

THE TYPE A BEHAVIOR PATTERN AND CARDIOVASCULAR RECOVERY
FROM A PSYCHOSOCIAL STRESSOR



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A THESIS
SUBMITTED TO THE FACULTY OF ARTS
IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR
THE DEGREE OF MASTER OF ARTS

DEPARTMENT OF PSYCHOLOGY
LAKEHEAD UNIVERSITY
THUNDER BAY, ONTARIO, CANADA

APRIL 1981

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ABSTRACT

The present study compared Type A's and Type B's in terms of their rate of cardiovascular recovery from a psychosocial stressor. Thirty-two male caucasian students were exposed to an uncontrollable perceptual conflict task (the Stroop color-word task) under conditions that made salient a sense of time pressure and competition. Heart rate and finger pulse amplitude were measured before, during and after task involvement. Results showed that although Type A's evidenced a tendency toward lower heartrates during stress, they recovered significantly slower than their Type B counterparts upon removal of the stressor. The peripheral vascular response data were equivocal for three reasons. First, there was a significant resting difference which confounded reactivity scores (Type A's showed less). Second, both A's and B's showed maximal vasoconstriction while under stress (i.e., a 'basement' effect was observed). And third, neither group recovered to its pre-stress level. The pathogenic potential of prolonged sympathetic nervous system arousal was addressed in the discussion. A theoretical model to account for inconsistencies in the psychophysiologic Type A literature was also presented.

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THE PROBLEM

Coronary heart disease (CHD) is the largest single cause of death in the industrialized Western world, yet little is known about the etiology of the illness. Traditional risk factors, such as cholesterol, hypertension, obesity, and cigarette smoking account for only about 50% of the incidence of CHD in middle aged American men (Keys, Aravanis, Blackburn, van Burchem, Buzina, Djordjenc, Fidanza, Kurvonen, Menotti, Puddy & Taylor, 1972).

Much evidence now exists to show that psychosocial factors are associated with CHD (Jenkins, 1971, 1976). Most prominent among these factors is the Type A behavior pattern (TABP). The U.S. National Heart, Lung, and Blood Institute has indirectly recognized the TABP as a significant risk factor in the pathogenesis of CHD by sponsoring a 1977 conference entitled, "The Forum on Coronary-Prone Behavior". Later, in early December 1978, a review panel of distinguished investigators was called together by the institute and they concluded that the increased risk of CHD associated with the TABP is independent of the risk imposed by age, systolic blood pressure, serum cholesterol and smoking, and is approximately of the same order of magnitude as the risk associated with any of the traditional factors (Cooper, Detre, & Weiss, in press).

STATEMENT OF THE PROBLEM

Although the statistical connection between the TABP and CHD seems well established, the underlying mechanisms linking the two remain obscure. The results of recent studies however, have suggested that Type A behavior may translate into coronary heart disease through excessive sympathetic nervous system drive. A review of the psychophysiological experiments that have examined Type A behavior shows that virtually every study has focussed its attention solely upon the magnitude aspect of sympathetic reactivity. Recovery patterns have not been studied. Whether Type A's take longer than Type B's to physiologically recover from the stressors of life is an empirical question that merits investigation because maintenance of the defense alarm reaction beyond the time required for coping responses is maladaptive and may promote disease (Abbondanza & Hermsmeyer, 1978; Buell & Sime, 1979; Cox, Evans & Jamieson, 1979; Frankenhaeuser, 1979; Goleman & Schwartz, 1976; Johansson, 1976; Johansson & Frankenhaeuser, 1973). Indeed, as a potential coronary risk factor, slow physiologic recovery may be equally as important as the magnitude of initial reactivity (Buell & Sime, 1979). The present study tested the hypothesis that, compared to Pattern B individuals, Pattern A individuals would take longer to recover physiologically, after being exposed to a challenging, uncontrollable psychosocial stressor.

The Type A Behavior Pattern: Description

Although the association between behavioral attributes and clinical manifestations of coronary heart disease was documented over three centuries ago (Harvey, 1628), the relationship was not studied systematically until the 1950's when two pioneering cardiologists, Drs. Meyer Friedman and Ray Rosenman, introduced the concept of the Type A coronary-prone behavior pattern. The classic and often quoted definition, and the one preferred by Rosenman and Friedman, describes the Type A behavior pattern as, "a characteristic action-emotion complex which is exhibited by those individuals who are engaged in a relatively chronic struggle to obtain an unlimited number of poorly defined things from their environment in the shortest period of time, and, if necessary against the opposing efforts of other things or persons" (Friedman, 1969, p. 84). More specifically:

the Type A behavior pattern is characterized primarily by aggressiveness, ambition, drive, competitiveness and a profound sense of time urgency. Some or most of these traits are present in various degrees in most men, but the man with pattern type A has them to an excessive and often inordinate degree. Certain typical muscular or motor phenomena are often

associated with these emotional traits. Speech is usually forceful, rather rapid, often explosively uneven and emphatic, and accompanied by sudden gestures such as fist clenching and taut facial grimaces. Locomotion and mannerisms are rapid, reflecting enhanced drive, competitive striving, chronic restlessness, impatience and a sense of time urgency. The man with pattern type A appears to be excessively driven to achieve and willingly committed to getting things done, while struggling against the inflexible factor of time itself and the competing and obstructing influences of other persons and things" (Rosenman and Friedman, 1971, p.80).

The most widely agreed upon conceptualization of the behavior patterns suggests that Type A individuals possess an exaggerated need to master or control their world and that Pattern A behavior is a response style for coping with perceived challenges (or threats) to environmental control (Burnam, Pennebaker & Glass, 1975; Carver & Glass, 1978; Glass, 1977ab; Matthews, Glass, Rosenman & Bortner, 1977). Type A behavior then, is a multifaceted behavioral syndrome which arises when a psychologically predisposed individual

is confronted with challenging or threatening situations (Jenkins, 1978).

The Type A Behavior Pattern: Assessment

A variety of measuring instruments have been used in attempts to quantify the TABP, these include: the Structured Interview (Rosenman, 1978); the Jenkins Activity Survey (Jenkins, Zyzanski & Rosenman, 1979); the Bortner Test Battery (Bortner & Rosenman, 1967); the Bortner Rating Scale (Bortner, 1969); the Cardiac Risk Test (van Doornen, 1979); the Thurstone Activity Scale (MacDougall, Dembroski & Musante, 1979); the Gough Adjective Check List (MacDougall et al., 1979); the Framingham Check List (Haynes, Feinleib & Kannel, 1980); the Vickers Rating Scale (Caplan & Jones, 1975); the Sales Rating Scale (Burke, Weir & DuWors, 1979); and the Rating of Statements List (van Dijkl, 1978; van Dijkl & Nagelkerke, 1979). The two most commonly used however, are the Structured Interview, and the Jenkins Activity Survey (JAS).

The Structured Interview is a highly structured stressful assessment technique in which the voice stylistics and psychomotor mannerisms of the respondent are of primary interest; verbal content is also considered, but is not of major importance (Rosenman, 1978). The interview is designed to elicit the TABP, and individuals who display it in its most extreme form are designated Type A-1 in contrast

to Type A-2, the less severe condition. Conversely, individuals at the other end of the continuum, who exhibit almost total tranquility, are designated Type B-4, in contrast to Type B-3, the less extreme Type B.

Although the Structured Interview is acknowledged to be the most accurate measure of the TABP (MacDougall et al., 1978; Rosenman, 1978) it has several drawbacks that limit its use. First, researchers wishing to use the interview method of assessment usually receive specialized training. Second, the assessment is not truly objective since it depends on the clinician's subjective interpretation of the interviewee's behavior. And third, it does not provide for numerical quantification of Pattern A. Hence the time and expense involved in getting trained, and the subjective nature of the interview have made it cost-inefficient.

In an attempt to obviate problems associated with the Structured Interview a psychometric test known as the (JAS) was developed. The JAS is a self report questionnaire which was originally designed to mimic or duplicate the Structured Interview. It was first developed in 1967 by Jenkins, Rosenman, and Friedman. Since then it has undergone many revisions. The latest one, form C (Jenkins, Zyzanski & Rosenman, 1979), was the first to be published for the scientific community.

Alternate form reliability for the JAS is high, while test-retest reliability for periods covering one to four years range between .65 and .76 (Jenkins, Rosenman & Zyzanski, 1974; Waldron, 1980). Jenkins et al., (1974) note that the reliability coefficients that they obtained probably underestimated the true stability of the test because changes in items and scoring procedures occurred between testing periods. Reliability coefficients reflecting the degree of internal consistency range from .73 to .85 (Jenkins et al., 1979; Verhagen, Nass, Appels, van Bastelaer & Winnubst, 1979).

The JAS contains three subscales, which have been labelled 'Speed and Impatience,' 'Hard-Driving Competitiveness,' and 'Job Involvement'. There is also a scale to designate overall Type A behavior (Zyzanski & Jenkins, 1970).

Administration of the JAS to a college student population is not appropriate because the test was designed for working adults, therefore a student version (form T) was developed by Krantz, Glass and Snyder (1974). Some of the items on the job involvement subscale were completely inapplicable to students and consequently were dropped from form T, leaving the speed and impatience and hard-driving competitive subscales and the overall A-B scale. In addition, the wording of several other items was modified. For instance, the question "Do you ever set deadlines or

quotas for yourself at work or at home?" was changed to "Do you ever set deadlines or quotas for yourself in courses or other things?". The extent to which these modifications affect the validity of the questionnaire is unknown, however the results of social-psychological laboratory experiments suggest the student JAS is indeed measuring Type A behavior. A number of these studies are referred to below.

The Type A Behavior Pattern: Construct Validity Evidence

Time Urgency

One of the major features of the Pattern A syndrome is a chronic sense of time urgency which is mirrored in an accelerated pace of life. Type A's lead an extremely rushed, rapid-paced existence. They eat fast. They talk fast. They think fast. They hurry others along and become very irritated or angry when forced to slow their frenetic pace. Bortner and Rosenman (1967) were the first investigators to experimentally verify the time urgency aspect of the Type A pattern. They found that when subjects were required to estimate the passage of a five minute time period, Type A's overestimated. That is, Type A's signalled the end of the interval significantly sooner than did their Type B counterparts. The authors reasoned that Type A's became more impatient while waiting, and consequently felt that more time had passed than actually did. In another time estimation experiment it was reported that Type A

students judged a one-minute interval as passing sooner than Type B students (Burnam, Pennebaker & Glass, 1975). Again, the "impatience" hypothesis was invoked to account for the finding. Price and Clarke (1978) have also provided evidence that Type A's and Type B's differ on their perception of time. Replicating previous findings, they showed that Type A's overestimated the passage of time, however, this was only true for longer time intervals (i.e., 135 seconds). Finally, European researchers have reported evidence to support the construct validity of the time urgency component of Pattern A. An investigation in Holland has found that 'time anxiety' (the fear that time passes too quickly) correlated positively with a translated version of the JAS in both coronary patients and healthy controls (Verhagen, Nass, Appels, van Basterlaer & Winnibust, 1979).

Several other experiments have provided evidence to show that Type A individuals show a greater sense of time urgency than Type B individuals. Glass, Snyder and Hollis (1974), in a two part study, reported that Type A students, presumably because of their heightened impatience, showed greater decrements in performance on a task which required low rates of responding than did Type B's. This finding was replicated by Glass (1977), who had subjects write at a slower than normal speed. A similar finding has been reported by a team of Swedish researchers who found that Type A men who were admitted to a coronary care unit for non-coronary chest pain reported significantly more

impatience in response to queueing than healthy controls (Ahnve, de Faire, Orth-Gomer & Theorell, 1979). A recent experiment by Goldband, Nielson & Patton (1980) has also found similar results. The task used in this study required subjects to respond individually to questions from the student JAS (the questions were presented via microcomputer). Subjects were asked to consider their answer to each question, but to wait (eight seconds) until instructed to actually make the response. Results showed that Type A's made significantly more premature responses than Type B's. Finally, Gastorf (1980) has provided empirical support for Friedman's (1969) hypothesis that since Type A's live with a chronic sense of time urgency they should be more punctual than Type B's. It was found that when scheduled to meet at a specified time, Type A's arrived significantly earlier than Type B's.

In conclusion, the evidence demonstrating that Type A's are more time-urgent and impatient than Type B's is both strong and consistent.

Hard-Driving Competitiveness

A second major feature of the Type A pattern involves hard-driving competitive characteristics. Friedman's (1969) clinical observation that Type A's are extremely hard-driving, ambitious, achievement oriented, and competitive, has received experimental support from numerous

studies. At least two laboratory studies have found that Type A's tend to work near maximum capacity regardless of the demand characteristics of the situation. Burnam et al., (1975) found no performance differences between A's and B's in the presence of a time deadline, however, when a time deadline was absent Type A's attempted a significantly greater number of problems. Manuck and Garland (1979) found much the same pattern; A's and B's performed equally well when a monetary reward was made contingent upon performance, however when the cues for potential reward were minimized, Type A's performed significantly better than Type B's. Results of recent Swedish research lend support to the American findings. Frankenhaeuser, Lundberg and Forsman (1980) showed that, when given a free choice of work pace, Type A's selected a significantly faster pace than Type B's (i.e., subjected themselves to a greater workload). The results of these experiments suggest that "Type A's approach all tasks in an indiscriminately hard-driving manner, whereas Type B's respond more closely to the precise nature of task requirements" (Burnam et al., 1975, p.78).

Further evidence demonstrating that Type A's are overly hard-driving and competitive comes from research showing that compared to Type B students, Type A students studied longer, attended classes more hours per week, took more courses, had higher grade point averages (Waldron, 1980), received more academic honors in college (Glass, 1977), achieved higher educational status (Appels, Jenkins &

Rosenman, 1980; Waldron, 1978), and scored higher on achievement motivation (Ray & Bozek, 1980).

Recent evidence suggests that the excessive drive among Type A's may reflect an avoidance-oriented motive to avoid failure. Gastorf and Teevan (1980) found that scores on the hard-driving competitive subscale of the student JAS were positively related to fear of failure motive (as measured by the Thematic Apperception Test's Hostile Press index). This finding is consonant with Glass's (1977a) assertion that the TABP is a coping style aimed at environmental control and that a Type A's accelerated lifestyle is in effect an attempt to avoid or escape the anxiety resulting from failure to cope successfully (i.e., failure to maintain control). There is some empirical support for the "failure-avoidant" hypothesis. In a controlled laboratory study, Krantz, Glass and Snyder (1974) demonstrated that after being exposed to a task in which there was little personal control, Type A's performed significantly better than Type B's on another task. The authors suggested that experience with the uncontrollable stressor motivated (energized) the A's to work harder on the subsequent task. It would not be unreasonable to suggest that Type A's may have found the experience of loss of control very similar to a "failure" experience, and because of this, overcompensated on the subsequent task in order to avoid the anxiety associated with loss of control (i.e., failure).

