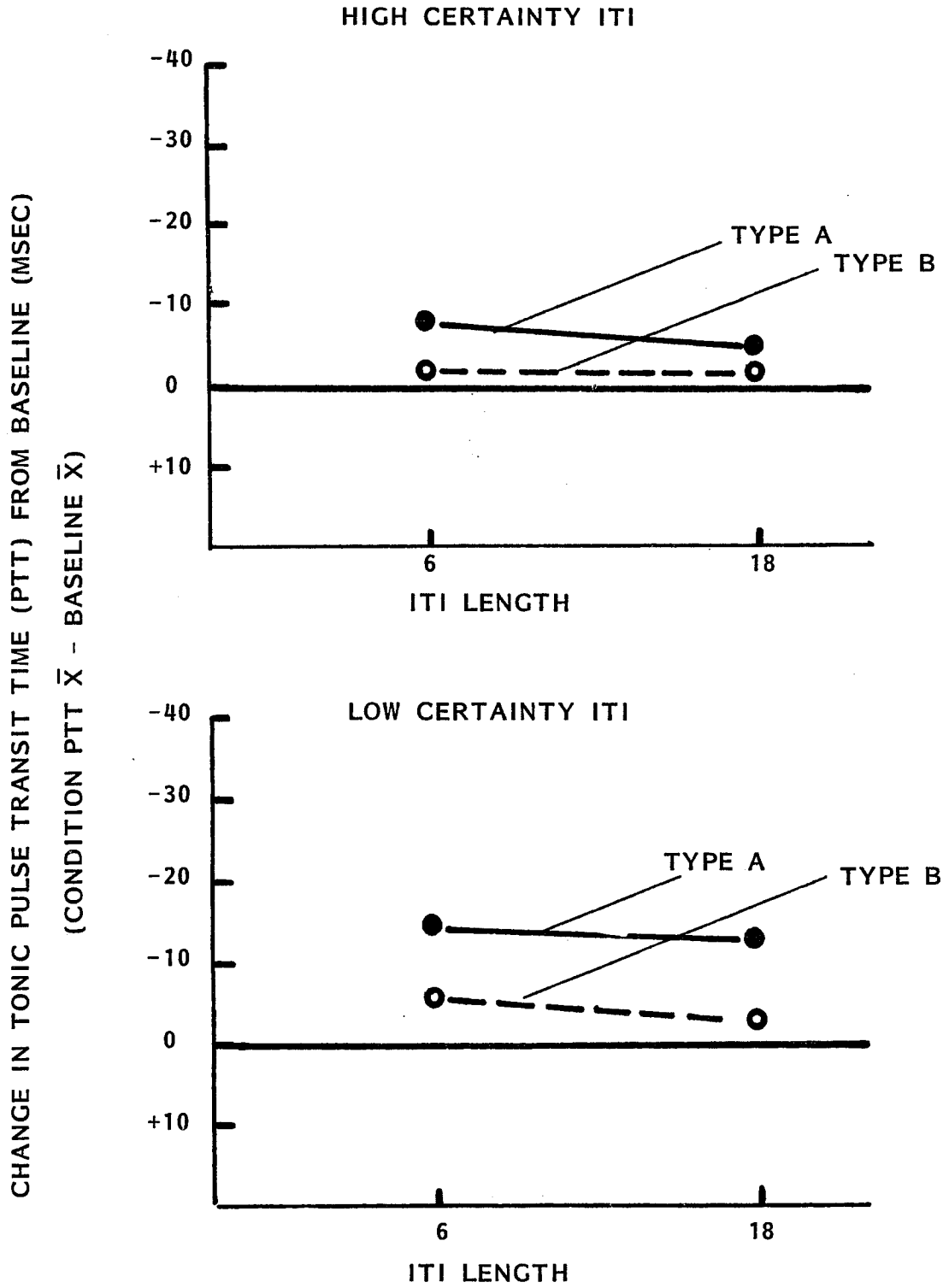

Figure 7. Change in tonic pulse transit time (Condition $\bar{X}$- Baseline $\bar{X}$) from baseline for Type A and Type B subjects during the high certainty (top graph) and low certainty (bottom graph) conditions with 6- and 18-sec ITIs.

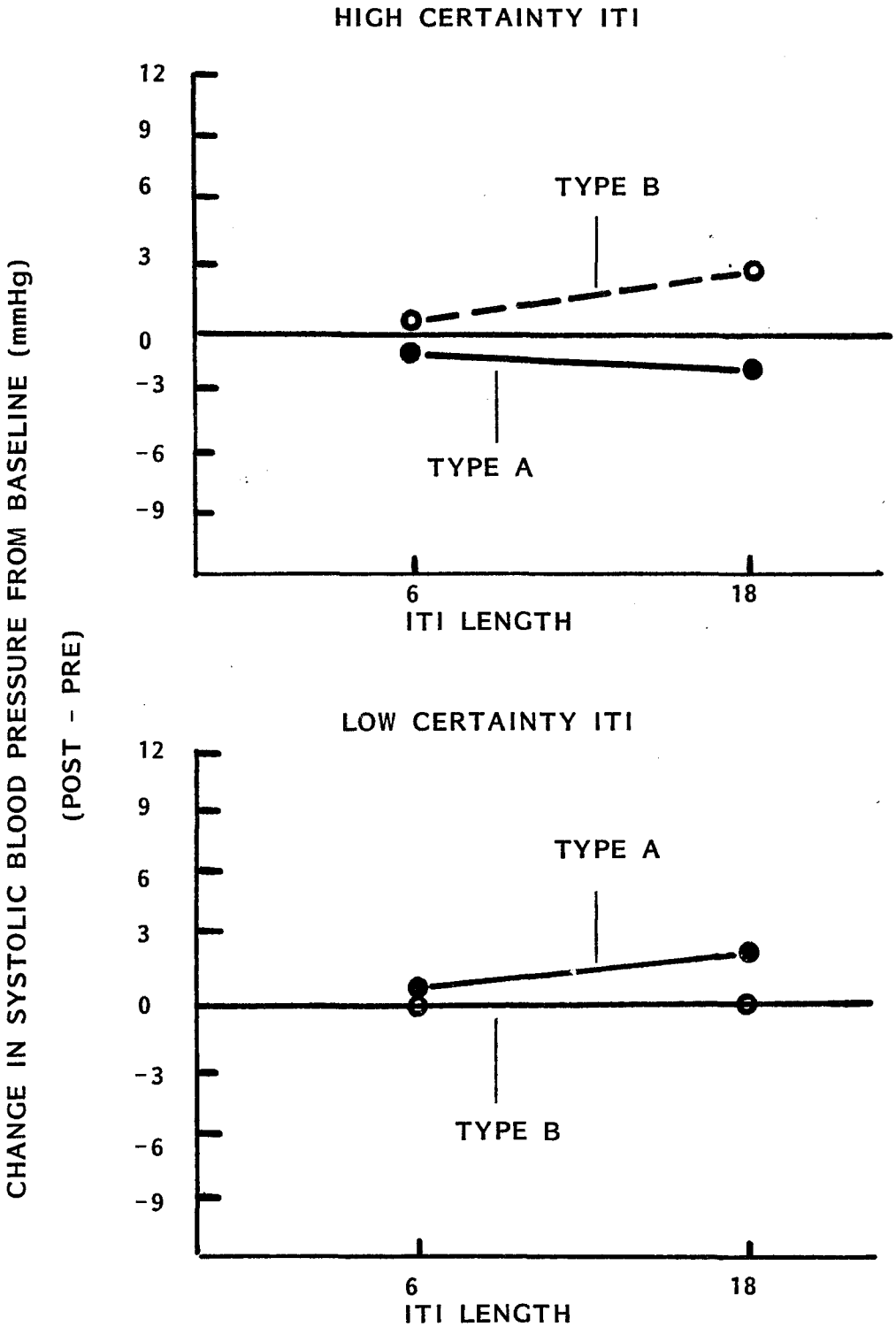

Figure 8. Change in systolic blood pressure (post minus pre) in Type A and Type B subjects during the high certainty (top graph) and low certainty (bottom graph) conditions with 6- and 18-sec ITIs.

