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Heart Rate Reactivity to Mental Arithmetic: The Influence of Baseline Protocols and Task Demand Characteristics.

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A Thesis

In

The Department

of

Psychology

Presented in Partial Fulfillment of the Requirements
for the Degree of Doctor of Philosophy
at Concordia University,
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Abstract

Heart Rate Reactivity to Mental Arithmetic:
The Influence of Baseline Protocols and Task
Demand Characteristics.

Thomas G. Brown, Ph.D.
Concordia University, 1990

There is increasing concern over the impact methodological variables may exert on observations of cardiac response to psychological stress. Laboratories use a variety of stress-inducing tasks and experimental protocols. The present study examined the influence of a number of methodological variables on heart rate (HR) responses to a commonly employed psychological stressor, mental arithmetic (MA). The reliability of two types of baseline measurement, the clarification of physical versus psychological sources of reactivity in MA, and the reliability of HR to MA were examined. Three studies, employing males aged 20-40 years, were conducted. In the first, subjects underwent testing twice, approximately one month apart. In each session, HR was monitored over a 10-minute period in which no stress was anticipated. In the second, subjects were monitored for HR during two equivalent and counterbalanced MA tasks, one which required verbal answers and one in which verbal answers were not needed. In the third study, subjects performed the same two types of MA tasks as above twice, approximately a month apart. Resting baseline HR recorded when no stressors were presented was stable and

significantly correlated over 10 minute periods ($r = .74$ and .89 for Sessions 1 and 2 respectively) as well as during repeated testing over a one month period ($r = .65$). These results suggest that baseline HR taken in a separate no-stress session are reliable over both short and long periods of time. HR to non-verbal MA was uniformly lower than HR to verbal MA ($p < .05$) throughout the duration of the tasks. These results corroborate recent reports that physical task demands can contribute significantly to cardiac responding with MA problems. Finally, when HR to both verbal and non-verbal MA was analysed for reliability over a one month period, only HR to verbal MA remained stable over one month. Thus, verbal responding to MA may contribute to the stability of HR responses to these tasks. Overall, these findings suggest that the magnitude of cardiovascular reactivity to psychological stress may be influenced by a number of methodological factors in the laboratory, and indicate that a clearer understanding of the role of behavior on heart health requires further attention to such concerns.

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To my beloved companion, Dominique, who kept me afloat, and whose only compensation was often her own further sacrifice; and to my family, who were there for me and understood. My love and thanks.

Finally, I dedicate this work to the memory of my father, whose survival and achievement during a particularly dark period of history is an ongoing inspiration to me. If I have one regret it is that he is not here to share in this comparatively modest accomplishment.

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Introduction

The study of changes in cardiovascular function to psychologically stressful tasks (i.e., tasks of varying degrees of conceptual, cognitive and motor complexity) is a popular laboratory paradigm in psychophysiological research. However the copious literature emerging from this inquiry, though reflecting broad experimental interest, has come under increasing criticism for a number of methodological shortcomings (Weiss, 1986; Matthews, Weiss & Detre, 1984; Matthews, 1986). While much of the research has focussed on the role of behavior in cardiovascular pathogenesis, clarification of precisely how stress-inducing laboratory tasks in and of themselves influence cardiovascular functioning has lagged. Furthermore, the experimental protocols, tasks and data analysis strategies vary greatly from study to study, with little apparent concern for the impact these differences may have on the findings. One practical consequence of this lack of uniformity has been that comparative analysis and synthesis of data, as well as systematic replication of studies, are increasingly problematic. As further understanding of the behavioral correlates of cardiovascular health may hinge on these methodological and parametric issues, empirical inquiry towards their clarification is warranted.

Physical Determinants of Heart Rate

Many questions remain unclarified as to the manner in which psychological stress influences cardiovascular functioning. In contrast, the manner in which physical stress influences cardiovascular functioning has been well-documented (Astrand & Rhyming, 1954; Mathews & Fox, 1976; Nadel, 1985). Of the many indices of cardiovascular function, heart rate has shown itself to be a meaningful and reliable indicator of both transient and chronic cardiovascular adaptations to increased physical demands. With a bout of exercise for example, heart rate increases rapidly, mediated through enhanced sympathetic and inhibited parasympathetic activation at the sinoatrial node of the heart. In response to a submaximal intensity of exercise lasting several minutes, the increase in heart rate and stroke volume achieve a cardiac output that precisely fulfills the physiologic demand of that workload. The increase in blood flow serves to provide adequate oxygen and nutrients to the exercising muscles while carrying away carbon dioxide, heat and other catabolites of organic work production.