Two groups of investigators have reported that Type A men report significantly more dissatisfaction with their life goal achievements than Type B men (Keegan, Sinha, Merriman & Shipley, 1979; Romo et al., 1974). Viewed from a control systems perspective, this finding suggests that upon attaining each of their life goals Type A's reset their "life goal set point" to a significantly higher level than Type B's. If this "readjustment" results in a relatively large discrepancy between the "ideal" and the "actual" levels of achievement, compensatory behaviors (e.g., working harder, setting more deadlines, etc.) may be activated in order to minimize the discrepancy. Thus the hard-driving Type A may become ensnarled in a never ending vicious cycle of Type A behavior. This speculation is consistent with the observation that coronary patients engage in higher goal-setting behavior than noncoronary patients (Rine & Bonanci, 1976).

In conclusion, empirical evidence strongly supports Friedman's (1969) clinical observation that hard-driving competitiveness is a major component of the Type A behavior pattern.

Job Involvement

A third major aspect of the Type A behavior pattern is excessive job involvement. The job involvement dimension of the behavior pattern deals with the degree to which a person

is dedicated to or involved in his vocation. Individuals who are extremely involved are commonly called "workaholics", they are deeply engaged in a challenging, high-pressure job that frequently carries excessive supervisory responsibilities. Research has shown that Type A men, compared to Type B men, work more hours per week (Burke & Weir, 1980; Howard, Cunningham & Rechitzer, 1977), experience more work overload (Caplan & Jones, 1975; Howard et al., 1977; Keenan & McBain, 1979; van Dijkhuizen, 1979), and achieve higher occupational status (Appels et al., 1980; Waldron, 1978). In an indirectly related study, Romo, Siltanen, Theorell and Rahe (1974) found that a very high percentage of post-infarction men (from three countries: Finland, Sweden, and the U.S.) admitted on a questionnaire that they frequently worked overtime and took work home. The American sample contained the highest percentage of subjects who reported taking work home. Workaholic Type A's find job related activities highly reinforcing; indeed, their self esteem seems to be intimately related to their level of productivity. Burke and Weir (1980) state that "one may conclude that the work role and work activities must be of central importance in the value systems of Type A individuals" (p.36).

Hostility

Friedman and Rosenman's (1974) clinical observation that Type A's harbor more hostility than Type B's has recently received some empirical support.

Glass (1977a) tested Friedman's (1969) assertion that compared to Type B's, Type A's exhibit more irritation and anger when forced to slow their activity level. He found that when a confederate deliberately slowed down the pace of a conversation, Type A's reacted with more irritation and impatience. Glass stated that "This finding suggests that A's do harbor more hostile feelings than do their Type B counterparts" (p.164). A more recent study (Carver & Glass, 1978) has shown that A's showed significantly more aggressiveness after being exposed to an insoluble task than B's.

Psychometric studies carried out in different countries have also found a relationship between Type A behavior and aggression/hostility. Dimsdale, Hackett, Glock and Hutter (1978) found that, among 109 American coronary patients, there was a significant positive association between Pattern A and anger (anger was measured by the Profile of Mood States). Using Swedish university students as subjects, Lundberg (1979) reported that extreme Type A's tended to evidence higher aggression-hostility scores than extreme Type B's. van Dijn (1978) reported that Dutch speaking Type A's scored significantly higher on 'aggressivity' than than

Type B's. Chesney, Black, Chadwick & Rosenman (in press) have recently replicated this finding in a U.S. sample. They found that on the aggression scale of the adjective checklist, Type A's scored significantly higher than Type B's.

Hostility and irritability/impatience may be instrumental in the pathogenesis of CHD. Matthews, Glass, Rosenman and Bortner (1977), in a reanalysis of some of the Western Collaborative Group Study data, attempted to identify subfactors of the TABP that were associated with the subsequent occurrence of coronary disease. They found, after an examination of item content, that potential for hostility and irritability were especially prominent predictors. Indeed, Williams et al., 1980 reported that scores on the hostility scale of the MMPI were significantly and positively related to Type A behavior, moreover, it was found the hostility was significantly related to coronary atherosclerosis, independent of behavior pattern and sex.

In sum, empirical evidence strongly supports the construct validity of the Type A behavior pattern. The data indicate that the behavior pattern is comprised of at least four major components: time urgency, hard-driving competitiveness, job involvement, and hostility. Determining the relative pathogenic potential of these components is a challenge for future researchers.

The Type A Behavior Pattern and Coronary Heart Disease:
Association

There are an impressive number of studies to show that in the U.S., Type A behavior discriminates between coronary and noncoronary populations (for a review, see Jenkins, 1976). These findings have also been replicated in Poland (Zyzanski, Wrzesniewski & Jenkins, 1979), Belgium (Kittel, Kornitzer, Zyzanski, Jenkins, Rustin & Degre, 1978), Holland (Appels, Jenkins & Rosenman, 1980; Verhagen, Nass, Appels, Bastelaer & Winnubust, 1979), and Britain (Heller, 1979). Furthermore, Type A behavior is significantly related to the prevalence of CHD even after controlling for traditional risk factors such as age, cholesterol, blood pressure, and cigarette smoking (Shekelle, Schoenberger & Stamler, 1976). A major problem with retrospective epidemiological studies however, is that they cannot answer that question of whether the behavior pattern preceded or succeeded the illness. Prospective studies have resolved this issue.

A landmark prospective epidemiological investigation called the Western Collaborative Group Study has provided strong evidence to show that the TABP significantly predicts the incidence of both new and recurrent CHD. This study, in which 3,154 apparently healthy men were followed for 8.5 years, found that men who were initially identified as Type A exhibited a significantly higher rate of new CHD compared to their Type B counterparts (Rosenman, Friedman & Straus,

1966; Rosenman, Friedman, Straus, Jenkins, Zyzanski & Wurm, 1970; Rosenman, Brand, Jenkins, Friedman, Straus & Wurm, 1975; Rosenman, Brand, Scholtz & Friedman, 1976). The increased risk was not an artifact arising from the influence of other risk factors; Type A behavior constitutes a significant and independent risk factor for CHD (Brand, Rosenman, Sholtz & Friedman, 1976; Brand, 1978; Rosenman, Brand, Jenkins, Friedman, Straus & Wurm, 1975; Rosenman, Brand, Sholtz & Friedman, 1976). The overall estimated risk for Type A's, after statistical adjustment for other risk factors, was approximately double the risk of Type B's.

Results from a second prospective study, the Framingham Heart Study, have recently confirmed the Western Collaborative Group Study findings. In this study, 1674 coronary free individuals were assessed for the presence of the Type A behavior pattern (using a psychometric questionnaire), and followed for an eight year period. Multivariate analyses revealed that, after statistically controlling for the influence of other risk factors, Type A men were approximately two times as likely to develop CHD as Type B men (Haynes, Feinleib & Kannel, 1980).

Today, there are at least three other prospective epidemiological investigations that are researching the relationship between Type A behavior and CHD: the Chicago Heart Association Detection Project Industrial Study

(Shekelle et al., 1976); the U.S. Multiple Risk Factor Intervention Trial (Kornitzer, DeBacker, Dramaix & Thilly, 1977); and the Brussels Controlled Multifactorial Prevention Trial (Kittel, Kornitzer, Zyzanski, Jenkins, Rustin & Degre, 1980). Data from these studies will take years to mature, and will be very important in adding sustenance to the predictive validity of the Type A risk factor.

Men who have already suffered an infarct, and who also display the TABP are at especially high risk for reinfarction (Jenkins, Zyzanski, Rosenman & Cleveland, 1971; Jenkins, Zyzanski & Rosenman, 1976; Rosenman, Friedman & Jenkins, 1967). Discriminant function analyses revealed that, among all the risk factors available, TABP was the single strongest predictor of recurrent CHD (Jenkins et al., 1976). Furthermore, Type A behavior discriminated between recurrent and single event samples even after statistically controlling for the influence of traditional risk factors (Jenkins et al., 1976).

Another line of evidence linking CHD to Pattern A behavior comes from studies that have found Type A behavior to be related to the extent and severity of coronary atherosclerosis (Blumenthal, Kong & Rosenman, 1975; Blumenthal, Williams, Kong, Schamberg & Thompson, 1978; Frank, Heller, Kornfeld, Sporn & Weiss, 1979; Williams, Haney, Lee, Kong, Blumenthal & Whalen, 1980; Zyzanski,

Jenkins, Ryan, Flessas & Everist, 1976). This association seems to indicate that Type A behavior plays a role in accelerating the atherosclerotic process (Blumenthal et al., 1978). Further evidence to support this hypothesis comes from a study which reported a significant relationship between Type A behavior and documented progression of coronary atherosclerosis (Krantz, Sanmarco, Selvester & Matthews, 1979). Thus, as well as having more extensive coronary artery disease than Type B's, the disease seems to evolve more rapidly among Type A's.

In conclusion, one can say with reasonable confidence that the relationship between CHD and the TABP is in no way spurious, and that the behavior pattern is indeed a significant risk factor that cannot be "explained away" by other risk factors.

The Type A Behavior Pattern and Coronary Heart Disease:
Physiologic Mechanisms

Very little is known about the pathophysiologic mechanism(s) linking the TABP to CHD. This uncertain state of affairs however, is not surprising considering research efforts have largely focussed on providing statistical validation of the concept; only recently has there been a need to specify a mechanism. It is natural then, to find that current thinking concerning these mechanisms is speculative and based on circumstantial evidence.

The suggestion has recently been put forward that a potential mechanism linking the TABP to CHD may be frequent, intense, and sustained autonomic nervous system arousal. This suggestion stems from the results of a spate of recent psychophysiologic studies reporting that Pattern A individuals respond to psychosocial stressors with larger magnitude increases in cardiovascular arousal than Pattern B individuals. It should be noted however, that physiologic differences between A's and B's are found only when certain types of Pattern A and B individuals are placed in particular types of stressful situations, and that delineating the exact nature of these person and situation variables is one of the major challenges now facing researchers in the area.

The most consistent and robust physiological finding is that Type A's respond with significantly greater systolic blood pressure increases than Type B's (Dembroski et al., 1977; Dembroski, MacDougall, Shields, Petito & Luchene, 1978; Dembroski, MacDougall & Lushene, 1979b; Dembroski, MacDougall, Herd & Shields, 1979a; Glass, Krakoff, Contrada, Hilton, Kehoe, Mannucci, Collins, Snow & Elting, Exp.1 & Exp.2, 1980; Glass, Krakoff, Finkelman, Snow, Contrada, Kehoe, Mannucci, Isecke, Collins, Hilton & Elting, in press; MacDougall, Dembroski & Krantz, 1980; Katkin, 1979; Manuck, Craft & Gold, Exp.1 & Exp.2, 1978; Manuck & Garland, 1979; Weider & Matthews, 1979). A few studies have also reported that the magnitude of environmentally

induced diastolic blood pressure response differentiates A's from B's (Dembroski et al., 1978; Dembroski et al., 1979b; Glass et al., Exp.2, 1980; Houston & Jorgensen, 1980; Pittner & Houston, 1980; van Doornen, 1979; Waldron, Hickey, McPherson, Butensky, Gruss, Overall, Schmader & Wohlmuth, 1980). It has also been shown, albeit less consistently, that Type A's react to some stressors with significantly greater rest-to-task increases in heart rate than Type B's (Dembroski et al., 1977; Dembroski et al., 1978; Dembroski et al., 1979a; Glass et al., 1980; Manuck & Garland, 1979; Pittner & Houston, 1980; Van Egeren, 1979a).

Another measure of sympathetic arousal is the degree of finger pulse amplitude reactivity. Finger pulse amplitude refers to the rapid or phasic component of blood flow, and reflects the pumping action of the heart as modified by peripheral vascular mechanisms (e.g., vasoconstriction or vasodilation). Changes in finger pulse amplitude may roughly be interpreted in terms of changes in vasomotor tone (Brown, 1972, p.188), which in turn reflects changes in sympathetic arousal (Ackner, 1956). Increased arousal produces cutaneous vasoconstriction, which causes a lessening of the pulse amplitude. The degree of finger pulse amplitude reactivity has been found to be greater among Type A's than Type B's (Dembroski et al., 1979a; van Doornen, 1979; Van Egeren, 1979a).

Although many studies have demonstrated that in general Type A's respond to situations with short term, or acute physiologic overactivation, the precise mechanisms relating these phasic changes to subsequent long-term disease are unclear. It has, however, been suggested that cardiovascular hyperreactivity may translate into increased risk of CHD by initiating and/or accelerating the atherosclerotic process (Blumenthal et al., 1978; Herd, 1978; Williams, 1975; Williams et al., 1978). There is much speculation about exactly how heightened sympathetic activation promotes coronary artery disease. One theory posits that repeated instances of arousal may, through increases in blood pressure and flow turbulence, cause arterial 'injury' or vascular endothelium 'damage' (Herd, 1978; Ross & Glomset, 1976). Chronically elevated levels of circulating catecholamines may also cause lesions in coronary blood vessels (Haft, 1974). Furthermore, heightened sympathetic discharge may adversely affect blood clotting mechanisms and bring on thrombosis (Davies & Reinert, 1965; Davis, 1974; Eliot & Todd, 1976). Sympathetic hyperreactivity may also be instrumental in precipitating acute clinical CHD events inasmuch as the demand for increased myocardial oxygen consumption may strain the capacity of an already diseased coronary system and produce serious arrhythmias or fatal ventricular fibrillation (Eliot, 1979; Malik, 1973; Myers & Dewar, 1975; Williams et al., 1978).

Additional supporting evidence for the pathogenic potential of cardiovascular hyperreactivity comes from psychophysiological studies with coronary patients. Schiffer, Hastley, Schulman & Abelman, (1976) exposed subjects to a psychosocial stressor and found that the magnitude of arousal (heart rate and blood pressure) clearly discriminated between persons exhibiting signs of angina pectoris and controls. A similar finding has been reported by Sime, Pierrynowsky & Sharratt, (1977), who compared the magnitude of environmentally induced cardiovascular arousal in post infarct patients and matched controls, and found that the degree of heart rate and blood pressure changes discriminated between the groups: post infarct patients were more reactive. In a recent replication, Sime, Buell and Eliot (1980) found that, in response to a stress quiz, patients with angina, hypertension, and/or electrocardiogram changes evidenced significantly higher diastolic pressure responses than either patients without these symptoms or healthy controls.