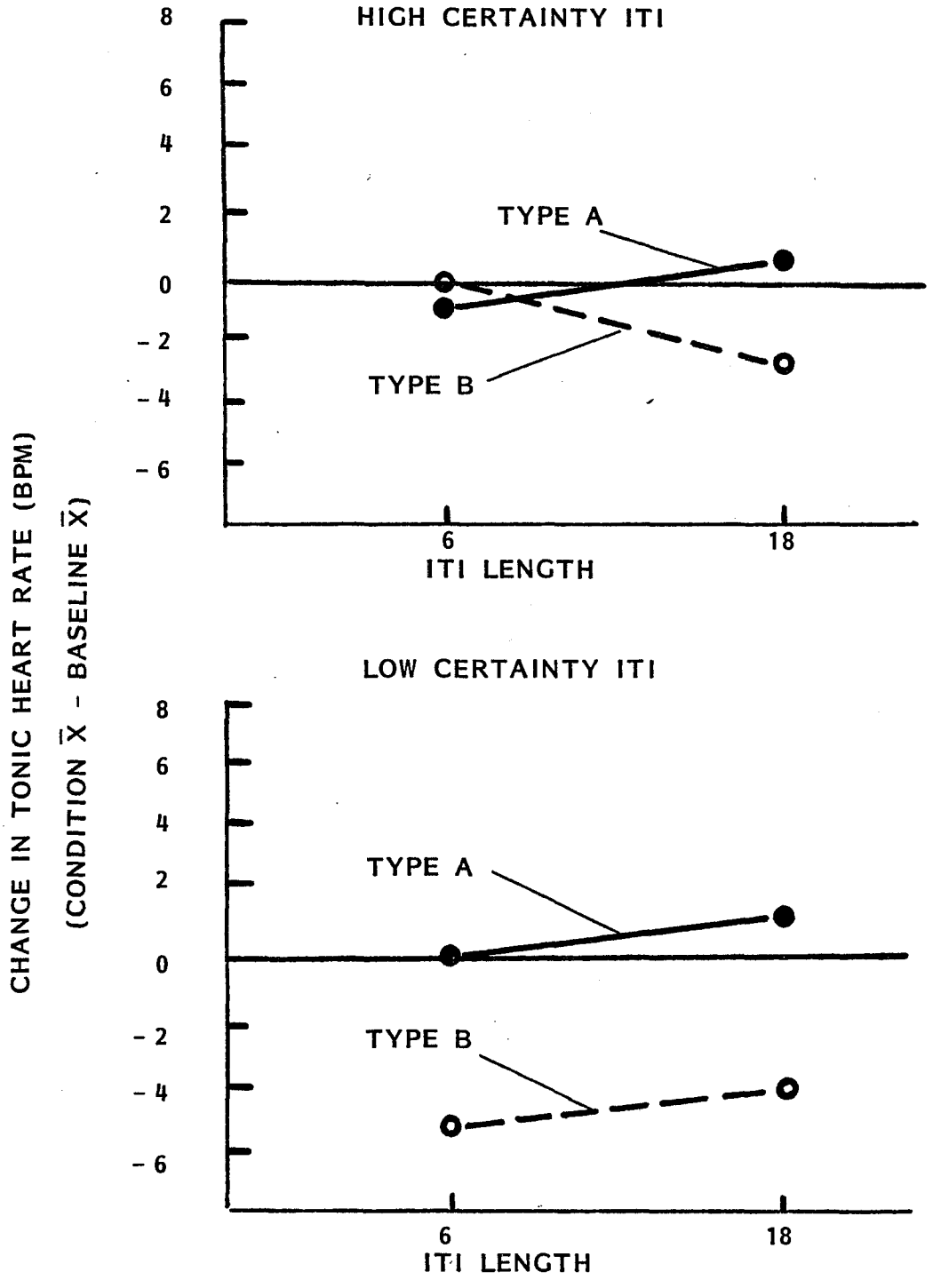

Figure 9. Change in tonic heart rate from baseline (Condition $\bar{X}$-Baseline $\bar{X}$) in Type A and Type B subjects during the high certainty (top graph) and low certainty (bottom graph) conditions with 6- and 18-sec ITIs.

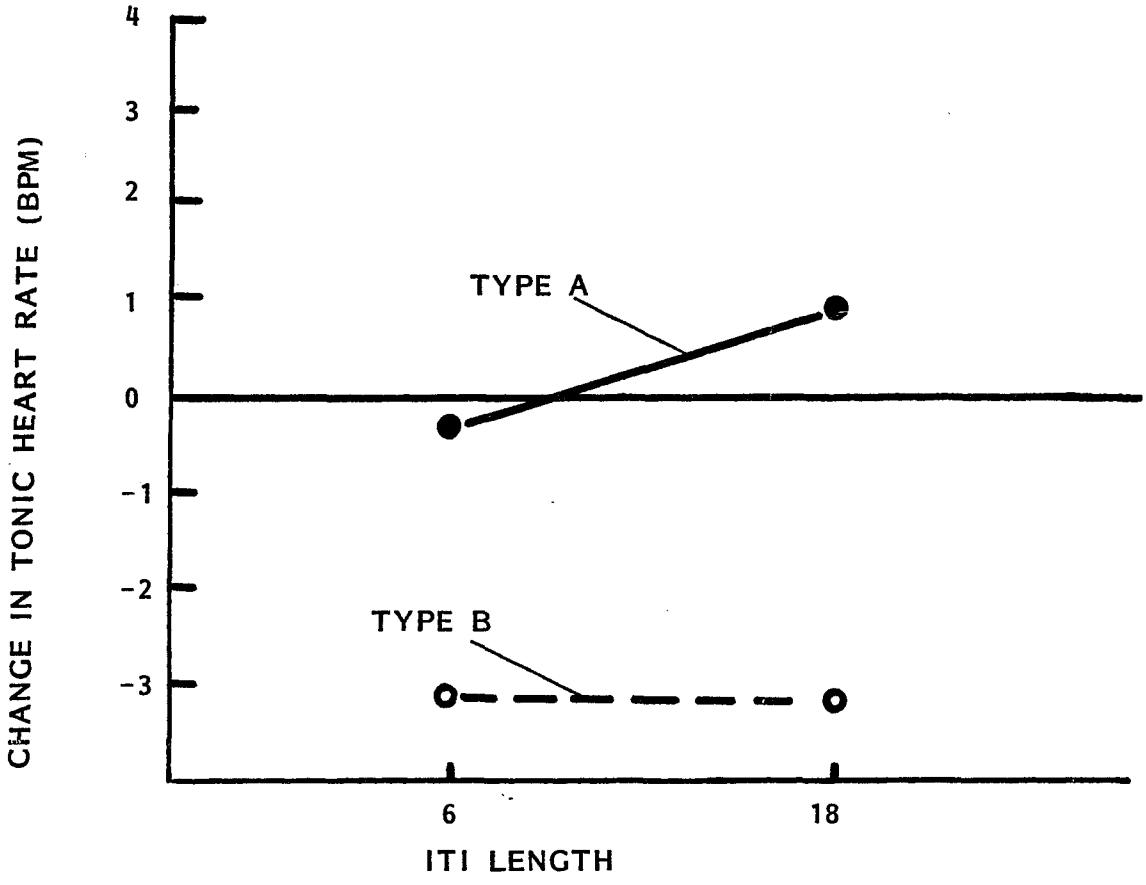

Figure 10. Change in tonic heart rate for Type A and Type B subjects during ITI 6-sec and 18-sec conditions.

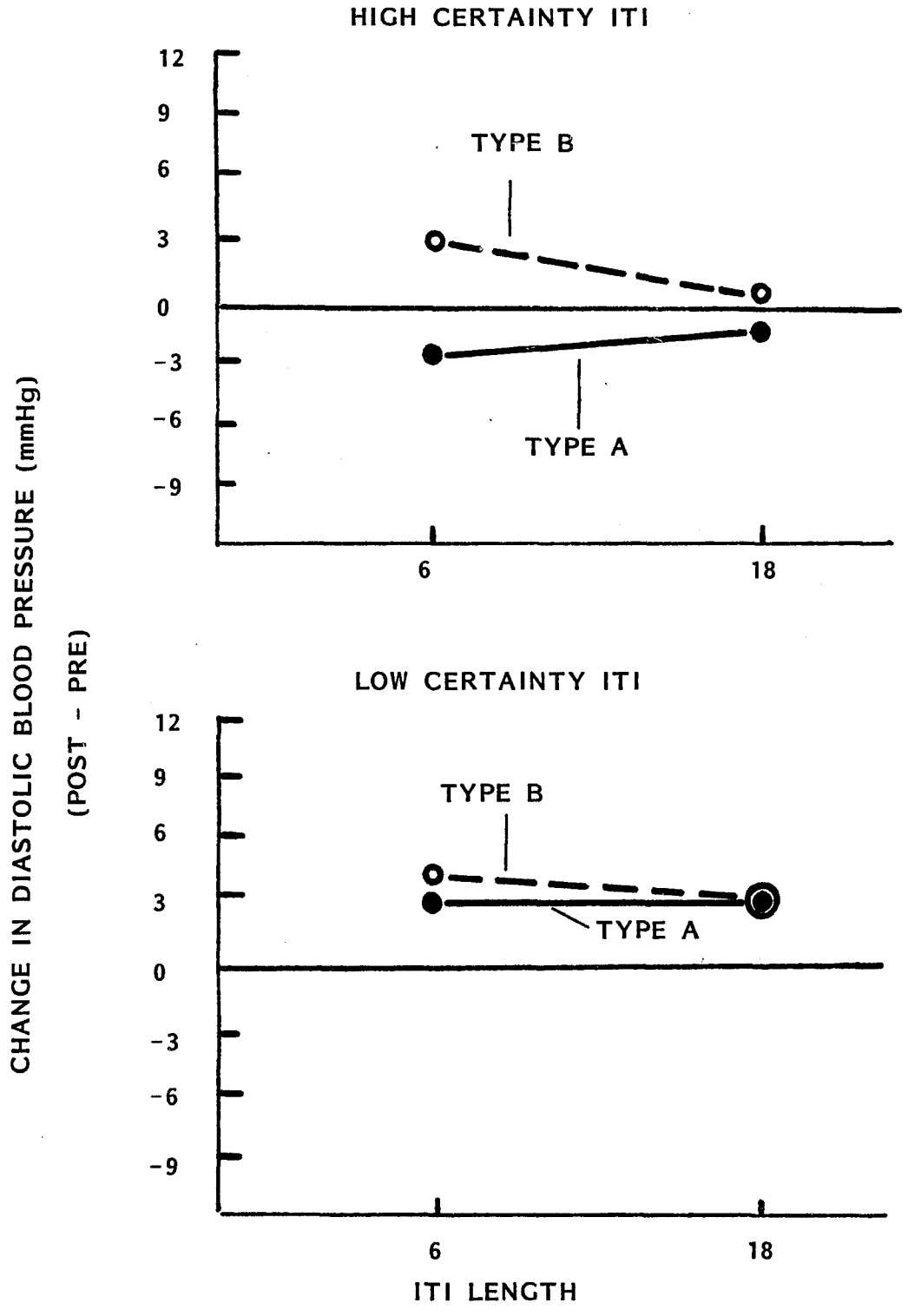

Figure 11. Change in diastolic blood pressure (post-pre) line (post-pre) for Type A and Type B subjects during the high certainty (top graph) and low certainty (bottom graph) conditions with 6- and 18-sec ITIs.