Graduated linear loading in submaximal workloads result in systematic increases in heart rate up to a maximum level regardless of the modality of work. Specific workloads in bicycle ergometry are achieved by regulating both the resistance on a bicycle flywheel and pedalling cadence. The resultant measure of work is expressed as kilograms of resistance over meters travelled per minute (i.e., kg-meters/min). A 30-year old individual of high aerobic fitness might be expected to achieve a heart rate of approximately 120 beats per minute at 900 kg-meters/min (i.e., 3 kg resistance x 300 meters / minute) after approximately five minutes of exercise at this fairly intense workload. The increase in heart rate with exercise reflects the axiomatic relationship between cardiac output and the heightened physiologic effort of the organism, measured as oxygen utilization, or VO_2.

Maximal heart rate is determined by a combination of individual factors, such as age, aerobic fitness and genetic attributes. A typical maximal heart rate for a 30-year old male would be approximately 190 beats/min. Once heart rate plateaus at its maximum frequency, exhaustion soon follows (Mathews & Fox, 1976). This marks the cardiovascular system's failure to meet the requisite physiologic demand to maintain the current intensity of work.

Aerobic power is recognized as a definitive indicator of physical fitness (Nadel, 1985). This reflects the body's maximal ability to consume oxygen, and consequently represents the limiting factor in the amount of work that can be produced in a given period of time. A commonly used indirect method of aerobic power measurement employs observations of heart rate with precise, graduated submaximal uploading of work output. With heart rates above 120 beats per minute, when the heart is considered to be primarily under the influence of sympathetic innervation, the slope of the regression line describing the relationship between heart rate and work provides an indirect estimate of individual fitness (Astrand & Ryhming, 1954). Steep increases in heart rate indicate lower aerobic power, while attenuated heart rate increases reflect greater capacity. The development of nomograms based upon the heart rate responses to work, estimated maximum heart rates by age, and direct aerobic power measurement using large population samples, permits the calculation of estimated maximal oxygen consumption, a quantity that may be expressed in relative units of milliliters of oxygen consumed per minute per kilogram of body weight (Berne & Levy, 1977; Nadel, 1985).

Physical Versus Psychological Determinants of Heart Rate

Changes in heart rate have also been observed in the apparent absence of increased metabolic demands. Tasks involving some manner of conceptual, cognitive and/or perceptual-motor challenge and usually perceived as subjectively arousing, have also been seen to alter physiological functioning. Physiological alterations to these tasks, variously described as 'psychologically stressful', 'stress-inducing' and 'stressor' tasks, are often pervasive across several autonomic systems, and may mimic both qualitatively and quantitatively those seen with exercise. The change in autonomic indices from a stable, low level resting condition with task presentation has been coined "psychophysiological reactivity".

Early explanations of reactivity seen with stress-inducing laboratory tasks were based on the physical determinants of autonomic activity. For example, when increases in heart rate were observed when rapid verbal solutions to challenging arithmetic equations were required, it was hypothesized that these alterations resulted from the tensing up of muscles occurring under these circumstances (Hassett, 1978). As in exercise, it was argued, increases in heart rate reflected active muscles and their need for increased cardiac output. As further support of this hypothesis, other tasks requiring more quiet attentiveness, and therefore less physical activity, were observed to result in a lowering of heart rate.

The assumption that reactivity seen with psychological stress was physically determined persisted until a little more than a decade ago. Since that time, consistent observations of an 'uncoupling' of heart rate from direct measures of physical effort (i.e., VO_2) suggested that reactivity seen with psychological stress was not sufficiently explained by physiological factors alone (Obrist, 1976; Turner & Carroll, 1985). However, the specific attributes of these stressful tasks that produce psychophysiological reactivity were unclear at that time, and continue to be up until the present.

Both qualitative and quantitative dimensions of the stressful tasks have been posited as influencing the cardiovascular expressions of reactivity. To explain observed differences in reactivity, a qualitative distinction between tasks invoking either active-coping or passive-coping has been proposed (Obrist et al., 1978).

Active-coping is proposed as occuring when aversive consequences of task performance can be influenced by the individual. Difficult mathematical, conceptual or verbal tasks in which subjects in a laboratory setting are motivated to demonstrate competence in close proximity to an experimenter (e.g., mental arithmetic, competitive video games or public speaking) are examples of this type of task. In contrast, passive coping occurs with tasks requiring only awareness and inactive toleration of disturbing perceptual stimuli, as with exposure to audiovisual presentations of grisly accident scenes or pornographic films.