The degree of cardiovascular lability also seems to discriminate between those post infarct patients who eventually suffer a reinfarction and those who do not. In a two year follow-up, Sime, Buell and Eliot (1979), reported that those patients who experienced recurrent infarcts were the same patients who had shown a significantly higher degree of cardiovascular reactivity two years before when being exposed to a stressor.

One final bit of evidence which suggests that cardiovascular hyperreactivity may be related to coronary disease comes from a prospective epidemiological study which found that the magnitude of diastolic blood pressure increase in a cold pressor test was the best single predictor of new cases of CHD over a twenty year period (Keys, Taylor, Blackburn, Brozek, Anderson & Simonson, 1971).

In conclusion, research supports the hypothesis that Type A's are at higher risk for CHD than Type B's because of more intense activation of the sympathetic nervous system. The evidence, however, is only circumstantial because it is not known whether physiological hyperreactivity is a predictor for CHD in asymptomatic individuals without previously diagnosed heart disease. It is important also to note that to date only the magnitude aspect of hemodynamic reactivity has been related to pattern A behavior. There is no evidence that Type A's experience either more frequent or more prolonged episodes of arousal than Type B's.

The Present Study

Although it seems fairly well established that Type A's respond to some situations with more intense physiologic activation than Type B's, nothing is known about how these individuals recover upon removal of the stressor. If we

assume that repeated instances of slow physiologic demobilization carry pathogenic potential, then an important question is whether Type A's, compared to Type B's, experience more prolonged cardiovascular arousal following the removal of a stressor. The present study tested this hypothesis. Subjects were challenged to perform well under instructions that were designed to create a highly competitive and time urgent situation. Heart rate, and finger pulse amplitude (digital vasomotor behavior) were monitored before, during, and after task involvement. A brief, self-report questionnaire was administered upon termination of the post-stress relaxation period.

METHOD

Subjects

The subjects were 32 male Caucasian introductory psychology students who volunteered to participate in a "personality and physiology" experiment. All subjects received a one point credit toward their final grade in the course. Their mean age was 20.7. The standard deviation was 2.7. The range was 17 to 29.

Apparatus

Three paper and pencil measures of Type A behavior were used: the student version of the Jenkins Activity Survey (JAS, Form T) (Krantz et al., 1974), and two other Type A inventories: the Cardiac Risk Test (van Doornen, 1979), and the Thurstone Activity Scale (MacDougall et al., 1979).

The student JAS, which is a 44 item self report Type A inventory based on the adult JAS but modified specifically for college students, is comprised of three scales: the overall A-B scale (JAS-A/B), the Speed and Impatience scale (JAS-S/I), and the Hard-Driving Competitive (JAS-H/C) scale. Scores on the overall A-B scale may vary from 0-21, but usually cluster around 7 or 8. Mean JAS-S/I scale scores usually fall in the 15-20 range, however may vary from 0-42. JAS-H/C scale scores may vary from 0-34, but usually fall in the 10-15 range. High scores indicate more Type A behavior.

The Cardiac Risk Test was developed in Holland by van Doornen (1979). It is a heterogeneous compound of 22 translated items that have been found to discriminate Dutch infarction patients from controls. Although a factor analysis (van Doornen, 1979) has yielded three factors: impatience, activity, and goal directed striving, together describing 40% of the test variance, scale scores for these factors are not available. Each item is scored on a single five point scale, with a high score indicating more Type A behavior.

The Thurstone Activity Scale is a modified version of the activity scale of the Thurstone Temperament Schedule, in which a five-point Likert scale was used instead of the original four-point system. It includes 20 items such as "Do you speak louder than most people?" Each item is scored on a single scale, with a high score indicating more Type A behavior.

A 15 item post experimental questionnaire (Appendix) was used to measure cognitive, affective, and somato-visceroperceptive reactions. Questions were phrased in such a way that subjects could circle one of four possible answers. The following is a typical example of the questions asked: "How challenging did you find the task?"
1) not at all, 2) somewhat, 3) moderately so, 4) very much so.

A photoplethysmographic transducer (Model # 9553) which was placed on the first phalanx of the right hand middle finger, provided continual information about finger pulse amplitude and heart rate. It was connected to a voltage pulse pressure coupler (Model # 9853A) of a Beckman polygraph (Type R), which was set at a fixed gain for all subjects.

The task stimulus was a modified Stroop color-word conflict chart (Stroop, 1935). On the 22" x 32" chart, names of colors were printed in conflicting colors of ink (e.g., the word "red" may have been printed in blue ink).

Seven different names of colors (color-words) were used (green, yellow, orange, blue, brown, red, and black). These color-words were printed in seven different colors of ink (green, yellow, orange, blue, brown, red or black). Each color-word appeared in 1/2 inch stencil. The chart, which contained a total of 126 color-words (six columns of twenty-one), was affixed at eye level to a wall four feet in front of the subject.

Procedure

Upon completing the Type A questionnaires, the subject was seated in a comfortable high backed chair and told that the aim of the investigation was to see how personality relates to heart rate. The photoplethsmographic transducer was attached, and the subject was asked to close his eyes and relax for six minutes. At the conclusion of the adaptation period the subject was instructed to open his eyes, and then in a serious voice he was told "In a minute you will be presented with a difficult intellectual task, and in order to achieve a high score you will have to think quickly and really concentrate." The Stroop color-word conflict task was then explained using a practice wall chart which contained example test stimuli. On the chart, names of colors were printed in conflicting colors of ink and the subject was told to verbalize the color of ink while ignoring the word content.

After familiarization with the example test stimuli, the subject was told, "You will now be tested on how well you can do on the actual task ... there is a six minute deadline therefore it is very important that you concentrate and read quickly if you are to obtain a high score ... if you make a mistake you must stop and correct it before you continue; for each mistake you make five points will be subtracted from your final score ... when you finish the test I will compare your score with the scores of other students who have previously completed the test." The subject was then told to close his eyes while the experimenter set a conspicuously placed timer-clock to ring in six minutes. The actual test stimuli were exposed and the subject was asked to open his eyes and begin reading.

At the conclusion of the six minute stress period the timer-clock rang, and the subject was told to stop reading from the chart, close his eyes, and relax for six minutes. After the recovery period ended, the subject was told that his score would be compared to the scores of other students immediately after he completed a 15 item self report questionnaire. After this, he was told that his score would not be compared to the scores of other students, and the reason for the deception was explained. The subject was then thanked, asked to remain silent about the experiment, and excused.

Measures and Scoring

Heart rate (HR) was measured by counting the number of beats that occurred on the polygraph output in each minute. The last minute of the initial six minute adaptation period served as the base rate. A single stress HR score was obtained for each subject while he responded to the Stroop test by averaging the six one-minute stress HRs together. An average recovery heart rate was obtained through an identical procedure.

Finger pulse amplitude (FPA) scores were derived for each minute by calculating the distance, in millimeters, between the peaks and troughs of heart beats that occurred at five equidistant points within each minute. The mean of these five values was then computed and served as that minute's measure of FPA. The last minute of the initial adaptation period was used as the base score, and single stress and recovery scores were obtained by averaging the six one-minute stress and recovery scores respectively.

Performance data consisted of the number of Stroop color-words completed during the six minute stress period.

Method of Data Analysis.

For each physiologic parameter the data were subjected to hierarchical multiple regression analyses (Kim & Kohout, 1975, P. 336). Multiple regression is a multivariate

statistical technique which allows for the assessment of the independent effects of multiple independent variables (called predictors) upon a single dependent variable (called the criterion). In the present experiment the multiple regression approach was useful in partitioning the sources of variance in the physiologic data. The technique, for example, allowed an analysis of heart rate recovery in which recovery heart rate scores were adjusted for both base heart rate and stress heart rate values. The multiple regression approach then, is conceptually similar to the analysis of covariance, but instead allows for the examination of a predictor variable that is in continuous rather than category form.

Two main sets of multiple regression analyses were done on the data from each of the two physiologic response channels. The first was done using the stress measure as the criterion, and base and Type A behavior as predictors. The second analysis performed on each of the two physiological response parameters employed the recovery measure as the criterion variable. For these analyses the variability associated with base was removed first, followed by the variability associated with stress and Type A behavior. As in all the analyses, the proportion of variability in the criterion associated with Type A behavior was removed last to ensure that its effects would be independent of individual differences in base and/or stress scores. Although the data were analyzed using heirarchical

multiple regression, figures will be presented using median splits. The predictor variable for all of the following analyses will be JAS scores since the JAS accounted for greatest percentage of the variance in the criterion variables. During the presentation of the results, the term 'Type A behavior' will be used to refer to the overall A-B scale of the JAS. Other scales of the JAS will be referred to by name (i.e., JAS-S/I or JAS-H/C). Summary multiple regression tables of the results of analyses using the Cardiac Risk Test and Thurstone Activity Scale inventories as measures of Type A behavior can be found in Appendix

RESULTS

The Type A predictor variables for the following analyses will be the three scales of the JAS: the overall A-B scale (JAS-A/B), the speed and impatience scale (JAS-S/I), and the hard-driving competitive scale (JAS-H/C). The two criterion variables will be heart rate (HR) and finger pulse amplitude (FPA).

Type A Measures

Table 1 presents a correlation matrix of the Type A measures. The mean JAS-A/B score of 7.53 is typical of young college males (cf. Glass, 1977a).

Table 1. Correlations (r)*, means and standard deviations for JAS scales (n=32).

Variable	1.	2.	3.
1. JAS - A / B		.775	.575
2. JAS - S / 1			.510
3. JAS - H / C			
MEAN	7.53	17.24	11.12
S.D.	3.15	6.00	4.27

* $p < .01$ when $r > .40$

Heart Rate

Mean heart rate (HR) and standard deviations for each Type A measure across each of the three experimental periods are presented in Table 2. A correlated t-test revealed that the mean base-to-stress HR increase of 17.2 b.p.m. was highly significant ($t=-10.31$, $df=31$, $P<.001$).

Using mean stress heart rate as the criterion, the variability associated with base heart rate and Type A behavior was removed. Results showed that after partialling out the effect of base HR upon stress HR, no relationship was found between any of the JAS scales and mean stress HR. The only variable that was associated with mean stress HR was base HR, which accounted for 59.25% of the base HR variance $F(1,30)=43.62$, $P<.001$. Surprisingly, the standardized regression coefficient (i.e., beta) was negative, indicating HR hyporesponsivity on the part of Type A individuals. This can be seen in Figure 1.

Analyses at successive minutes of stress revealed that tonic HR during the fourth minute was significantly higher for B's compared to A's. Base HR accounted for 53.8% of the fourth minute HR variance $F(1,29)=32.54$, $P<.001$, and Type A behavior accounted for 6.4% of the remaining variance in the criterion $F(1,28)=4.47$, $P<.05$.

Table 2. Means and standard deviations for heart rate (in b.p.m.) during base, stress, and recovery periods for individuals scoring above and below the median on each of the JAS scales.*

	<u>B A S E</u>		<u>S T R E S S :</u>		<u>R E C O V E R Y</u>	
	\bar{X}	S.D.	\bar{X}	S.D.	\bar{X}	S.D.
JAS-A/B						
Type A	65.9	11.0	79.4	14.3	66.8	9.9
Type B	65.0	8.2	86.6	14.6	66.1	9.7
JAS-S/l						
High	66.7	11.0	81.3	16.6	67.4	10.1
Low	64.3	8.5	85.4	12.4	65.5	9.3
JAS-H/C						
High	67.7	11.3	85.8	17.9	69.6	10.6
Low	63.8	8.5	80.4	11.5	64.0	8.2

*n=17 for Type A's (or high scorers) and
n=15 for Type B's (or low scorers)

HEART RATE (B.P.M.)

○--○ Type A

△--△ Type B

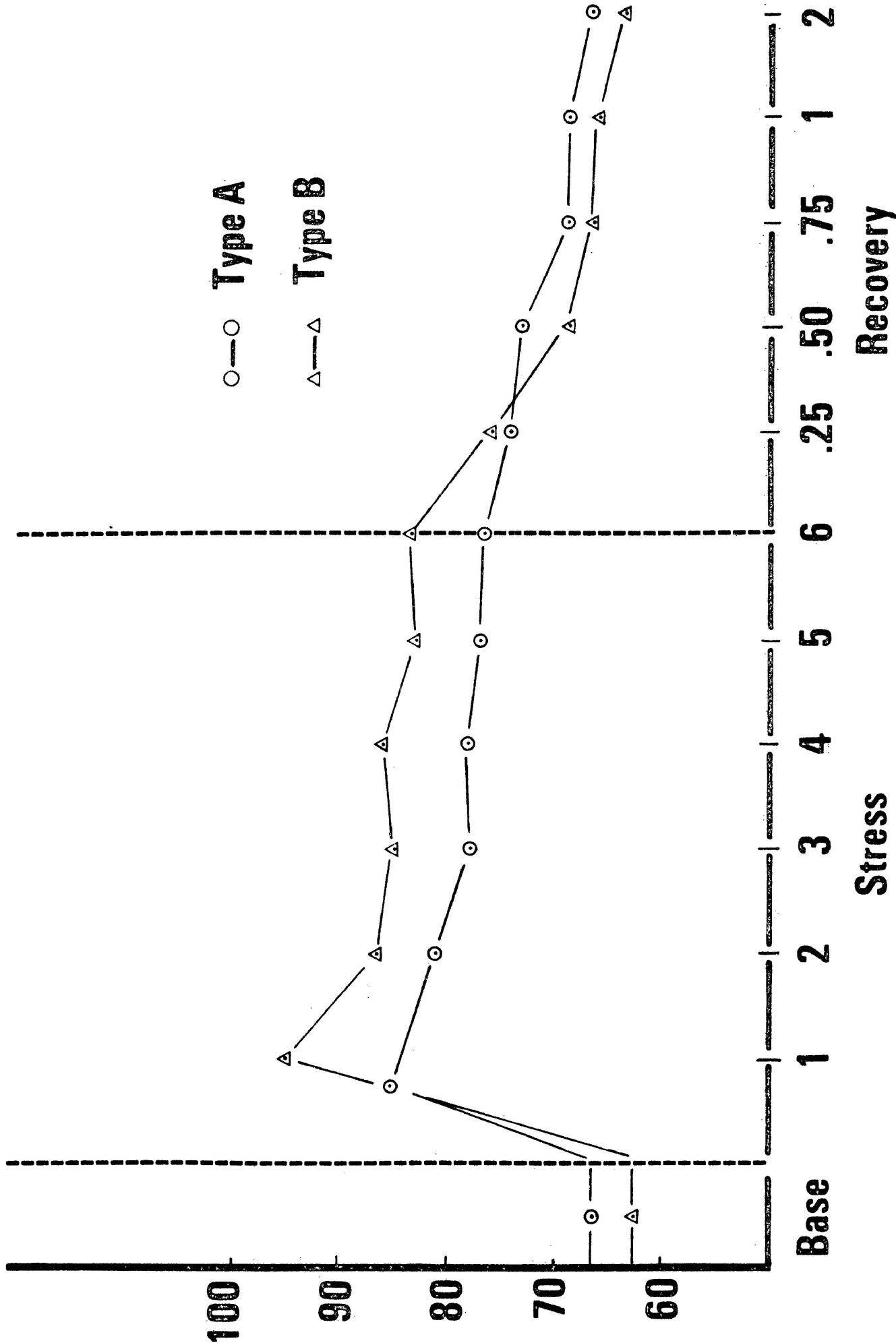


Figure 1. Heart rate as a function of time (in minutes) for individuals scoring above and below the median on the JAS-A/B scale.