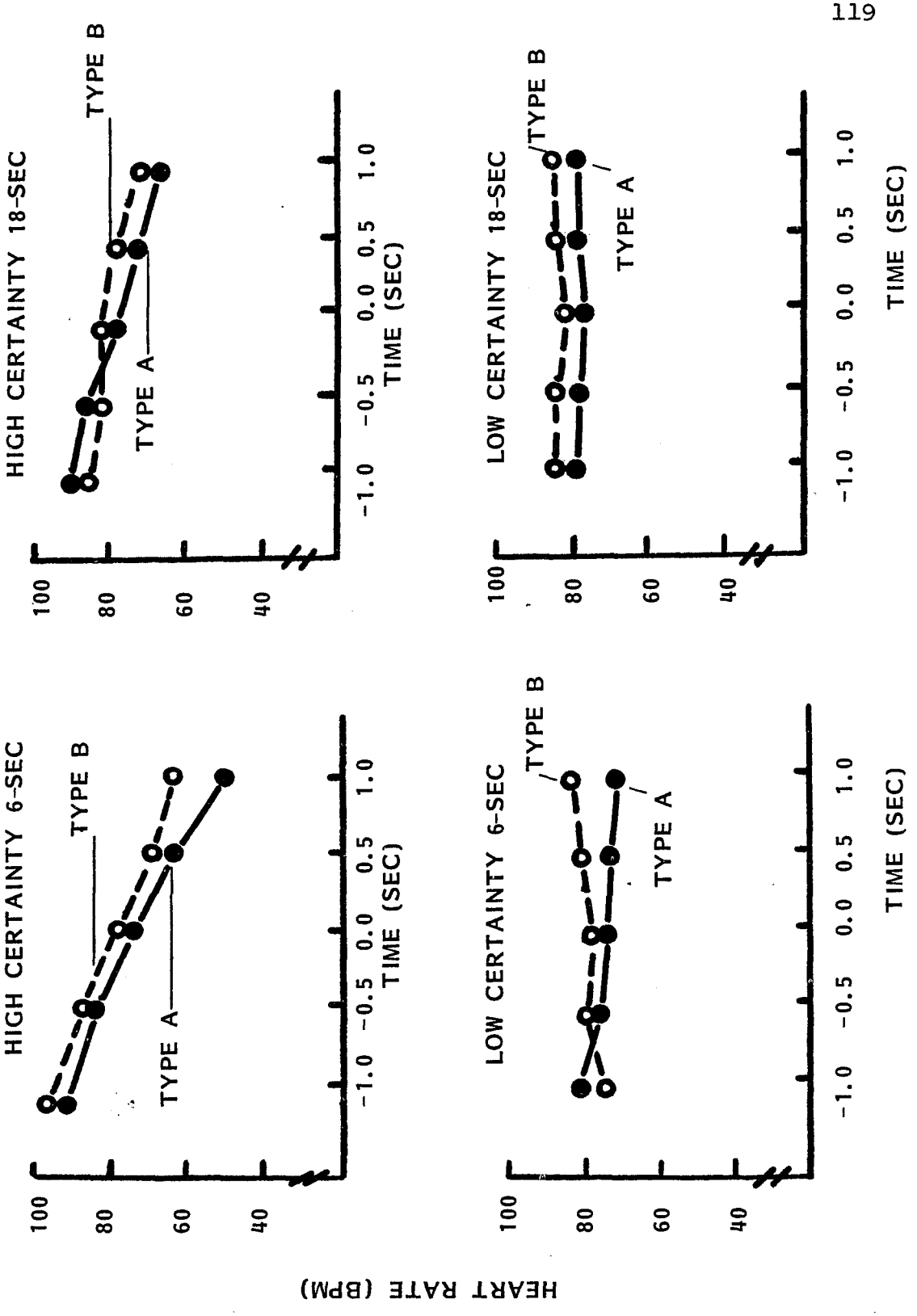

Figure 12. Phasic heart rate in beats per minute for Type A and Type B subjects during the high certainty 6-sec ITI condition (top, left), high certainty 18-sec condition (top, right), low certainty 6-sec condition (bottom, left) and low certainty 18-sec condition (bottom, right).

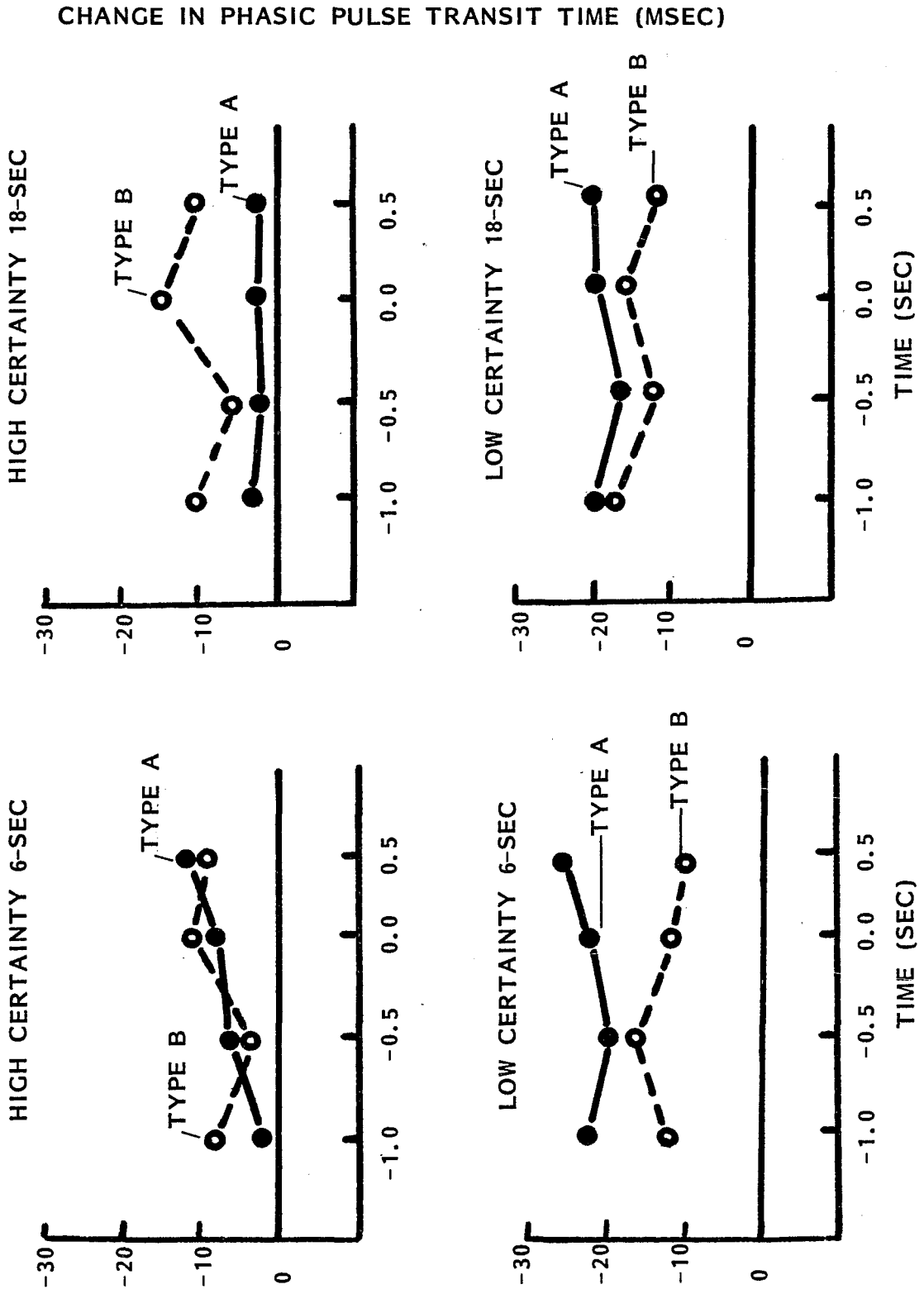

Figure 13. Change in phasic pulse transit time from baseline for Type A and Type B subjects during the high certainty 6-sec ITI condition (top, left), high certainty 18-sec condition (top, right), low certainty 6-sec condition (bottom, left), and low certainty 18-sec condition (bottom, right).

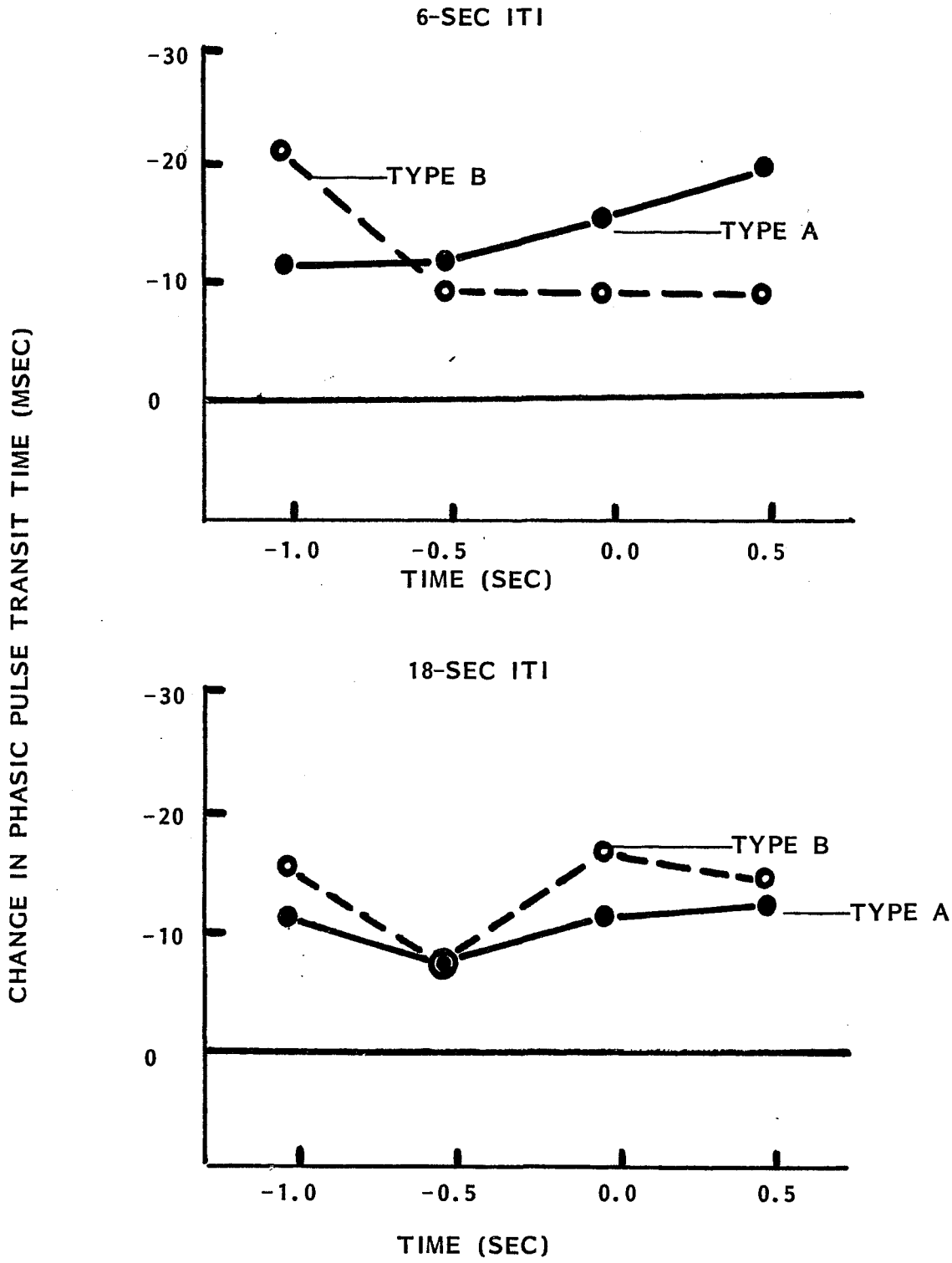

Figure 14. Change in phasic pulse transit time from baseline for Type A and Type B subjects during the ITI 6-sec (top) and 18-sec (bottom) conditions.

Figure 15. Effects of ITI Certainty and Length on ERPs in Type A and Type B subjects. All data represent evoked responses from the parietal location following presentation of the target stimuli. Vertical dashed lines are the means of the latency windows which are represented by the horizontal lines showing polarity.

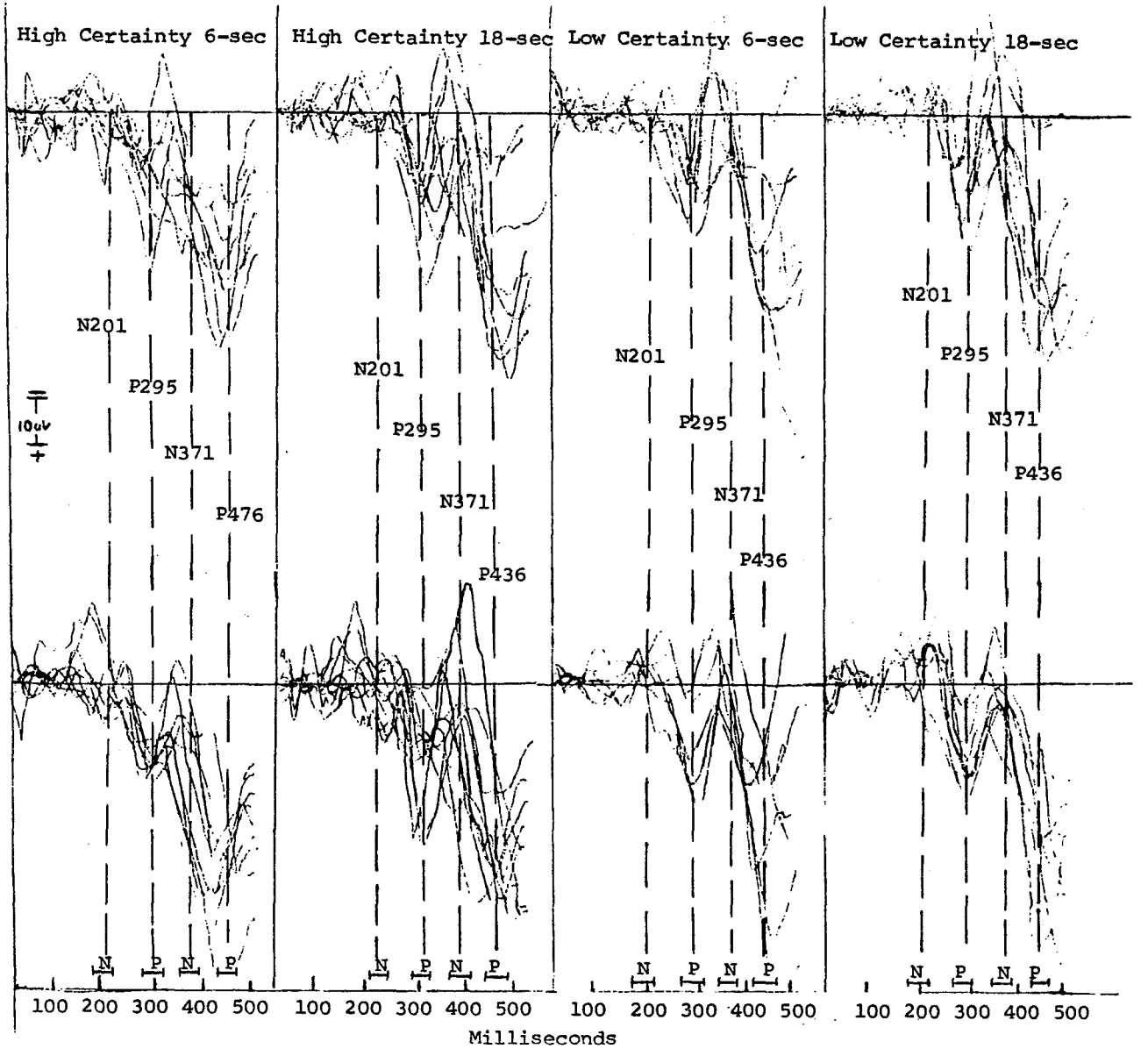
High Certainty 6-sec
High Certainty 18-sec
Low Certainty 6-sec
Low Certainty 18-sec
Type As
Type Bs
N201
P295
N371
P476
P436
10uv
N
P
100 200 300 400 500
Milliseconds

Figure 15

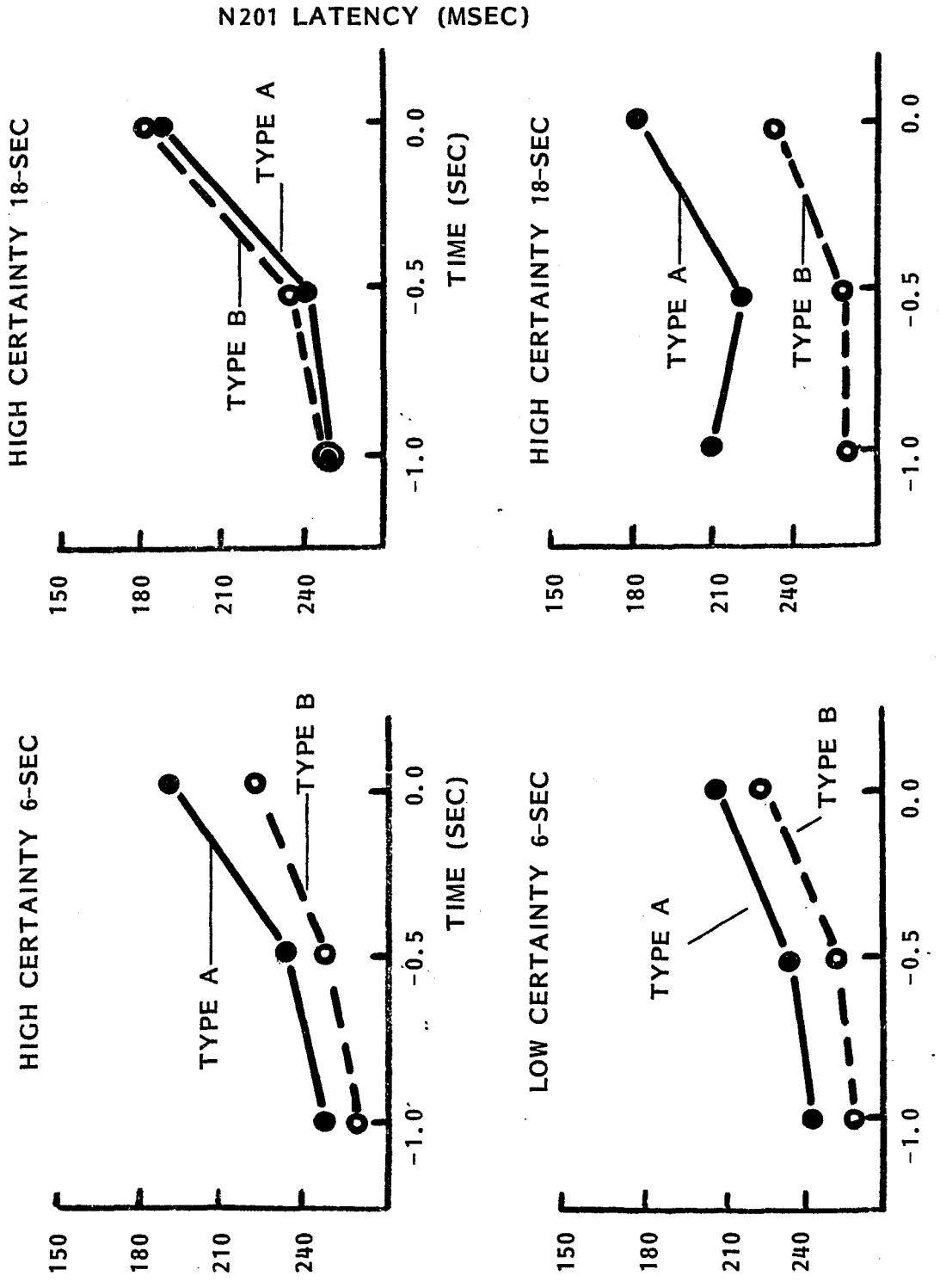

Figure 16. N201 latency across time for Type A and Type B subjects during the high certainty 6-sec ITI condition (top, left), high certainty 18-sec condition (top, right), low certainty 6-sec condition (bottom, left), and low certainty 18-sec condition (bottom, right).