The second set of analyses used mean recovery HR as the criterion variable. It was found that neither the JAS-A/B nor JAS-S/I scale accounted for a significant proportion of the variance in this criterion. The JAS-H/C scale however, was significantly associated with mean HR recovery. The variability associated with base heart rate (89.4%) was significant $F(1,28)=236.20$, $P<.001$, as was the variability (3.0%) associated with stress HR $F(1,27)=10.80$, $P<.01$. JAS hard-driving competitive scores accounted for 1.13% of the unique variability in recovery mean HR $F(1,26)=4.57$, $P<.05$. The direction of the relationship was positive indicating that hard-driving/competitive individuals recovered significantly less than did non hard-driving/competitive persons.

Analyses at successive minutes of recovery revealed that during the first minute Type A's recovered significantly less than their Type B counterparts. Base HR accounted for 83.88% of the first minute recovery HR variance $F(1,29)=150.96$, $P<.001$. The proportion of variability associated with stress HR was 9.15%, $F(1,28)=36.80$, $P<.001$. Type A behavior was associated with 1.87% of the unique variability in recovery HR scores $F(1,27)=9.93$, $P<.01$. Further analyses revealed that during the first minute of recovery, individuals who scored high on the JAS-H/C scale recovered significantly less than individuals who scored low. Hard-driving competitive scores accounted for 1.82% of the first minute recovery HR variance

$f(1,30)=9.98, P<.01$). The JAS-H/C scale was also related to HR scores during the second minute of Recovery. A multiple regression analysis revealed that base HR accounted for 86.52% of the second minute recovery HR variance $F(1,30)=192.57, P<.001$, while stress HR was associated with 2.48%, $F(1,29)=6.53, P<.025$. JAS-H/C scores were positively associated with 2.34% of the variability in the second minute recovery HR scores $F(1,28)=7.30, P<.025$. In figure 1, the HR means for Type A's and B's (JAS-A/B median split) during four equidistant points in the first minute of recovery are presented in order that the differential recovery rates can be seen. The graph does not extend beyond the second minute of the recovery period because after two minutes of recovery both groups returned to, and remained at, their basal levels.

Finger Pulse Amplitude

Mean finger pulse amplitude (FPA) and standard deviations for each Type A measure across each of the three experimental periods are presented in Table 3.

Correlational analyses showed that Type A's had a significantly lower resting FPA level than their Type B counterparts. For the overall JAS-A/B scale, $r=-.35, P<.05$, while for the JAS-S/I and JAS-H/C scales $r=-.336, P<.06$, and $r=-.408, P<.02$, respectively. A correlated t-test revealed that the mean base-to-stress decrease of -12.4 mm. was highly significant ($t=7.62, df=31, P<.001$).

Figure 2 shows graphically the mean FPA scores for Type A's

Table 3. Means and standard deviations for finger pulse amplitude (measured in millimeters of pen deflection) during base, stress, and recovery periods for individuals scoring above and below the median on each of the JAS scales.*

	<u>B A S E</u>		<u>S T R E S S</u>		<u>R E C O V E R Y</u>	
	\bar{X}	S.D.	\bar{X}	S.D.	\bar{X}	S.D.
JAS-A/B						
Type A	17.3	10.5	8.8	5.2	13.2	7.4
Type B	26.2	10.1	9.3	4.2	18.2	8.3
JAS-S/1						
High	18.2	12.2	8.3	5.4	14.1	8.3
Low	25.1	8.6	9.8	3.7	17.1	7.8
JAS-H/C						
High	16.9	12.1	7.6	4.7	12.2	7.2
Low	24.9	9.1	10.1	4.4	18.1	8.0

*n=17 for Type A's (or high scorers) and n=15 for Type B's (or low scorers)

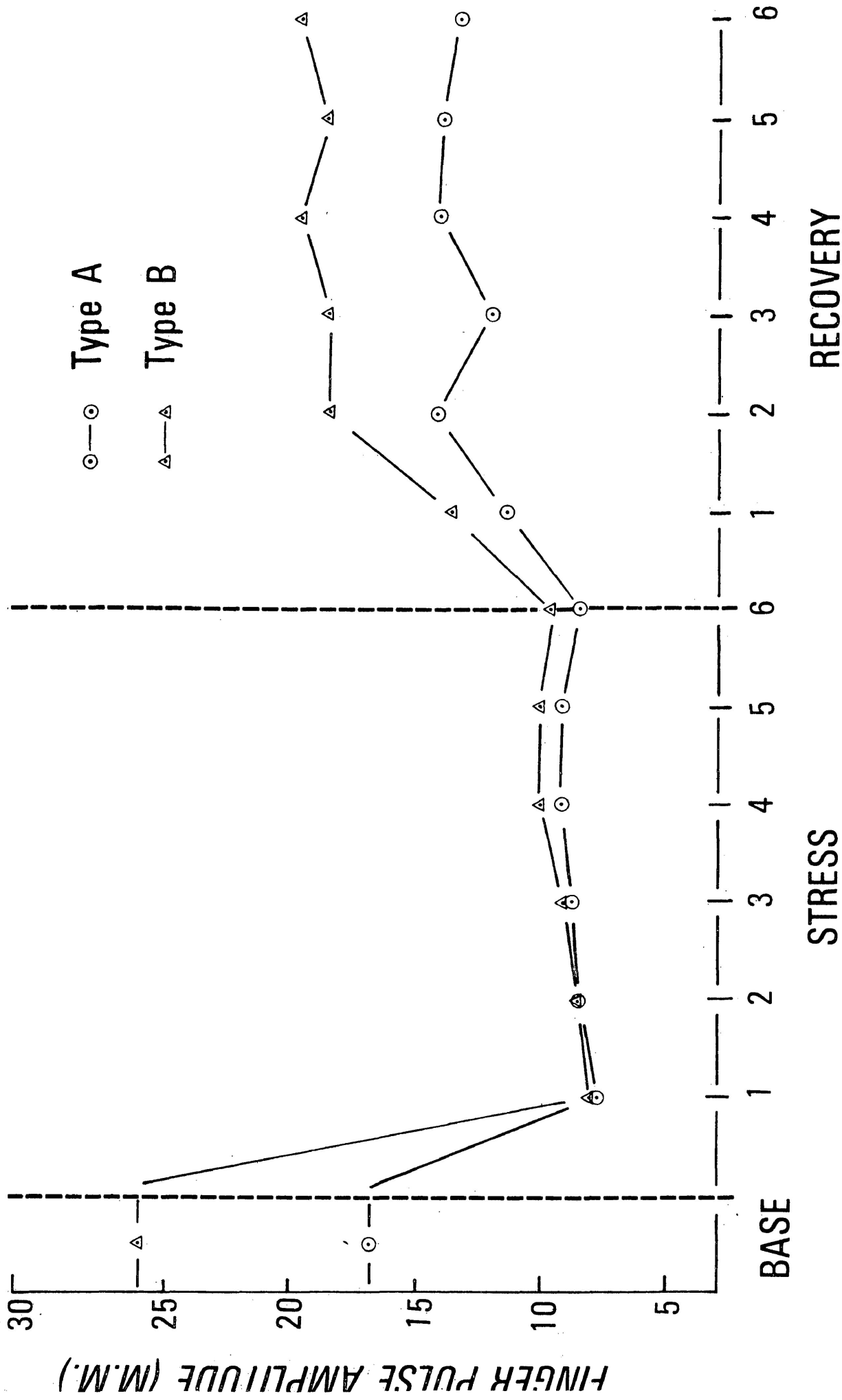


Figure 2. Finger pulse amplitude (in millimeters of pen deflection) as a function of time (in minutes) for individuals scoring above and below the median on the JAS-A/B scale.

and Type B's (JAS-A/B median split) during the three phases of the experiment.

Finger pulse amplitude scores were analysed using two different statistical procedures because neither approach alone gave a true picture of the data. Indeed, for reasons that will be outlined in the Discussion, it was concluded that the FPA results are inconclusive. The first statistical approach employed the heirarchical multiple regression technique, while the second approach correlated Type A behavior with percent change scores (cf., Van Egeren, 1979).

Multiple Regression Approach

Using mean stress FPA as the criterion variable, the proportion of variability associated with base and Type A behavior were removed in that order. Results showed that after partialling out the effects of basal scores upon stress scores, no relationship was found between any of the JAS scales and mean stress FPA. Type A's however, showed a tendency toward smaller decreases in FPA than Type B's (indicating less vasoconstriction among A's compared to B's). Basal scores accounted for 39.21% of the variability in mean stress FPA scores $F(1, 28)=18.1, P<.001$. Analyses at successive minutes of stress revealed no significant JAS effects for any of the three scales.

Using mean recovery FPA as the criterion variable, the proportion of variability associated with base FPA was removed followed by the variability associated with stress FPA and Type A behavior. Results showed that after adjusting for the effects of base and stress scores, no relationship was found between any of the JAS scales and mean recovery FPA. Base values accounted for 52% of the variance in recovery scores $F(1,28)=30.80$, $P<.001$. Stress values showed no significant association with mean recovery scores.

Analyses at successive minutes of the recovery period revealed that during the first and second minutes, hard-driving competitive individuals evidenced significantly greater FPA than non hard-driving competitive individuals (indicating significantly greater release of vasoconstrictor tone). Using the first minute recovery FPA as the criterion, and the hard-driving competitive scale of the JAS as the predictor variable, a multiple regression analysis showed that base FPA accounted for 46.6% of the variance $F(1,29)=25.3$, $P<.001$. Stress values accounted for a further 29.14% of the recovery variance $F(1,28)=29.14$, $P<.001$. JAS-H/C scores accounted for 3.55% of the unique variability $F(1,27)=4.23$, $P<.05$.

Using FPA scores during the second minute of recovery as the criterion, a similar analysis showed that base values accounted for 55.75% of the criterion variance

$F(1,29)=36.53$, $P<.001$. Stress values were associated with 7.5% of the variance $F(1,28)=48.11$, $P<.001$. Finally, JAS hard-driving competitive scores accounted for 6.5% of the unique second minute Recovery FPA variance $F(1,27)=6.00$, $P<.03$. Neither the JAS-A/B nor JAS-S/I scale was associated with FPA during any minute of the Recovery period.

Relative Percent Change Approach

Relative mean percentage change FPA is a commonly used index of photoplethysmographically derived measures of changes in vascular activity (c.f., Van Egeren, 1979a, 1979b). In the present experiment percent change FPA was calculated by dividing average values of the criterion by average values of the precriterion sample and multiplying this ratio by 100; [% Change FPA = (Criterion FPA / Base FPA) X 100] that is, precriterion values served as the baseline and were arbitrarily set at 100%. A stress FPA of 40% then, indicates a greater increase in vasoconstrictor tone (i.e., more physiologic arousal) than a stress FPA of 70%.

Correlational analyses indicated that Type B's showed a significantly greater increase in vasoconstrictor tone than their Type A counterparts; this is consistent with the trends observed using the hierarchical multiple regression technique. The left half of table 4 presents the mean relative percent changes in pulse amplitude (Stress % FPA)

Table 4. Mean relative percent change in finger pulse amplitude (measured in millimeters of pen deflection) during stress and recovery periods for individuals scoring above and below the median on each of the JAS scales.*

	<u>S T R E S S</u>	<u>R E C O V E R Y</u>
	%	%
JAS-A/B		
Type A	62.7	156
Type B	38.6	217
JAS-S/1		
High	65.3	180
Low	46.2	205
JAS-H/C		
High	61.5	176
Low	43.6	190

*n=17 for Type A's (or high scorers) and
n=15 for Type B's (or low scorers)

for individuals scoring above and below the median for each of the three JAS scales.

The JAS-A/B and JAS-H/C scales correlated significantly with stress % FPA ($r = -.46$, $P < .004$, and $r = -.40$, $P < .01$, respectively). The JAS-S/I scale was only weakly and non-significantly related to the degree of finger pulse amplitude reactivity ($r = -.27$, $P < .07$).

In regard to calculating the degree to which FPA recovered from stress, the mean stress FPA (see Table 3, middle column) served as the precriterion value from which % recovery was calculated. Correlational analyses indicate that Type B individuals evidenced a non-significant tendency for greater release of vasoconstrictor tone than their Type A counterparts; this is opposite to the results obtained using the multiple regression analysis. The mean % recovery values for individuals scoring above and below the medians for each JAS scale are presented in the right hand column of Table 4. The correlation coefficients for relative percent recovery for the JAS-A/B, JAS-S/I, and JAS-H/C scales are, $r = -.25$, $P < .09$; $r = -.02$, N.S.; $r = .02$, N.S., respectively. More detailed analyses carried out at each minute of recovery revealed that Type A individuals show significantly less % FPA recovery during the third and fourth minutes of recovery ($r = -.35$, $P < .03$, and $r = -.31$, $P < .05$, respectively).