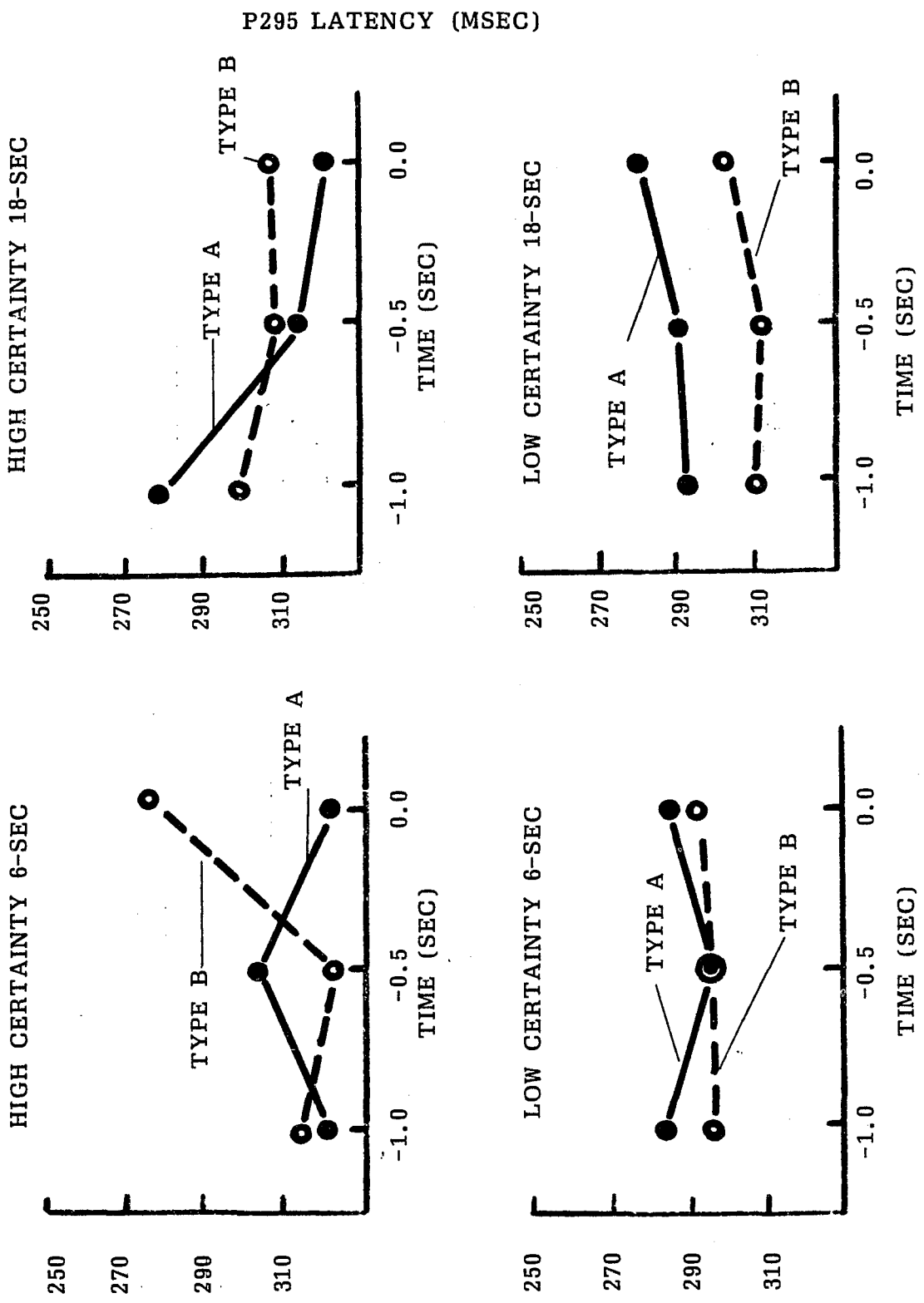

Figure 17. P295 latency across time for Type A and Type B subjects during the high certainty 6-sec ITI condition (top, left), high certainty 18-sec condition (top, right), low certainty 6-sec condition (bottom, left) and low certainty 18-sec condition (bottom, right).

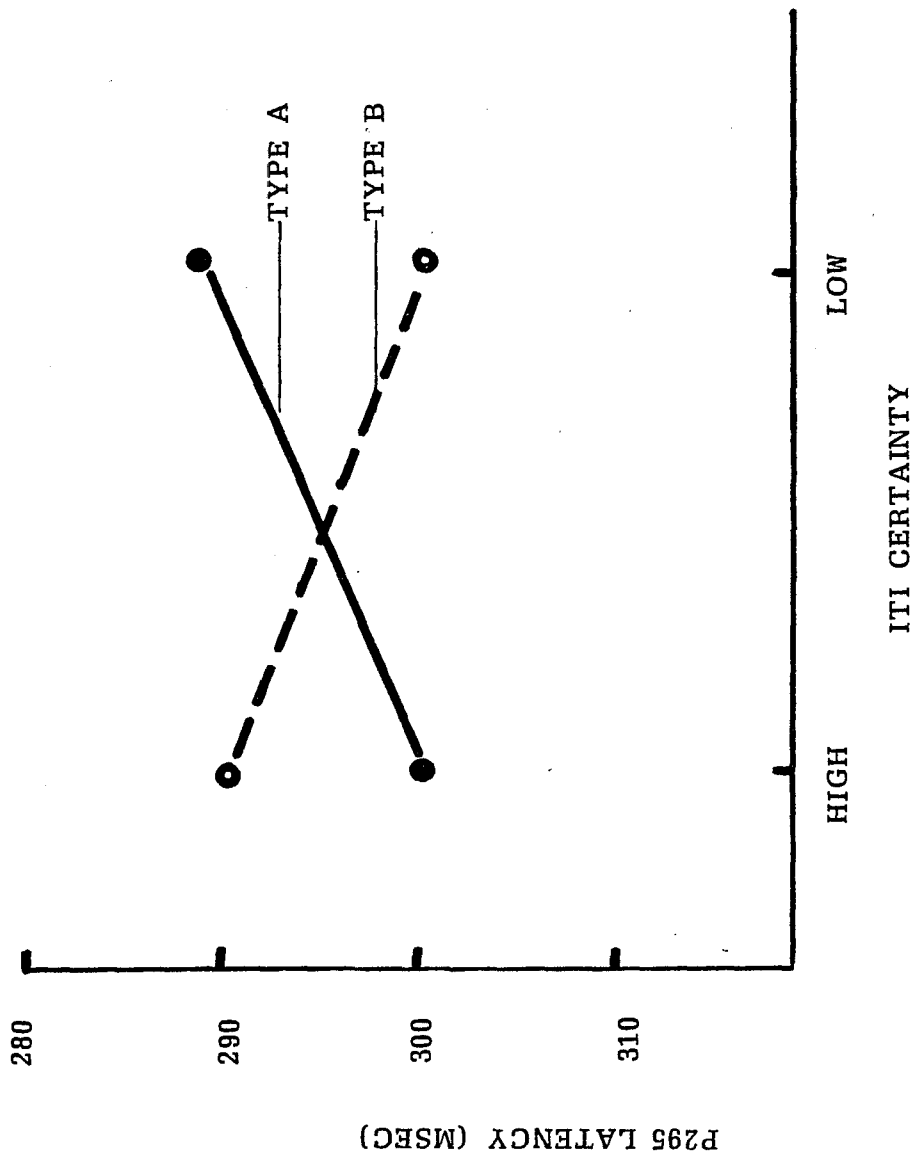

Figure 18. P295 latency for Type A and Type B subjects during high certainty and low certainty ITI conditions.