Self-Report and Behavioral Measures

Results show that Pattern A was associated with several affective reactions. The JAS-A/B scale was significantly associated with perceived anger ($r=.435$, $P<.02$), time pressure ($r=.343$, $P<.06$), and impatience ($r=.388$, $P<.02$). The JAS-S/I scale was significantly related to anger ($r=.423$, $P<.02$), time pressure ($r=.445$, $P<.02$), impatience ($r=.445$, $P<.01$), and frustration ($r=.357$, $P<.05$). The JAS-H/C scale correlated with perceived pleasure ($r=.407$, $P<.03$).

No performance differences were found on the number of Stroop color-words attempted.

DISCUSSION

In general, the findings reported here support the hypothesis that Type A individuals experience more prolonged physiological arousal following the removal of a psychosocial stressor than their Type B counterparts. Specifically, the results showed that Pattern A subjects had significantly higher heart rates than Pattern B subjects during the first minute of the recovery period. This recovery difference was not an artifact of initial reactivity level differences, in fact, Type B's showed a greater heart rate increment to the stressor and yet still recovered more rapidly than Type A's. Furthermore, subjects

scoring high on the hard-driving competitive component of the Type A pattern were more highly aroused during recovery than were their low scoring counterparts.

The heart rate recovery results replicate the findings of a very recent study by Houston and Jorgensen (1980). These investigators found that although no differences were observed during task involvement, Type A students had significantly higher heart rates during the recovery period compared to Type B students. The present heart rate recovery finding is also consistent with the results of a study by Krantz, Schaeffer, Davia, Dembroski, MacDougall and Shaffer (1980), who found that Type A's showed a significant trend to maintain their level of heart rate arousal throughout the structured interview, while Type B's, on the other hand, showed relatively large decrements in pulse rate as the interview progressed. The present results, however, are at odds with findings reported by Glass et al., (in press) who failed to note any recovery differences between A's and B's on heart rate, systolic blood pressure, diastolic blood pressure, or plasma catecholamine level. The lack of A-B differences in their study may have been due to an insensitive index of recovery; physiologic measures were taken every two minutes throughout a 15 minute recovery period and the average of these readings served as the recovery measure. Such a global index of recovery may have failed to detect differences in the time pattern of physiologic recovery.

The finding that Type A's take longer to recover physiologically from a stressful encounter supports the hypothesis that Pattern A individuals are at higher risk for coronary heart disease because over a lifetime they may experience more frequent, more intense, and more prolonged episodes of sympathetic arousal than do their pattern B counterparts. Specifically, the present data suggest that Type A individuals may be at higher risk for coronary heart disease because of maladaptively prolonged sympathetic arousal.

When generalizing from the present results however, several issues should be addressed. First, the effect of behavior pattern upon recovery lasted for only a relatively short period of time (i.e., less than a minute). Second, the magnitude of the relationship between behavior pattern and heart rate recovery was a seemingly small one. And third, paper-and-pencil tests are not the most sensitive measures of the TABP.

With respect to the first issue, it should be recognized that the exact time course of a single episode of physiological demobilization is biologically trivial. It is the cumulative effect of repeated occurrences of slow recovery that may have deleterious consequences. Based on evidence showing that Type A's experience a significantly greater number of stressful life events than Type B's (Somes, Garrity & Marks, 1980), one could hypothesize that

the cardiovascular system of Pattern A individuals is, over the course of years, subjected to a greater total amount of recovery related strain. Also, it is quite likely that the stressors encountered in day-to-day living are significantly more intense than was the simulated naturalistic stressor employed in the present study. This being the case, one would expect that the amount of prolonged sympathetic arousal after the termination of a 'real life' stressor would be substantially larger than was observed in the present study.

The second issue which should be addressed concerns the magnitude of the behavior pattern-heart rate recovery relationship. In the Results section, the impact of Type A behavior upon the first minute heart rate recovery scores was assessed using the squared semi-partial correlation. This statistic indicated that 1.87% of the variance in raw HR recovery was uniquely associated with Type A behavior. Another, possibly more accurate measure of the impact of Type A behavior upon recovery rates is the square of the partial correlation. The partial correlation squared is the proportion of the residual recovery variability (i.e., after removing the effects of base and stress upon recovery) which is associated with Type A behavior, whereas the semi-partial correlation squared represents the proportion of the total variability in recovery heart rate that is uniquely associated with behavior pattern. Therefore, when using the partial correlation squared, one treats the variance in

recovery scores that is associated with base and stress as noise; after removing this noise, the proportion of the residual (uncontaminated) recovery heart rate variance that is associated with Type A scores is then assessed. The partial correlation squared for Type A behavior and first minute recovery heart rate is 26.89%, indicating that about 27% of the variance in residual recovery heart rate scores is predictable from Type A scores. From this perspective then, it would appear that the magnitude of the A-B-recovery relationship is fairly substantial.

One final point is that the method used to assess Type A behavior in the present experiment is a less sensitive measure of the behavior pattern than is the structured interview. Brand et al., (in press) reported that although the JAS predicted CHD morbidity and mortality in the Western Collaborative Group Study, it was a poorer predictor than the structured interview. Another study (Blumenthal et al., 1978) found that although Type A behavior as determined by the structured interview method was associated with coronary atherosclerosis, JAS scores were not. Finally, it has been shown that the JAS is a poorer predictor of challenge-induced cardiovascular arousal than the structured interview (Dembroski et al., 1978; Dembroski et al., 1979). It is quite possible then that if the present study had used the structured interview to measure Type A behavior, recovery differences between A's and B's may have been greatly enhanced.

The suggestion that Pattern A individuals are at higher risk for coronary heart disease because of harmfully prolonged cardiovascular arousal may be more true for some Type A individuals than others. In the present experiment the hard-driving competitive component of Pattern A showed a stronger (longer lasting) association with recovery than the speed and impatience dimension or overall Type A score. This pattern of findings suggests that only certain components of the behavior pattern hold pathogenic potential while others may be biologically benign. In this context, it is important to recall that Matthews et al., (1977), in a factor analysis of structured interview ratings of Type A behavior, demonstrated that the competitive-drive component of Pattern A was one of the two factors (the other was impatience) that correlated significantly with the future occurrence of CHD. These authors point out that the hard-driving competitive dimension underlying the Western Collaborative Group Study diagnostic interviews closely resembles the hard-driving competitive factor of the JAS. The Matthews et al finding is consistent with the results of three retrospective studies which reported significantly higher JAS hard-driving competitive scale scores among CHD cases compared to controls (Zyzanski et al., 1979).

The present finding that JAS hard-driving competitive scores correlated positively with perceived pleasure may also have implications for understanding the mechanisms whereby hard-driving competitive individuals are at high

risk for CHD. If one assumes that Type A's scoring high on competitive drive generally enjoy intensely competitive situations, then it would stand to reason that they may also seek out and experience a greater number of these type of situations than Type B's. Recent research has shown that Type A's do indeed experience significantly more life changes than Type B's (Somes, Garrity & Marx, 1980). Thus, the speculation that Type A's experience more frequent episodes of sympathetic arousal than Type B's may also be true.

Although there is evidence to suggest that the hard-driving competitive component of the Pattern A constellation may be relatively more pathogenic than other components, it should be noted that a few studies do not support this hypothesis. Zyzanski et al., (1976) has shown that the relationship between the hard-driving competitive scale of the JAS and coronary atherosclerosis disappears when the number of infarcts is statistically controlled for. Moreover, the hard-driving competitive scale of the JAS failed to predict the future onset of CHD in the Western Collaborative Group study (Jenkins et al., 1974).

In conclusion, much more research is needed before the relative pathogenic potential of various components of Pattern A can be determined. Future research should examine more closely the relationship between hard-driving competitiveness and coronary mortality and morbidity.

In an attempt to determine whether Type A subjects showed an initially greater peripheral vascular response than Type B subjects, JAS scores were correlated with percent change finger pulse amplitude scores (c.f., Van Egeren, 1979a, 1979b). It was found that Type B's showed a significantly greater vascular response than did their Type A counterparts. This finding replicates that of Lovallo and Pishkin (1980). In order to determine whether the greater reactivity among B's was due to their elevated resting levels, a multiple regression analysis, in which task amplitudes were adjusted for base levels, was conducted. The results of this analysis revealed that after partialling out the effects of resting levels upon reactivity, the association between Type A behavior and peripheral vascular response disappeared. A similar difference between the two analytic approaches emerged from the analysis of the recovery FPA data. The percent change analysis showed that Type A's recovered less, while the multiple regression analysis indicated the reverse pattern.

There are three aspects of the FPA data which may explain why these two analytic approaches gave different results. First, Type A's showed a significantly lower resting FPA level than Type B's, consequently measures of reactivity and recovery may have been contaminated by the large initial difference. Second, both groups seemed to reach a maximum level of vasoconstriction during task involvement (i.e., a 'basement effect' was observed). And

lastly, neither group, after six minutes of recovery, returned to their basal level ($t=4.02$, $df=30$, $p<.001$). While any one of these occurrences would have created difficulties in analysis and interpretation, the occurrence of all three precludes a meaningful examination of A-B differences in response to or recovery from the stressor. The FPA results, therefore, are inconclusive.

Nevertheless, it is interesting to speculate about the reasons for two of the findings. First, the significant resting difference may in itself be an important finding. It is unlikely that the difference was an artifact arising from group differences in skin thickness, finger size, pigmentation, or site of transducer placement. The error introduced by these variables should randomize itself evenly between the groups. A more likely explanation for resting group differences is that Type A's were more aroused during the initial adaptation period than Type B's. Goldband, Nielson and Patton (1980) found that Type A and B subjects responded with differential physiological reactivity to the administration of the JAS. Therefore, the lower finger pulse amplitude among Type A's in the present study may have been due to the reactive effects of completing the Type A inventories. It should be noted however, that resting differences in optically derived measures of finger pulse amplitude have traditionally been viewed with great distrust (Brown, 1967, p,64; Brown, 1972, p. 188), and researchers have generally not made absolute level between-groups

comparisons. Since there is no reason to suspect that Type A's and B's systematically differ on variables that affect finger pulse amplitude levels (i.e., skin thickness or pigmentation), it would seem valid and appropriate to make absolute level comparisons. Clearly, this issue should be reexamined.

A second unexpected finding is the observation that both A's and B's showed stabilized recovery FPA levels that were well below their initial resting levels. This 'baseline shift' is puzzling. Perhaps it reflects vasoconstriction due to pressure exerted by the photoplethysmographic transducer. This issue should also be examined further.

Contrary to expectation, the present stressor did not cause greater base-to-stress increases in heart rate among Type A's than Type B's. The expectation that Pattern A individuals would show greater heart rate increases than Pattern B stems from the 'positive' results of a few studies (Dembroski et al., 1977; Dembroski et al., 1978; Dembroski et al., 1979; Glass et al., Exp. 1, 1980; Manuck & Garland, 1979; Pittner & Houston, 1980; Van Egeren, 1979a). A careful review of the psychophysiologic Type A studies that have examined heart rate however, reveals that 'negative' results are far more prevalent than 'positive' results (Debacker, Kornitzer, Kittel et al., 1979; Dembroski et al., 1978; Dembroski et al., 1979b;

Frankenhauser, Lundberg & Forsman, 1979; Frankenhauser, Lundberg & Forsman, in press; Glass, 1977; Glass et al., 1980; Goldband, 1980; Houston & Jorgensen, 1980; Krantz et al., 1980; Lovallo & Pishkin, 1980; Lundberg & Forsman, 1979; MacDougall, Dembroski & Krantz, 1981; Manuck, Craft & Gold, 1978; Manuck & Garland, 1979; Price & Clarke, 1978; Sherwitz, Berton & Leventhal, 1978; Sime, Buell & Eliot, 1979; Theorell et al., 1979; van Doornen, 1979, 1980; Van Egeren, 1979b). The present results, which showed that Type B individuals tended to show higher heart rates during task involvement than Type A's, replicates findings reported by MacDougall et al., (1981). Indeed, several experiments have noted a tendency for greater cardiovascular reactivity among B's compared to A's (Goldband, 1980; Manuck et al., 1979; Pittner & Houston, 1980; Sime & Parker, 1978; Steptoe & Ross, in press; Theorell et al., 1979). The present finding that B pattern individuals were significantly more aroused than A pattern individuals during the fourth minute of stress is consistent with results reported by Steptoe and Ross, (in press), who found that British Type B students responded to a psychosocial stressor with significantly larger decreases in pulse transit time (indicating larger increases in systolic blood pressure) than Type A students.