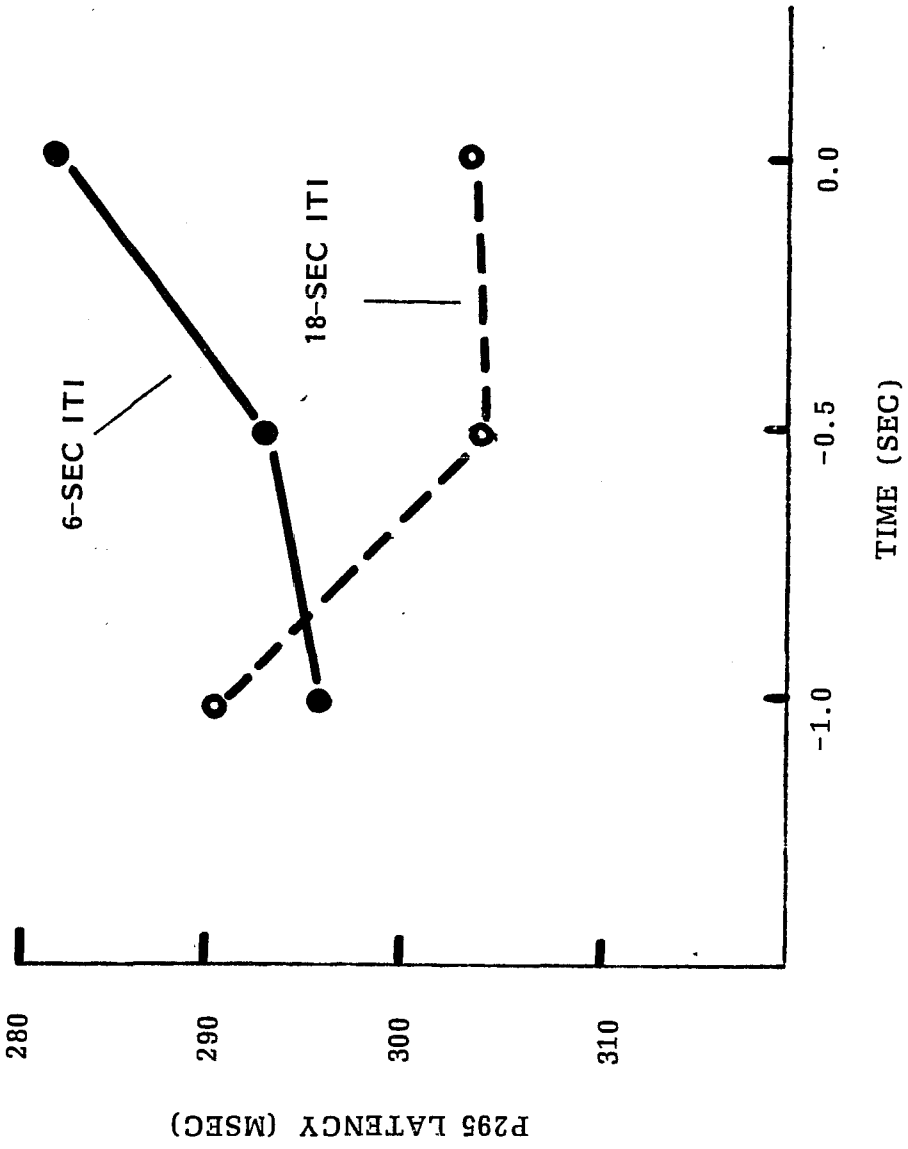

Figure 19. P295 latency across time for ITI 6-sec and 18-sec conditions.

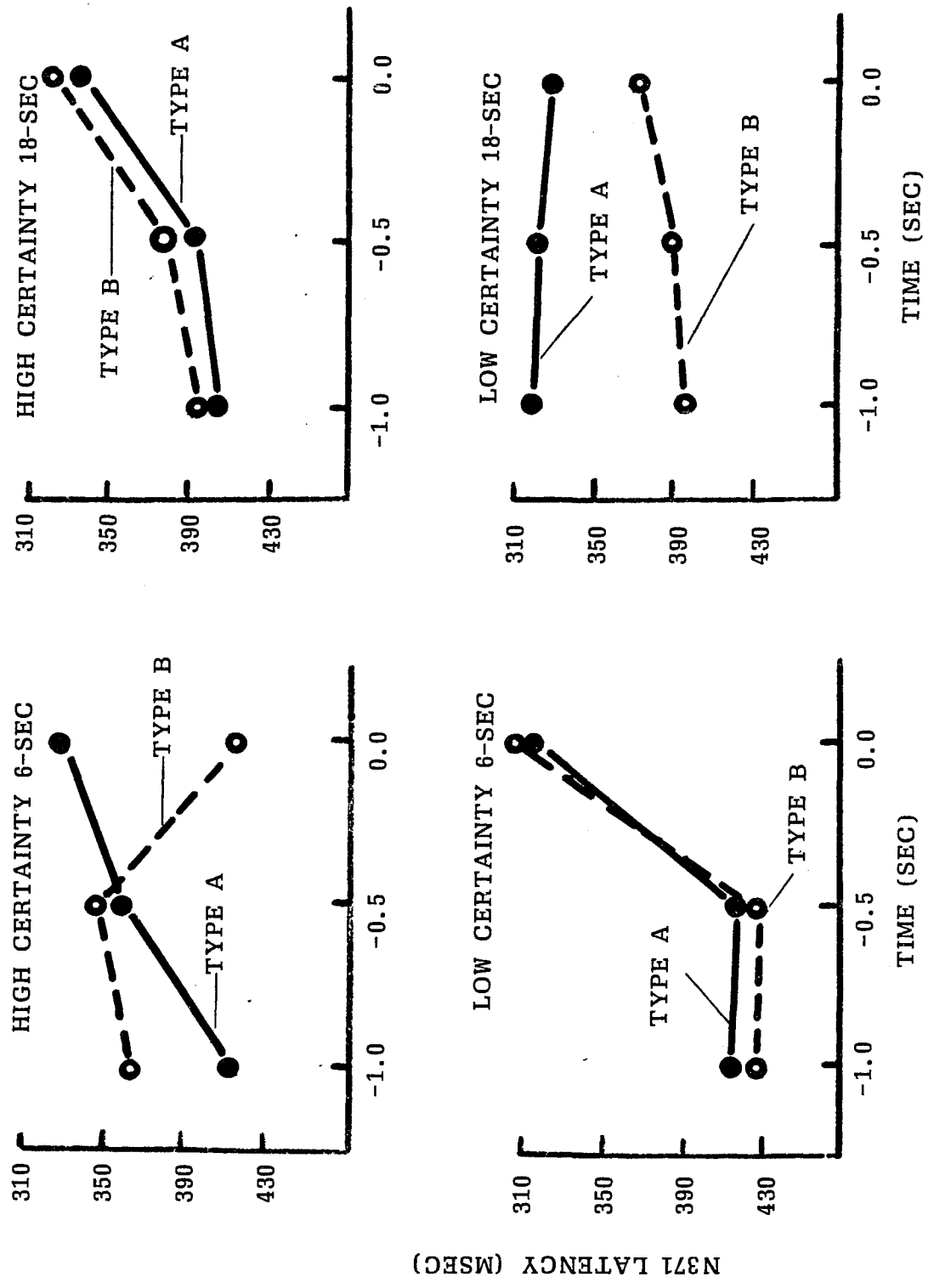

Figure 20. N371 latency across time for Type A and Type B subjects during the high certainty 6-sec ITI condition (top, left), high certainty 18-sec condition (top, right), low certainty 6-sec condition (bottom, left), and low certainty 18-sec condition (bottom, right).

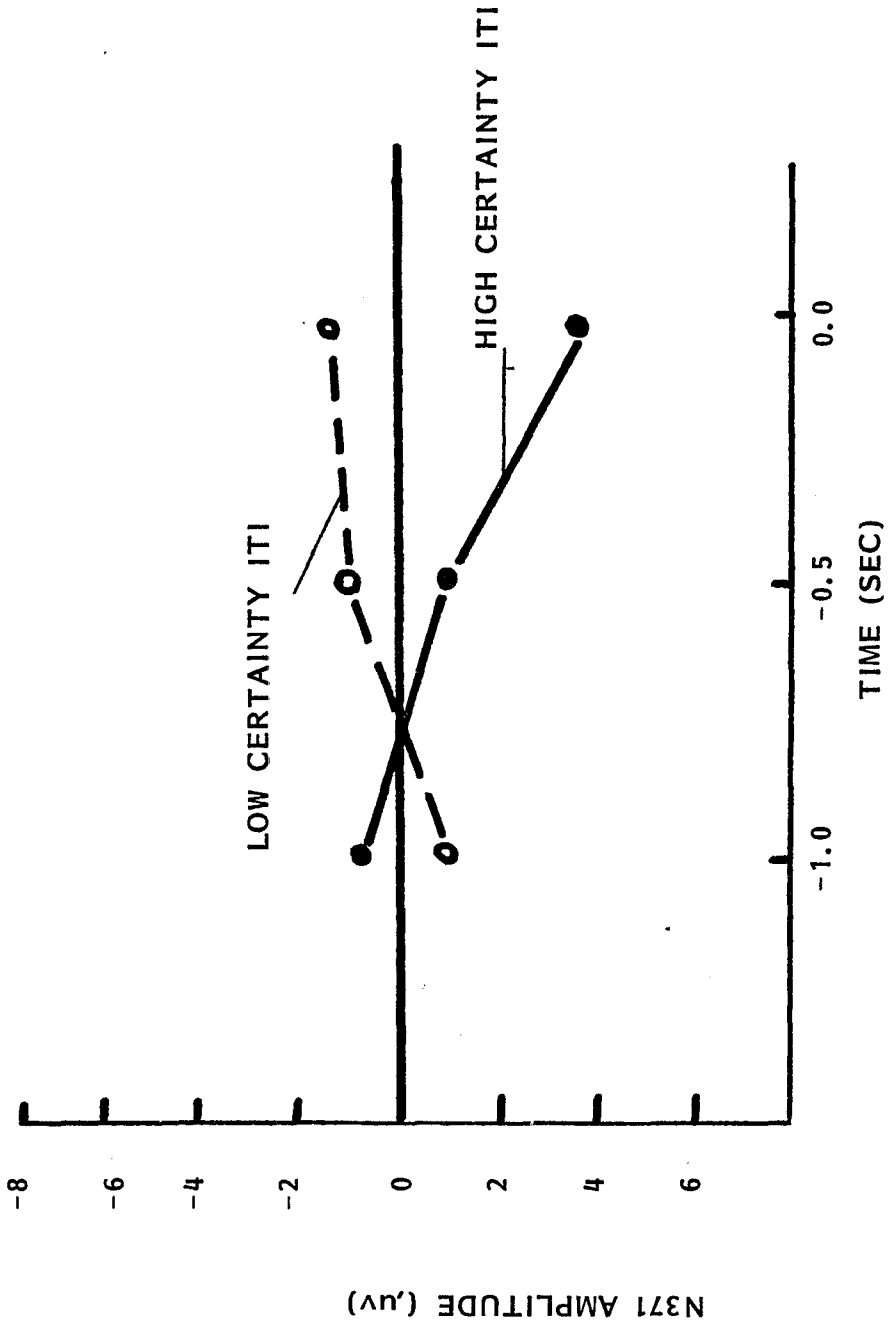

Figure 21. N371 amplitude across time for high certainty and low certainty ITI conditions.

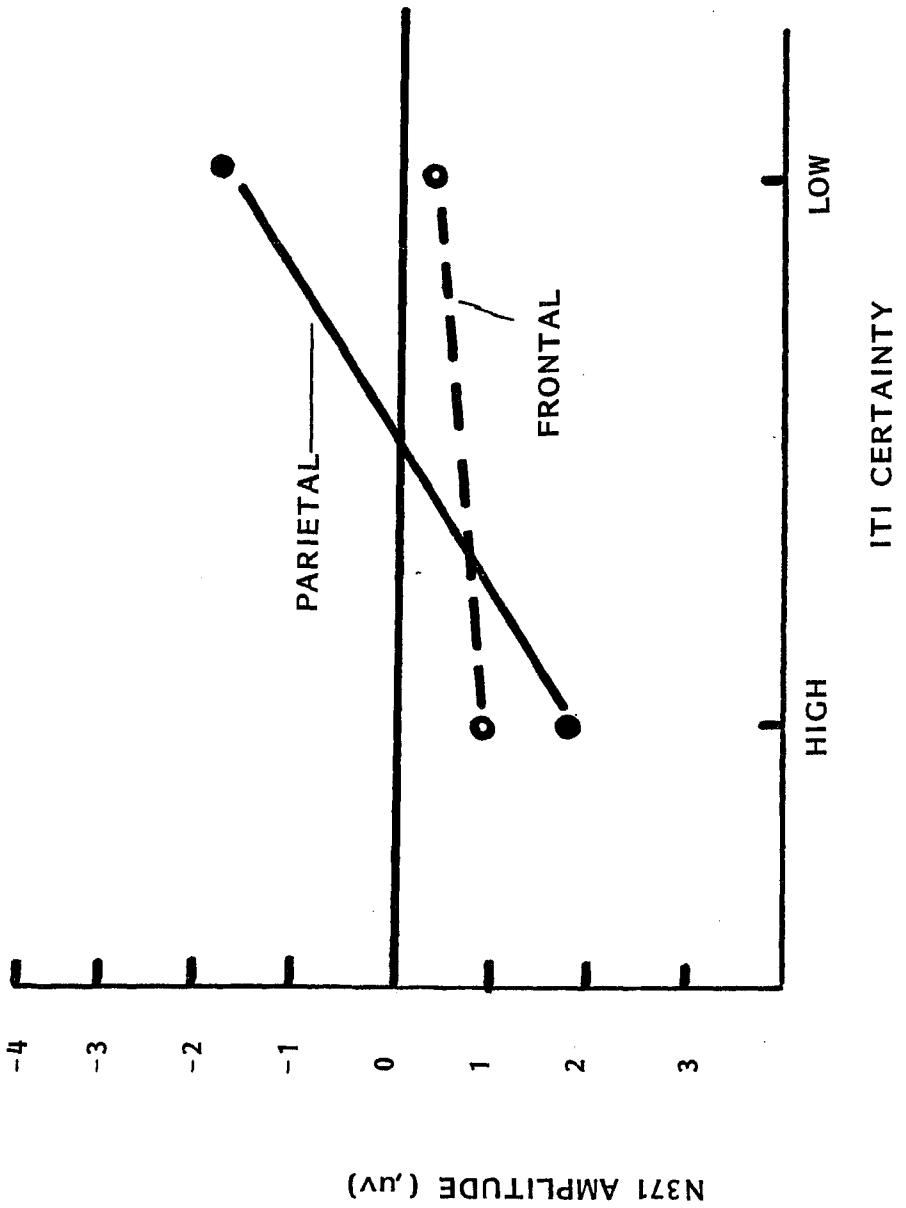

Figure 22. N371 amplitude for over parietal and frontal regions during high certainty and low certainty ITI conditions.

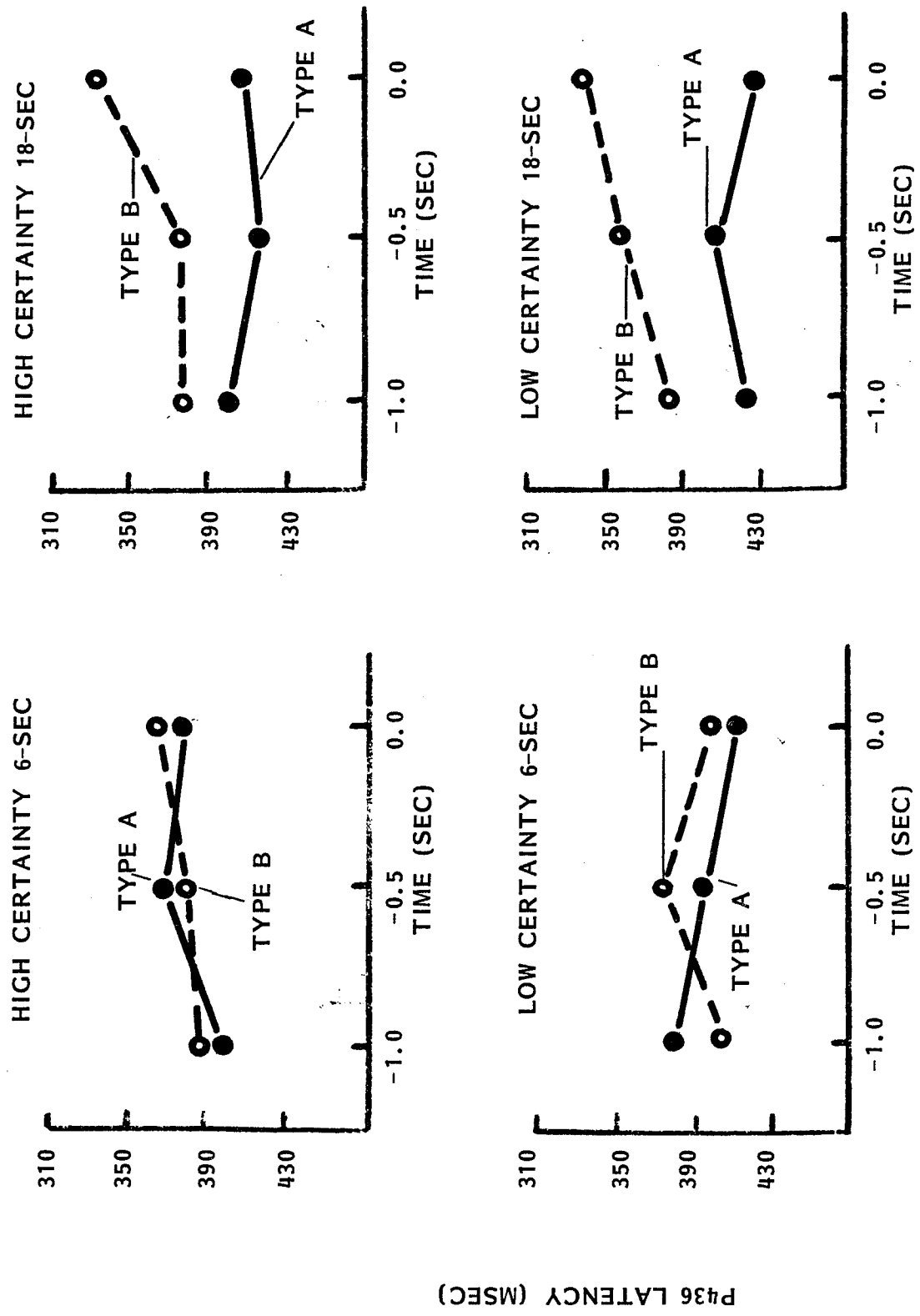

Figure 23. P436 latency across time for Type A and Type B subjects during the high certainty 6-sec ITI condition (top, left), high certainty 18-sec condition (top, right), low certainty 6-sec condition (bottom, left) and low certainty 18-sec condition (bottom, right).