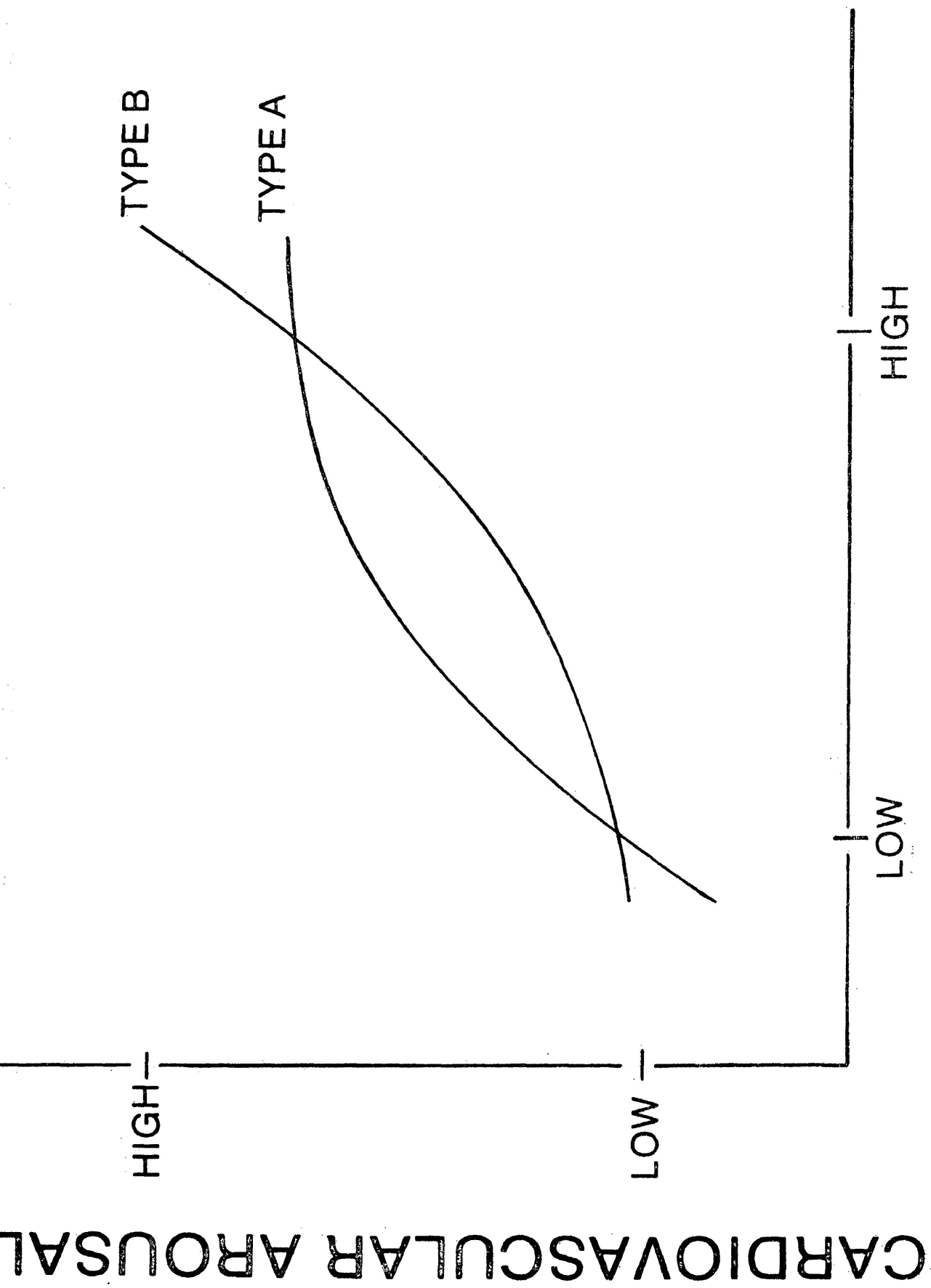
The inconsistencies in the psychophysiologic Type A literature may, in part, be explained by isolating the specific environmental circumstances that interact with particular aspects of the heterogeneous Type A pattern to produce heightened cardiovascular arousal. With respect to isolating potentially pathogenic components of Pattern A, the present results suggest that hard-driving competitiveness may be important. Only one of the three JAS scales -- the hard-driving competitive scale -- correlated positively (albeit non significantly) with the magnitude of heart rate reactivity. Furthermore, only the hard-driving competitive component of the JAS was significantly associated with mean heart rate recovery.

The results of at least two other psychophysiologic studies support the hypothesis that the hard-driving competitive aspect of Pattern A may be especially pathogenic. Dembroski et al., (1978) reported that the magnitude of heart rate and blood pressure reactivity correlated significantly with the hard-driving competitive scale of the student JAS; speed and impatience and overall Type A scores, on the other hand, showed no such

relationships. Similarly, Houston and Jorgensen (1980) found that reliable differences in systolic blood pressure reactivity occurred only between subjects scoring high and low on the hard-driving competitive dimension of the student JAS. Considering that Matthews et al., (1977) showed that competitive drive was a component of Pattern A which significantly predicted the incidence of CHD, these lab findings take on added importance.

With respect to isolating the types of situations that prompt heightened sympathetic activation among Type A's, there is evidence suggesting that reactivity differences may occur only when the environmental demand is moderately intense, and that under conditions of either extremely low or high demand A's and B's may react similarly. Figure 3 shows a graphic representation of this relationship.

Evidence to support the validity of differential response curves of A's and B's comes from investigations that generally fail to note differences in cardiovascular reactivity under conditions of low environmental demand (Dembroski, et al., 1979a; Goldband, 1980; Lott & Gatchel, 1978; Pittner & Houston, 1980; Scherwitz et al., 1978;). Indeed, two of these studies showed a tendency for blood pressure hyporesponsivity among Type A's relative to Type B's (Goldband, 1980; Pittner & Houston, 1980).



ENVIRONMENTAL DEMAND

Figure 3. Hypothesized differential physiologic response curves of Type A's and Type B's as a function of environmental demand.

Further evidence to support the model comes from studies that have failed to observe A-B reactivity differences under conditions of relatively intense environmental demand (Goldband, 1980; Manuck & Garland, 1979). In the latter experiment, A's showed greater reactivity than B's under moderately stressful conditions, however no differences were observed when the high stress condition was introduced. In addition, Glass et al., (1980) reported that when A's and B's were challenged to do well on a task, A's evidenced the same amount of arousal in a high environmental demand situation as in a moderately demanding situation (high environmental demand was a non-harassing, face-to-face competition with another individual, while moderate environmental demand was a performance challenge without face-to-face competition with another person). Type B's, on the other hand, became significantly more aroused under the high environmental demand situation than under the moderate demand condition.

Goldband (1980) has suggested a model relating physiologic response to environmental demand similar to the one shown in Figure 3. He proposed that,

"Type A subjects may be both under-responsive to low level task demands, and over-responsive to high level demands. [the present author views Goldband's 'high level demands' as

'moderate level demands'] It is possible that the Type A person has a snap-action threshold response to gradually increasing stress. At low levels of stress he may be underaroused, and remain underaroused until a "trigger point" is reached. At this level, the Type A subject may abruptly switch into an overaroused state, which may be maintained through severe stress. In contrast, the Type B subject may show a more linear relationship between increasing stress and physiological arousal." (pp.20-21).

It should be pointed out at this time that the results of some studies (e.g., Dembroski et al., 1979a) do not seem to fit well with the presently proposed model. The discrepancies may, in part, be explained by inter-experimenter variability in defining "level of environmental demand". What is 'high demand' for one experimenter may mean 'moderate demand' to another. For example, the 'high challenge' condition in the Dembroski et al., (1979a) study could be interpreted as a moderately demanding situation. At present, defining 'level of environmental demand' has a large subjective component to it. However, the possibility exists to operationalize the level of demand in terms of the magnitude of physiologic

response (eg. blood pressure or heart rate change) there by providing a rigorous test of the proposed model.

The results of Glass' study suggest that the lack of A-B reactivity differences in the present study may have been due to B's having risen to the explicit challenge of social competition. This, of course, is mere speculation because it is not possible to determine whether the opportunity to engage in social competition per se was responsible for hyperreactivity among B's. There were many aspects of the situation and task which could have differentially affected A's and B's, and these situational variables could have either magnified reactivity among B's or attenuated arousal in A's.

In general, results from the post-experimental self-report questionnaire lend support to the construct validity of the Type A concept. They also support the factorial validity of the Student JAS. The finding that overall A-B scores were significantly associated with perceived anger, time pressure, and impatience, is consistent with casual clinical observations of Friedman and Rosenman (1974), and the results of Glass' (1977) social-psychological experiments. Only the speed and impatience scale was significantly associated with perceived frustration; this scale also predicted impatience scores more accurately than did the overall A-B scale. These results indicate that the speed and impatience scale is

indeed tapping in on a different aspect of Pattern A than is the overall Type A scale. Considering the results of Glass' (1977a) 'slow down' experiment, it is not surprising that the present task, which also required a slow down, elicited more frustration, impatience and anger among individuals scoring high on the speed and impatience scale compared to individuals scoring low. The finding that hard-driving competitive scores were related only to perceived pleasure replicates the lab findings of Van Egeren (1979b). It is also consistent with the casual clinical impressions of Dunbar (1946) and Friedman (1969).

In conclusion, the present results support the hypothesis that Type A individuals may recover slower from stress arousal than Type B individuals. If we assume that maintenance of the defense alarm reaction beyond the time required for coping responses is maladaptive and promotes disease, then the present finding may have important implications for understanding the mechanisms whereby Type A behavior translates into coronary heart disease. The finding that A's recover slower in spite of initial hyporesponsivity also suggests that investigations that attempt to relate cardiovascular activity to Pattern A may bear more fruit if their focus is not tied solely to the magnitude aspect of sympathetic reactivity.

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APPENDIX 1.

TABLE 4.

Multiple regression summary table of Carit scores on mean recovery finger pulse amplitude.

Variable	R Square	RSQ Δ	Simple R	Beta	F
Base FPA	.524	.524	.724	.438	31.9***df=(1,29)
Stress FPA	.576	.052	.630	.347	3.4 df=(1,28)
Carit	.607	.031	-.368	-.195	2.1 df=(1,27)

***p<.001

TABLE 5.

Multiple regression summary table of Thurstone scores on mean recovery finger pulse amplitude.

Variable	R Square	RSQ Δ	Simple R	Beta	F
Base FPA	.524	.524	.724	.527	31.9***df=(1,29)
Stress FPA	.576	.052	.630	.302	3.4 df=(1,28)
Thurstone	.577	.001	-.145	-.035	<1 df=(1,27)

***p<.001

APPENDIX 2.

TABLE 6.

Multiple regression summary table of Carit scores on mean stress finger pulse amplitude.

Variable	R Square	RSQΔ	Simple R	Beta	F
Base FPA	.392	.392	.626	.697	18.1***df=(1,28)
Carit	.426	.034	-.051	.198	1.6 df=(1,27)

***p<.001

TABLE 7.

Multiple regression summary table of Thurstone scores on mean stress finger pulse amplitude.

Variable	R Square	RSQΔ	Simple R	Beta	F
Base FPA	.392	.392	.626	.674	18.1***df=(1,28)
Thurstone	.431	.039	.045	.202	1.9 df=(1,27)

***p<.001

APPENDIX 3.

TABLE 8.

Multiple regression summary table of Carit scores on mean recovery heart rate.

Variable	R Square	RSQΔ	Simple R	Beta	F
Base HR	.894	.894	.945	.714	236.2***df=(1,28)
Stress HR	.924	.030	.837	.294	10.7** df=(1,27)
Carit	.926	.001	.115	.038	<1 df=(1,26)

**p<.01
***p<.001

TABLE 9.

Multiple regression summary table of Thurstone scores on mean recovery heart rate.

Variable	R Square	RSQΔ	Simple R	Beta	F
Base HR	.894	.894	.946	.750	236.2***df=(1,28)
Stress HR	.924	.030	.837	.252	10.7 **df=(1,27)
Thurstone	.926	.001	-.142	-.039	<1 df=(1,26)

**p<.01
***p<.001

APPENDIX 4.

TABLE 10.

Multiple regression summary table of Carit scores on mean stress heart rate.

Variable	R Square	RSQΔ	Simple R	Beta	F
Base HR	.589	.589	.767	.808	40.1***df=(1,28)
Carit	.651	.062	-.124	-.250	4.8* df=(1,27)

*p<.05
***p<.001

TABLE 11.

Multiple regression summary table of Thurstone scores on mean stress heart rate.

Variable	R Square	RSQΔ	Simple R	Beta	F
Base HR	.589	.589	.768	.756	40.1***df=(1,28)
Thurstone	.630	.041	-.244	-.203	3.0 df=(1,27)

***p<.001

APPENDIX 5.

TABLE 12.

Heart rate (in b.p.m.) and finger pulse amplitude (in millimeters of pen deflection) during each minute of the experiment collapsed across Type A scores.*

<u>PERIOD</u>	<u>HEART RATE</u>		<u>FINGER PULSE AMPLITUDE</u>	
	MEAN	S.D.	MEAN	S.D.
Base	89.56	15.9	21.45	11.1
Stress 1	89.56	15.9	7.98	3.9
Stress 2	84.25	15.9	8.78	4.9
Stress 3	81.10	15.3	9.08	4.7
Stress 4	81.94	15.0	9.57	5.2
Stress 5	79.50	13.7	9.59	5.5
Stress 6	80.22	14.2	9.16	4.6
Stress \bar{X}	82.76	14.6	9.03	4.7
Recov 1	69.68	10.8	12.44	7.0
Recov 2	66.58	10.1	16.19	8.6
Recov 3	65.90	10.1	14.93	8.4
Recov 4	66.36	9.2	16.71	9.1
Recov 5	65.97	9.3	16.14	9.1
Recov 6	65.65	10.2	16.23	8.7
Recov \bar{X}	66.52	9.7	15.44	8.1

*n = 32

APPENDIX 6.

TABLE 13.

Mean self report scores for individuals scoring above and below the median for all Type A measures.*

ITEM # **	<u>JAS A/B</u>		<u>JAS S/1</u>		<u>JAS H/C</u>		<u>CARIT</u>		<u>THURSTONE</u>	
	A	B	Lo	Hi	Lo	Hi	A	B	A	B
1. Performance 1	1.7	1.8	1.9	1.5	1.7	1.8	2.1	1.5	2.0	1.5
2. Cold Fingers	2.0	1.5	1.8	1.6	2.3	1.6	1.9	1.7	1.7	1.9
3. Performance 2	2.1	2.0	2.1	2.0	2.1	2.0	2.0	2.1	2.1	2.0
4. Involvement	3.1	3.5	3.4	3.2	3.3	3.3	3.3	3.3	3.4	3.1
5. Stressfulness	2.7	2.9	2.9	2.7	2.6	2.9	2.6	2.9	2.7	2.9
6. Frustration	2.2	2.3	2.4	2.1	2.3	2.2	2.1	2.3	2.1	2.4
7. Heart Rate	2.5	2.7	2.6	2.5	2.7	2.6	2.4	2.8	2.4	2.8
8. Anger	1.4	1.1	1.5	1.1	1.5	1.2	1.4	1.2	1.4	1.2
9. Flushed	1.4	1.8	1.6	1.6	1.2	1.8	1.6	1.6	1.7	1.5
10. Impatience	2.1	1.5	2.1	1.5	2.1	1.7	2.1	1.6	2.0	1.6
11. Sweaty hands	2.1	2.0	2.1	1.9	1.8	2.1	2.3	1.8	2.1	1.9
12. Challenging	3.2	3.4	3.3	3.3	3.1	3.4	3.2	3.3	3.1	3.5
13. Pleasure	2.2	1.7	2.3	1.5	2.3	1.8	2.3	1.7	2.1	1.7
14. Time Pressure	2.6	1.7	2.4	1.8	2.5	2.0	2.4	1.9	2.4	1.7
15. Muscle Tension	2.1	2.0	2.2	1.9	1.8	2.2	2.2	1.9	2.2	1.9

*n = 17 for Type A's and n = 15 for Type B's.

** see Appendix for a complete description of each item.

APPENDIX 7.

TABLE 14.