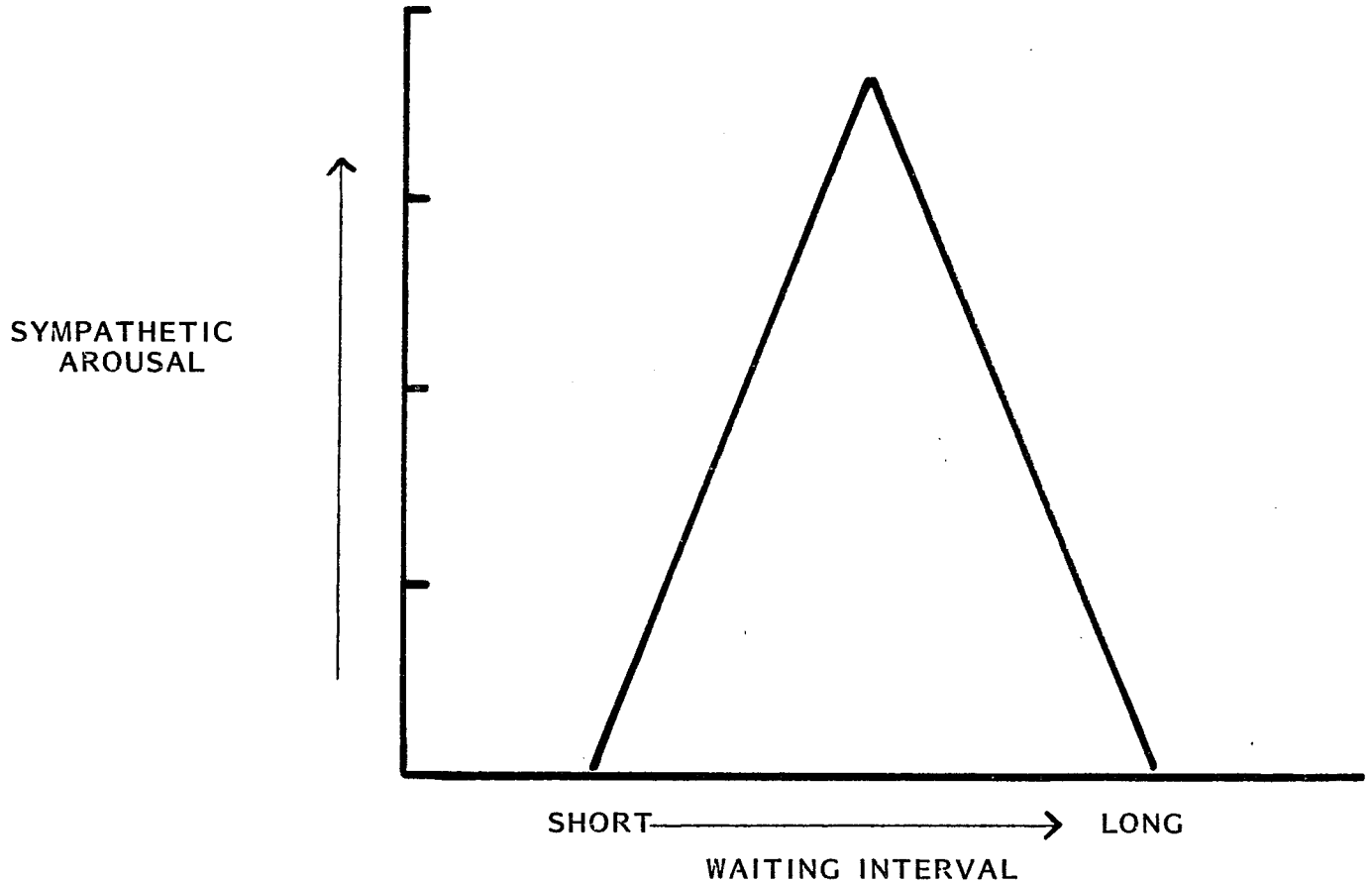

Figure 24. Possible relationship between waiting interval length and autonomic arousal in Type A and Type B individuals.

Table B-1

Summary of the effects of Type, ITI Length, and ITI Certainty on autonomic and ERP measures. Plus (+), minus (-), or zero (0) signs denote whether change in autonomic measures reflected an increase, decrease, or no change in arousal from baseline, respectively. In the case of phasic HR, plus, minus, and zero signs denote trends in absolute levels of HR rather than change scores. Less than ($<$), greater than ($>$) and approximately equal ($\approx$) signs denote relative effects of each level of the independent measures on autonomic arousal and ERP latency. An asterisk (*) denotes the effects which include the variable Time. Effects which were not significant are represented by NS.

Measures	Conditions					
	Type	p	ITI Length	p	ITI Certainty	p
Reaction Time	A < B	NS	6 sec < 18 sec	.001	High < Low	.001
Percent of Hits	A > B	NS	6 sec > 18 sec	.002	High ≈ Low	NS
Percent of False Alarms	A ≈ B	NS	6 sec < 18 sec	NS	High ≈ Low	NS
Phasic PTT	A(+) ≈ B(+)	NS	6 sec(+) ≈ 18 sec(+)	NS	High(+) < Low(+)	NS
Tonic PTT	A(+) > B(+)	NS	6 sec(+) ≈ 18 sec(+)	NS	High(+) < Low(+)	NS
Tonic HR	A(+) > B(-)	NS	6 sec(-) ≈ 18 sec(-)	NS	High(-) > Low(-)	NS
Phasic HR	A(-) ≈ B(-)	NS	6 sec (-) < 18 sec (-)	.0001	High(-) < Low(-)	.0001
Systolic BP	A(+) ≈ B(+)	NS	6 sec (+) < 18 sec (+)	NS	High(+) < Low(+)	NS
Diastolic BP	A(+) < B(+)	NS	6 sec (+) ≈ 18 sec (+)	NS	High(0) < Low(+)	.03
N201 Latency	A < B	.03	6 sec ≈ 18 sec	NS	High > Low	NS
P295 Latency	A < B	NS	6 sec < 18 sec	.02	High > Low	NS
N371 Latency	A < B	NS	6 sec < 18 sec	NS	High < Low	NS
P436 Latency	A > B	.02	6 sec < 18 sec	NS	High > Low	NS

Table B-2

Summary of the interaction effects of Type with ITI Length or Certainty on autonomic and ERP measures. Plus (+), minus (-), or zero (0) signs denote whether change in autonomic measures reflected an increase, decrease, or no change in arousal from baseline, respectively. In the case of phasic HR, plus, minus, and zero signs denote trends in absolute levels of HR rather than change scores. Less than ($<$), greater than ($>$) and approximately equal ($\approx$) signs denote relative effects of each level of the independent measures on autonomic arousal and ERP latency. An asterisk (*) denotes the effects which include the variable Time. Effects which were not significant are represented by NS.

	Conditions					
	ITI Length		p	ITI Certainty		p
	6-sec	18-sec		High	Low	
Reaction Time	A < B	A < B	NS	A < B	A < B	NS
Percent of Hits	A > B	A > B	NS	A > B	A > B	NS
Percent of False Alarms	A > B	A ≈ B	NS	A ≈ B	A > B	NS
*Phasic PTT	A(+) > B(+)	A(+) ≈ B(+)	.01	A(+) < B(+)	A(+) > B(+)	.009
Tonic PTT	A(+) > B(+)	A(+) > B(+)	NS	A(+) > B(+)	A(+) > B(+)	NS
Tonic HR	A(-) > B(-)	A(+) > B(-)	.04	A(0) > B(-)	A(0) > B(-)	NS
*Phasic HR	A(-) ≈ B(-)	A(-) ≈ B(-)	NS	A(-) ≈ B(-)	A(0) ≈ B(0)	NS
Systolic BP	A(0) ≈ B(+)	A(+) ≈ B(+)	NS	A(-) < B(-)	A(+) > B(0)	NS
Diastolic BP	A(0) < B(+)	A(+) < B(+)	NS	A(-) < B(-)	A(+) < B(+)	NS
N201 Latency	A < B	A < B	NS	A < B	A < B	NS
P295 Latency	A > B	A < B	NS	A > B	A < B	.02
N371 Latency	A < B	A < B	NS	A < B	A < B	NS
P436 Latency	A > B	A > B	NS	A > B	A > B	NS

Table B-3

Summary of the interaction effects of Type with both ITI Length and Certainty on autonomic and ERP measures. Plus (+), minus (-), or zero (0) signs denote whether change in autonomic measures reflected an increase, decrease, or no change in arousal from baseline, respectively. In the case of phasic HR, plus, minus, and zero signs denote trends in absolute levels of HR rather than change scores. Less than ($<$), greater than ($>$) and approximately equal ($\approx$) signs denote relative effects of each level of the independent measures on autonomic arousal and ERP latency. An asterisk (*) denotes the effects which include the variable Time. Effects which were not significant are represented by NS.

Measures	Conditions				
	High 6 sec	High 18-sec	Low 6-sec	Low 18-sec	p
Reaction Time	A < B	A < B	A < B	A > B	NS
Percent of Hits	A > B	A > B	A > B	A > B	NS
Percent of False Alarms	A > B	A < B	A < B	A > B	NS
*Phasic PTT	A(+) ≈ B(+)	A(+) < B(+)	A(+) > B(+)	A(+) > B(+)	NS
Tonic PTT	A(+) > B(+)	A(+) > B(+)	A(+) > B(+)	A(+) > B(+)	NS
Tonic HR	A(-) < B(0)	A(-) > B(-)	A(0) > B(-)	A(-) > B(-)	NS
*Phasic HR	A(-) < B(-)	A(-) ≈ B(-)	A(-) < B(+)	A(0) < B(0)	NS
Systolic BP	A(-) < B(+)	A(-) < B(+)	A(+) > B(0)	A(+) > B(0)	NS
Diastolic BP	A(-) < B(+)	A(-) < B(+)	A(+) < B(+)	A(+) ≈ B(+)	NS
N201 Latency	A < B	A > B	A < B	A < B	.02
P295 Latency	A > B	A > B	A < B	A < B	NS
N371 Latency	A > B	A > B	A < B	A < B	NS
P436 Latency	A > B	A > B	A > B	A > B	NS

Table B-4

Ranges of Tonic PTT (msec) for Type A and Type B Subjects Across Four Experimental Conditions

Conditions	Type A	Type B
High Certainty, 6-sec	179-335	241-323
High Certainty, 18-sec	184-351	234-337
Low Certainty, 6-sec	172-329	232-342
Low Certainty, 18-sec	180-346	248-322