Means and standard deviations for finger pulse amplitude (measured in millimeters of pen deflection) during base, stress and recovery periods for individuals scoring above and below the median on the Carit and Thurstone Type A questionnaires.*

	<u>B A S E</u>		<u>S T R E S S</u>		<u>R E C O V E R Y</u>	
	\bar{X}	S.D.	\bar{X}	S.D.	\bar{X}	S.D.
<hr/>						
CARIT						
Type A	15.7	10.2	8.7	5.5	12.8	7.7
Type B	26.5	9.5	9.2	3.9	18.0	7.8
THURSTONE						
Type A	19.3	12.0	9.2	5.2	14.1	7.2
Type B	23.9	9.8	8.8	4.2	16.9	9.0

*n = 17 for Type A's and n = 15 for Type B's.

APPENDIX 8.

TABLE 15.

Means and standard deviations for heart rate (measured in b.p.m.) during base, stress and recovery period for individuals scoring above and below the median on the Carit and Thurstone Type A questionnaires.*

	<u>B A S E</u>		<u>S T R E S S</u>		<u>R E C O V E R Y</u>	
	\bar{X}	S.D.	\bar{X}	S.D.	\bar{X}	S.D.
<hr/>						
CARIT						
Type A	65.9	10.9	79.4	14.5	66.3	9.1
Type B	65.2	9.2	85.7	14.5	66.9	10.4
THURSTONE						
Type A	66.8	10.4	79.4	13.0	65.6	8.6
Type B	65.2	9.5	86.6	15.8	67.5	10.6

*n = 17 for Type A's and n = 15 for Type B's.

APPENDIX 9.

TABLE 16.

Mean relative percent change in finger pulse amplitude (measured in millimeters of pen deflection) during stress and recovery periods for individuals scoring above and below the median on the Carit and Thurstone Type A questionnaires.*

	STRESS	RECOVERY
	%	%
CARIT		
Type A	65.3	157
Type B	39.1	209
THURSTONE		
Type A	60.0	165
Type B	41.8	204

*n = 17 for Type A's and n = 15 for Type B's.

APPENDIX 10.

TABLE 17.

Heartrate (in b.p.m.) and finger pulse amplitude (in m.m. of pen deflection) during each minute of the experiment for individuals scoring above and below the median on the JAS-A/B scale.*

PERIOD	HEART RATE				FINGER PULSE AMPLITUDE			
	TYPE A		TYPE B		TYPE A		TYPE B	
	\bar{X}	S.D.	\bar{X}	S.D.	\bar{X}	S.D.	\bar{X}	S.D.
Base	65.94	11.3	65.07	8.2	17.2	10.4	26.2	10.1
Stress 1	85.35	16.2	94.33	14.6	7.8	4.7	8.1	3.1
Stress 2	81.23	16.6	87.66	16.0	8.8	5.7	8.7	3.9
Stress 3	77.80	14.7	84.80	15.5	8.9	5.1	9.2	4.2
Stress 4	78.05	13.6	86.33	15.6	9.2	5.6	9.9	4.9
Stress 5	76.82	13.8	82.53	13.2	9.2	5.8	9.9	5.2
Stress 6	77.11	12.9	83.73	15.1	8.7	4.9	9.6	4.3
Stress \bar{X}	79.40	14.2	86.56	14.6	8.8	5.2	9.3	4.1
Recov 1	70.05	10.8	69.21	11.6	11.5	7.2	13.5	6.7
Recov 2	67.35	10.5	65.61	9.9	14.1	8.4	18.6	8.5
Recov 3	66.70	10.3	64.92	9.7	11.9	6.9	18.5	8.7
Recov 4	66.17	8.8	66.57	9.9	14.2	8.1	19.7	9.7
Recov 5	64.88	9.4	65.07	9.3	13.9	8.6	18.8	9.2
Recov 6	65.88	10.9	65.35	9.6	13.3	6.9	19.7	9.4
Recov \bar{X}	66.84	9.9	66.13	9.7	13.2	7.4	18.2	8.3

*n = 17 for Type A's and n = 15 for Type B's

APPENDIX 11.

TABLE 18.

Heart rate (in b.p.m.) and finger pulse amplitude (in m.m. of pen deflection) during each minute of the experiment for individuals scoring above and below the median on the JAS S/1 scale.*

<u>PERIOD</u>	<u>HEART RATE</u>				<u>FINGER PULSE AMPLITUDE</u>			
	<u>HIGH JAS S/1</u>		<u>LOW JAS S/1</u>		<u>HIGH JAS S/1</u>		<u>LOW JAS S/1</u>	
	\bar{X}	S.D.	\bar{X}	S.D.	\bar{X}	S.D.	\bar{X}	S.D.
Base	66.65	11.0	64.27	8.5	18.2	12.2	25.1	8.6
Stress 1	87.29	17.4	92.13	14.1	7.4	4.9	8.6	2.3
Stress 2	83.35	18.5	85.27	12.9	8.1	6.0	9.6	3.2
Stress 3	80.00	17.0	82.33	13.5	8.4	5.5	9.8	3.6
Stress 4	80.00	16.7	84.13	12.9	8.8	5.8	10.4	4.5
Stress 5	78.24	15.3	80.93	11.8	8.7	6.0	10.6	4.9
Stress 6	78.94	16.1	81.67	12.0	8.5	4.9	9.9	4.2
Stress \bar{X}	81.30	16.6	84.41	12.4	8.3	5.4	9.8	3.7
Recov 1	70.94	11.9	68.14	9.6	11.7	7.4	13.4	6.5
Recov 2	67.58	10.7	65.36	9.6	14.8	9.1	17.9	8.1
Recov 3	66.64	10.3	65.00	9.8	13.4	8.4	16.7	8.2
Recov 4	67.30	9.5	65.21	9.0	15.0	9.1	18.8	9.1
Recov 5	65.59	9.5	64.21	9.2	14.8	9.6	17.7	8.5
Recov 6	66.12	10.7	65.07	9.9	14.8	8.6	17.9	8.8
Recov \bar{X}	67.36	10.2	65.50	9.3	14.1	8.4	17.1	7.8

*n = 17 for HIGH JAS S/1 and n = 15 for LOW JAS S/1

APPENDIX 12.

TABLE 19.

Heart rate (in b.p.m.) and finger pulse amplitude (in m.m. of pen deflection) during each minute of the experiment for individuals scoring above and below the median on the JAS-H/C scale.*

PERIOD	HEART RATE				FINGER PULSE AMPLITUDE			
	HIGH JAS H/C		LOW JAS H/C		HIGH JAS H/C		LOW JAS H/C	
	\bar{X}	S.D.	\bar{X}	S.D.	\bar{X}	S.D.	\bar{X}	S.D.
Base	67.71	11.2	63.83	8.5	17.0	12.1	24.9	9.1
Stress 1	92.53	19.8	87.28	12.1	6.9	3.9	8.8	3.8
Stress 2	87.93	19.5	81.39	12.4	7.2	5.0	10.0	4.6
Stress 3	84.14	18.2	78.70	12.7	7.6	4.7	10.2	4.5
Stress 4	84.29	18.1	80.10	12.2	8.0	5.3	10.8	5.0
Stress 5	82.71	16.2	77.00	11.1	8.1	5.6	10.8	5.2
Stress 6	83.00	16.9	78.10	11.6	7.7	4.9	10.3	4.2
Stress \bar{X}	85.76	17.9	80.42	11.5	7.6	4.7	10.2	4.4
Recov 1	73.00	12.5	66.94	8.6	10.6	6.4	13.9	7.3
Recov 2	70.20	10.8	63.59	8.7	13.9	8.2	18.1	8.7
Recov 3	68.71	10.7	63.59	8.9	11.9	7.6	17.4	8.3
Recov 4	68.50	10.2	64.59	8.1	12.8	8.1	20.0	8.8
Recov 5	67.57	10.1	62.82	8.1	11.9	8.1	19.6	8.5
Recov 6	69.29	11.2	62.65	8.5	12.2	7.7	19.5	8.2
Recov \bar{X}	69.54	10.7	64.03	8.2	12.2	7.3	18.1	8.0

* n = 17 for HIGH JAS H/C and n = 15 for LOW JAS S/1

APPENDIX 13.

TABLE 20.

Heart rate (in b.p.m.) and finger pulse amplitude (in millimeters of pen deflection) during each minute of the experiment for individuals scoring above and below the median on the Carit Type A questionnaire.*

PERIOD	HEART RATE				FINGER PULSE AMPLITUDE			
	TYPE A		TYPE B		TYPE A		TYPE B	
	\bar{X}	S.D.	\bar{X}	S.D.	\bar{X}	S.D.	\bar{X}	S.D.
Base	65.87	10.9	65.24	9.2	15.7	10.2	26.5	9.5
Stress 1	84.40	15.1	94.12	15.5	7.7	5.2	8.2	2.5
Stress 2	81.87	16.2	86.35	15.9	8.7	6.2	8.9	3.6
Stress 3	78.00	14.8	83.82	15.6	8.9	5.6	9.2	3.8
Stress 4	78.00	14.1	85.40	15.2	9.2	6.0	9.9	4.7
Stress 5	77.00	14.3	81.71	13.1	9.2	6.2	10.0	5.0
Stress 6	77.40	14.4	82.70	13.9	9.0	5.0	9.3	4.4
Stress \bar{X}	79.44	14.5	85.69	14.5	8.8	5.5	9.2	3.9
Recov 1	69.87	10.4	69.50	11.5	11.2	7.7	13.6	6.2
Recov 2	66.60	9.8	66.56	10.7	13.4	8.9	18.8	7.7
Recov 3	65.93	9.6	65.88	10.6	11.5	7.3	18.1	8.3
Recov 4	65.93	8.0	66.80	10.4	13.6	8.4	19.6	9.1
Recov 5	64.50	8.4	65.40	10.2	13.6	8.9	18.6	8.8
Recov 6	64.93	10.1	66.30	10.6	13.2	7.2	19.0	9.2
Recov \bar{X}	66.29	9.1	66.74	10.4	12.8	7.7	18.0	7.8

*n = 17 for Type A's and n = 15 for Type B's.

APPENDIX 14.

TABLE 21.

Heart rate (in b.p.m.) and finger pulse amplitude (in m.m. of pen deflection) during each minute of the experiment for individuals scoring above and below the median on the Thurstone Type A questionnaire.*

PERIOD	HEART RATE				FINGER PULSE AMPLITUDE			
	TYPE A		TYPE B		TYPE A		TYPE B	
	\bar{X}	S.D.	\bar{X}	S.D.	\bar{X}	S.D.	\bar{X}	S.D.
BASE	65.82	10.4	65.20	9.4	19.4	12.0	23.8	9.7
Stress 1	85.30	14.0	94.40	16.9	8.0	4.9	7.9	2.8
Stress 2	81.53	14.5	87.33	17.4	9.0	5.8	8.5	3.8
Stress 3	77.41	13.4	85.27	16.6	9.3	5.3	8.8	4.0
Stress 4	78.01	12.7	86.01	16.6	10.0	5.6	9.1	4.9
Stress 5	76.53	12.8	82.89	14.1	9.6	5.7	9.4	5.4
Stress 6	77.29	12.8	83.53	15.3	9.4	4.6	8.9	4.7
Stress \bar{X}	79.39	13.1	86.58	15.8	9.2	5.2	8.8	4.1
Recov 1	69.31	10.1	70.07	11.9	12.0	7.4	13.0	6.7
Recov 2	65.69	9.6	67.53	10.9	14.6	8.5	17.9	8.8
Recov 3	65.19	9.4	66.67	10.7	12.9	7.0	17.1	9.4
Recov 4	65.50	7.7	67.26	10.7	15.2	7.6	18.3	10.5
Recov 5	63.81	8.3	66.20	10.3	15.0	8.1	17.3	10.1
Recov 6	63.94	9.9	67.47	10.5	14.5	6.6	18.0	10.3
Recov \bar{X}	65.57	8.9	67.53	10.6	14.1	7.2	16.9	8.9

*n = 17 for Type A's and n = 15 for Type B's.

APPENDIX 15.

Self Report Questionnaire

CHECK THE BOX WHICH BEST DESCRIBES HOW YOU FELT WHILE PERFORMING THE COLOR-WORD TASK. BE AS ACCURATE AS POSSIBLE.	not at all	some-what	moder-ately	very much so
) Do you think your performance was better than other students? _____				
) Did your fingers become cold during the test?				
) If you were given a second chance on the task, how much better would your performance be?				
) How involved or engaged were you in the task?				
) Generally, how stressful did you find it?				
) Did you feel frustrated? _____				
) How much did your heartrate increase?				
) Did you feel angry? _____				
) Did your face become flushed? _____				
) Did you feel impatient? _____				
) How sweaty did your hands become?				
) How challenging did you find the task?				
) Did you find the task pleasurable? _____				
) How "time pressured" did you feel?				
) Did your muscles become tense?				

How old are you? _____

Do you smoke? _____ If so, how many per day usually? _____

Are you presently under the influence of any drug? _____
If so, what drug?

How many cups of coffee have you had today? _____

In the hour before coming to this experiment did you engage in any vigorous physical exercise?

APPENDIX 17.

Instructions:

Below are 20 questions which might describe you. For each question place an "X" in one of the five brackets to the right of the question which best describes you. For example, if you are always more restless and fidgety than most people, mark "X" in the bracket beneath "always." Please check the way you think you are, not the way you would like to be. BE SURE TO ANSWER ALL 20 QUESTIONS.

	<u>Always</u>	<u>Fre-</u> <u>quently</u>	<u>Some-</u> <u>times</u>	<u>Infre-</u> <u>quently</u>	<u>Never</u>
1 Are you more restless and fidgety than most people?	()	()	()	()	()
2 Do you work quickly and energetically?	()	()	()	()	()
3 In conversation, do you gesture with hands and head?	()	()	()	()	()
4 Do you drive a car rather fast?	()	()	()	()	()
5 Are you rather deliberate in telephone conversations?	()	()	()	()	()
6 Are you in a hurry?	()	()	()	()	()
7 Do you eat rapidly even when there is plenty of time?	()	()	()	()	()
8 As a boy (or girl), did you prefer work where you could move around?	()	()	()	()	()
9 Do people consider you to be rather quiet?	()	()	()	()	()
10 Do you talk more slowly than most people?	()	()	()	()	()
11 Do you work fast?	()	()	()	()	()
12 Do you speak louder than most people?	()	()	()	()	()
13 Do you prefer to linger over a meal and enjoy it?	()	()	()	()	()
14 Do you like work that is slow and deliberate?	()	()	()	()	()
15 Do you let a problem work itself out by waiting?	()	()	()	()	()
16 Do you like to drive a car rather fast when there is no speed limit?	()	()	()	()	()
17 Is your handwriting rather fast?	()	()	()	()	()
18 Do you work slowly and leisurely?	()	()	()	()	()
19 Do you try to persuade others to your point of view?	()	()	()	()	()
20 Do you walk faster than most people?	()	()	()	()	()

APPENDIX 18
THE JENKINS ACTIVITY SURVEY
Form T

Medical research is trying to determine how life style may influence the health of people. This survey is part of such a research effort.

Please answer the questions on the following pages by marking the answers that are true for you. Each person is different, so there are no "right" or "wrong" answers. Of course, all you tell is strictly confidential--to be seen only by the research team. Do not ask anyone else about how to reply to the items. It is your personal opinion that we want. Please use the answer sheet provided to record your responses to the items in this booklet.

Your assistance will be greatly appreciated.

For each of the following items, please circle the number of the ONE best answer on your answer sheet.

1. Do you ever have trouble finding time to get your hair cut or styled?
 1. Never
 2. Occasionally
 3. Almost always
2. Does college "stir you into action"?
 1. Less often than most college students
 2. About Average
 3. More often than most college students
3. Is your everyday life filled mostly by
 1. Problems needing solution
 2. Challenges needing to be met
 3. A rather predictable routine of events
 4. Not enough things to keep me interested or busy
4. Some people live a calm, predictable life. Others find themselves often facing unexpected changes, frequent interruptions, inconveniences or "things going wrong." How often are you faced with these minor (or major) annoyances or frustrations?
 1. Several times a day
 2. About once a day
 3. A few times a week
 4. Once a week
 5. Once a month or less
5. When you are under pressure or stress, do you usually:
 1. Do something about it immediately
 2. Plan carefully before taking any action
6. Ordinarily, how rapidly do you eat?
 1. I'm usually the first one finished.
 2. I eat a little faster than average.
 3. I eat at about the same speed as most people.
 4. I eat more slowly than most people.
7. Has your spouse or some friend ever told you that you eat too fast?
 1. Yes often
 2. Yes, once or twice
 3. No, no one has told me this

8. How often do you find yourself doing more than one thing at a time, such as working while eating, reading while dressing, figuring out problems while driving?
1. I do two things at once whenever practical.
 2. I do this only when I'm short of time.
 3. I rarely or never do more than one thing at a time.
9. When you listen to someone talking, and this person takes too long to come to the point, do you feel like hurrying him along?
1. Frequently
 2. Occasionally
 3. Almost never
10. How often do you actually "put words in his mouth" in order to speed things up?
1. Frequently
 2. Occasionally
 3. Almost never
11. If you tell your spouse or a friend that you will meet them somewhere at a definite time, how often do you arrive late?
1. Once in a while
 2. Rarely
 3. I am never late.
12. Do you find yourself hurrying to get places even when there is plenty of time?
1. Often
 2. Occasionally
 3. Rarely or never
13. Suppose you are to meet someone at a public place (street corner, building lobby, restaurant) and the other person is already 10 minutes late. Will you
1. Sit and wait?
 2. Walk about while waiting?
 3. Usually carry some reading matter or writing paper so you can get something done while waiting?
14. When you have to "wait in line," such as at a restaurant, a store, or the post office, do you
1. Accept it calmly?
 2. Feel impatient but do not show it?
 3. Feel so impatient that someone watching could tell you were restless?
 4. Refuse to wait in line, and find ways to avoid such delays?
15. When you play games with young children about 10 years old, how often do you purposely let them win?
1. Most of the time
 2. Half of the time
 3. Only occasionally
 4. Never
16. Do most people consider you to be
1. Definitely hard-driving and competitive?
 2. Probably hard-driving and competitive?
 3. Probably more relaxed and easy going?
 4. Definitely more relaxed and easy going?
17. Nowadays, do you consider yourself to be
1. Definitely hard-driving and competitive?
 2. Probably hard-driving and competitive?
 3. Probably more relaxed and easy going?
 4. Definitely more relaxed and easy going?

18. How would your spouse (or closest friend) rate you?
1. Definitely hard-driving and competitive?
 2. Probably hard-driving and competitive?
 3. Probably relaxed and easy going?
 4. Definitely relaxed and easy going?
19. How would your spouse (or best friend) rate your general level of activity?
1. Too slow. Should be more active.
 2. About average. Is busy much of the time.
 3. Too active. Needs to slow down.
20. Would people who know you well agree that you take your work too seriously?
1. Definitely Yes
 2. Probably Yes
 3. Probably no
 4. Definitely No
21. Would people who know you well agree that you have less energy than most people?
1. Definitely Yes
 2. Probably Yes
 3. Probably No
 4. Definitely No
22. Would people who know you well agree that you tend to get irritated easily?
1. Definitely Yes
 2. Probably Yes
 3. Probably No
 4. Definitely No
23. Would people who know you well agree that you tend to do most things in a hurry?
1. Definitely Yes
 2. Probably Yes
 3. Probably No
 4. Definitely No
24. Would people who know you well agree that you enjoy "a contest" (competition) and try hard to win?
1. Definitely Yes
 2. Probably Yes
 3. Probably No
 4. Definitely No
25. Would people who know you well agree that you get a lot of fun out of your life?
1. Definitely Yes
 2. Probably Yes
 3. Probably No
 4. Definitely No
26. How was your "temper" when you were younger?
1. Fiery and hard to control.
 2. Strong, but controllable.
 3. No problem.
 4. I almost never got angry.
27. How is your "temper" nowadays?
1. Fiery and hard to control.
 2. Strong, but controllable.
 3. No problem.
 4. I almost never get angry.
28. When you are in the midst of studying and someone interrupts you, how do you usually feel inside?
1. I feel O.K. because I work better after an occasional break.
 2. I feel only mildly annoyed.
 3. I really feel irritated because most such interruptions are unnecessary.

(Remember, the answers on these Questionnaires are confidential information and will not be revealed to officials of your school.)

29. How often are there deadlines in your courses? (If deadlines occur irregularly, please circle the closest answer below.)
1. Daily or more often.
 2. Weekly.
 3. Monthly.
 4. Never
30. Do these deadlines usually
1. Carry minor pressure because of their routine nature?
 2. Carry considerable pressure, since delay would upset things a great deal?
31. Do you ever set deadlines or quotas for yourself in courses or other things?
1. No
 2. Yes, but only occasionally
 3. Yes, once per week or more often.
32. When you have to work against a deadline, is the quality of your work
1. Better?
 2. Worse?
 3. The same? (Pressure makes no difference)
33. In school do you ever keep two projects moving forward at the same time by shifting back and forth rapidly from one to the other?
1. No, never.
 2. Yes, but only in emergencies.
 3. Yes, regularly.
34. Do you maintain a regular study schedule during vacations such as Thanksgiving, Christmas, and Easter?
1. Yes
 2. No
 3. Sometimes
35. How often do you bring your work home with you at night or study materials related to your courses?
1. Rarely or never.
 2. Once a week or less often.
 3. More than once a week.
36. How often do you go to the school when it is officially closed (such as nights or weekends)? If this is not possible, circle 0.
1. Rarely or never.
 2. Occasionally (less than once a week).
 3. Once or more a week.
37. When you find yourself getting tired while studying, do you usually
1. Slow down for a while until your strength comes back.
 2. Keep pushing yourself at the same pace in spite of the tiredness.
38. When you are in a group, do the other people tend to look to you to provide leadership?
1. Rarely.
 2. About as often as they look to others.
 3. More often than they look to others.
39. Do you make yourself written lists of "things to do" to help you remember what needs to be done?
1. Never
 2. Occasionally
 3. Frequently

IN EACH OF THE FOLLOWING QUESTIONS, PLEASE COMPARE YOURSELF WITH THE AVERAGE STUDENT AT YOUR SCHOOL. PLEASE CIRCLE THE MOST ACCURATE DESCRIPTION.

40. In amount of effort put forth, I give

- 1. Much more effort
- 2. A little more effort
- 3. A little less effort
- 4. Much less effort

41. In sense of responsibility, I am

- 1. Much more responsible
- 2. A little more responsible
- 3. A little less responsible
- 4. Much less responsible

42. I find it necessary to hurry

- 1. Much more of the time
- 2. A little more of the time
- 3. A little less of the time
- 4. Much less of the time

43. In being precise (careful about detail), I am

- 1. Much more precise
- 2. A little more precise
- 3. A little less precise
- 4. Much less precise

44. I approach life in general

- 1. Much more seriously
- 2. A little more seriously
- 3. A little less seriously
- 4. Much less seriously

APPENDIX 19.

DEFINITION OF TERMS

1. Angina pectoris: a pain in the chest caused by temporary shortage of blood to the heart muscle. It may be precipitated by emotional or physical stress.

2. Atherosclerosis: the lesion directly responsible for coronary artery or coronary heart disease. It is the build-up of cholesterol and calcium deposits on the walls of coronary arteries that eventually form into lumps (plaques) that occlude the vessels. When these plaques decay or tear away from the arterial wall, they usually form clots that totally close off the lumen of the already narrowed artery.

3. Catecholamines: a group of chemicals in the body that are involved in stimulating the sympathetic nervous system. Although one of the catecholamines (norepinephrine) is a post ganglionic neurotransmitter in the sympathetic branch of the autonomic nervous system, it is also secreted into the blood by the adrenal medulla, which is an endocrine gland. In times of "flight or fight" (sympathetic nervous system arousal), the adrenal medulla releases both norepinephrine ('stress hormones') and epinephrine into the blood to prepare the organism for an "emergency". Excessive or prolonged levels of catecholamines in the blood are thought to be involved in the pathogenesis of coronary artery disease (Glass, 1977).

4. Cholesterol: a highly complex substance found in the blood and arteries. When the blood serum cholesterol rises above the basal level, a potentially pathogenic condition known as hypercholesterolemia exists. A high level of blood cholesterol has traditionally been thought of as the prime factor in the etiology of coronary artery and coronary heart disease. Psychosocial stressors are capable of causing significant increases in serum cholesterol.

5. Coronary artery disease: a symptomless disorder in which one or more of the coronary arteries is partially or totally occluded by one or more atherosclerotic plaques.

6. Coronary heart disease: when coronary artery disease has progressed to such a degree that coronary symptoms appear, coronary heart disease is said to exist. It comes in many forms: angina pectoris, myocardial infarction, congestive heart failure are just a few.

7. Epidemiology: a branch of medical or social science that studies the relationships of various factors to the frequency (incidence) and distribution (prevalence) of various diseases. Two types of investigations are carried out: retrospective and prospective. Retrospective studies are concerned with distribution or prevalence of diseases in various populations at a single point in time. Prospective studies are not concerned with the absolute level or

prevalence of a disease, but rather focus on the rate of new occurrences of a disease (incidence). Prospective studies explore morbidity and mortality rates.

8. Myocardial infarction: an area of necrosis (dead cells) in the heart muscle which is usually caused by the occlusion of two or more coronary arteries. Synonyms include: heart attack, "a coronary", coronary occlusion, and coronary thrombosis.

9. Pathogenesis: the development of a disease or disorder.

10. Plaque, arterial or atherosclerotic: a scarlike clump in an artery containing cholesterol fat and calcium. Plaques partially occlude the lumen, but are not extremely dangerous until they begin to decay and rupture, at which point the clot generating elements in the plaque rapidly close off the lumen of the already narrowed coronary artery. Emotional or physical stress may be instrumental in a plaque's decay.

11. Stress (Selye): refers to a dynamic state within an organism which arises from a demand for adaption. It is a phylogenetically old adaption pattern that is designed to prepare the organism for physical activity (flight or fight). When the stress response occurs in the absence of concomitant musculo-skeletal activity, a potentially

pathogenic condition is thought to exist (Bove, 1977). This dissociation between the autonomic-hormonal and musculo-skeletal systems may be a prime mechanism in the pathogenesis of CHD.

12. Stressor: any physical or non physical stimuli which evokes the stress response.

13. Type A Behavior Pattern: is considered to be an overt behavioral syndrome or style of living characterized by extremes of competitiveness, striving for achievement, aggressiveness (sometimes repressed), haste, impatience, and job involvement. It is thought to be a style of coping displayed by individuals who have an exaggerated need to master or control their world. The Type A behavior pattern emerges in certain predisposed "personality" types when they are confronted with certain types of challenging or competitive situations.

APPENDIX 20.

Verbal instructions Given to Subjects

"Thank you for filling out the questionnaires. We will now move on to the next part of the experiment. This funny looking machine is called a polygraph, and I'm going to use it to record your heartrate. It is completely harmless, and hooking you up is simple and painless and will only take a minute, so relax. (after subject is hooked up) Ok, for the next five minutes or so I want you to try and relax and become as calm as possible. Close your eyes and just relax. If you feel that it might help you to relax you might try to imagine a tranquil, relaxing scene. I'll tell you when to open your eyes again, now just relax. (after six minutes of relaxation) Please look straight ahead and open your eyes. In a minute you will be presented with a difficult intellectual task. In order to achieve a high score you will have to think quickly and really conentrate. When you finish the task I will compare your score with the scores of other students and tell you how you scored compared to most students, and what percentage of the students did worse and better than you. (explain task using example test stimuli) This chart contains practice examples of the intellectual task that you will next have to do. Your job is to look at each of the words listed and say out loud the correct color

of ink. The correct response to the first word in the list is blue. Do you understand? Read through the rest of the examples. (explain procedure for the real test stimuli) You will now be tested on how well you can do on the actual task. On the next wall chart you will find lists of words. The words are in columns. I want you to start reading on the left most column and read downwards. When you finish the first column go on to the next one. When you finish the entire chart, start over. If you make a mistake you must correct it before continuing. For every mistake you make, five points will be subtracted from your final score. There is a time limit of six minutes, therefore it is very important that you concentrate and read quickly if you are to get a high score. Remember, your task is to say out loud the correct color of ink. Any questions? Are you ready? Close your eyes while I remove the chart cover, and when I tell you to open your eyes, begin reading.

APPENDIX 21.

Table 22. Correlations (r)*, means, and standard deviations of Type A measures (N = 32).

Variable	1.	2.	3.	4.	5.
1. JAS - A / B		.775	.575	.646	.650
2. JAS - S / 1			.510	.545	.581
3. JAS - H / C				.302	.266
4. CARIT.					.500
5. THURS					
MEAN	7.53	17.24	11.12	66.84	65.62
S.D.	3.15	6.00	4.27	8.91	7.26

* $P < .01$ when $r > .40$; $P = N.S.$ when $r < .40$