These classifications have also been hypothesized as paralleling the strength of sympathetic versus parasympathetic mediation of cardiovascular functioning (Obrist et al., 1974). Tasks requiring active-coping have reliably produced increases in heart rate and systolic blood pressure, suggesting a predominance of sympathetic, beta-adrenergic influence on the cardiovascular system (Bunnell, 1982). By contrast, passive-coping tasks, such as viewing accident scenes, have been hypothesized as resulting in less beta-adrenergic influence but increased parasympathetic, alpha-adrenergic tone. Thus, exposure to these tasks has been observed to result in greater increases in diastolic blood pressure than seen with tasks involving active-coping (Schneiderman & Pickering, 1985).

There is empirical support for this hypothesis. In one study (Light & Obrist, 1980 a), pairs of subjects were exposed to a reaction time task. For one subject in each of the pairs, avoidance of shock was contingent on performance. The other subject was exposed simultaneously to the same task as the first, but was unable to avoid shock through individual performance. Instead, this subject was shocked whenever the first received shock. Whereas both subjects received shock with the same frequency and temporal spacing, heart rate and systolic blood pressure reactivity was seen to be greater in subjects who could actively avoid shock as compared to their 'yoked' counterparts who could not.

Further support for the importance of the sympathetic mediation of heart rate accompanying active-coping comes from several studies that have employed beta-adrenergic antagonists. For example, administration of the beta blocker propranolol has been found to attenuate heart rate responses significantly when subjects were exposed to active-coping type tasks (Light, 1981; Fredrickson et al., 1985; Sherwood, Allen, Obrist & Langer, 1986). In contrast, administration of propranolol resulted in no such attenuation when passive-coping tasks were employed.

While this classification seeks to differentiate tasks meaningfully into two broad categories on the basis of both their psychological demands and physiologic responses, there is, however, much variability within these categories. For example, different active-coping tasks are seen to result in dissimilar magnitudes of reactivity. Several task characteristics have been posited to explain these differences. These include the level of task difficulty (Light & Obrist, 1983; Carroll, Turner & Hellawell, 1986), the degree of aversive outcome (Perkins, 1984), the number of task presentations (Myrtek & Spital, 1986) and the physical effort required for task performance (Krantz, Manuck & Wing, 1986).

This latter characteristic is methodologically of particular importance. When autonomic changes with exposure to stress are associated with behavioral processes, one underlying assumption may be that the psychological processes aroused by the task, and not the task's physical demand characteristics, are the primary determinants of reactivity. However, active-coping tasks contrived in the laboratory have invariably involved some form of somatic involvement as an aspect of responding, from verbalization to hand or arm gestures. It is therefore important to assess the degree to which the physical and/or psychological task demands contribute to overall measures of reactivity.

There is data from various research literatures that bear on this issue. Speech is a common component of stressful tasks employed in the laboratory. In research examining coronary risk behavior patterns, the physical effort of speech has been examined in the context of its impact on cardiovascular reactivity. Simple verbalization of a text has been found to increase heart rate significantly (Thomas et al., 1984; Lynch et al., 1982; Long et al., 1982), while increasing the speed of verbalization within subjects has resulted in greater increases in heart rate (Friedmann et al., 1982; Lynch, 1985). While providing support for the contribution of verbalization to cardiac reactivity, these studies fail to differentiate clearly between the psychological and physical components of stressful tasks.

Brown et al. (1988) specifically examined the importance of the physical demands of a psychologically stressful task in relation to the overall expression of heart rate reactivity. A significant increase in heart rate was found to occur with simple repetition of numbers in place of the responses to mental arithmetic, without the psychological challenge or cognitive effort involved in generating solutions to the problems. Furthermore, reactivity associated with the speech motor activity involved in responding to mental arithmetic amounted to approximately 50 percent of the increases in the heart rate seen with exposure to mental arithmetic with verbalization of responses in the same subjects.

In other research (Linden, 1986), changes in heart rate were observed in the laboratory under three conditions: reading neutral text aloud; performing mental arithmetic but without vocalization (quiet); and performing mental arithmetic and vocalizing the responses (aloud). Similar heart rate change occurred between the reading aloud and mental arithmetic (quiet), while arithmetic (aloud) resulted in the greatest heart rate increases of all the conditions.

These findings appear consistent with those of Brown et al., (1988), and follow from the demand similarities of reading neutral text and numbers versus mental arithmetic aloud (i.e., referent word production versus abstract symbol processing with speech motor activity). The omission of verbalization in mental arithmetic resulting in lower heart rate compared to arithmetic (aloud) suggests that the presence or absence of the speech motor activity involved in producing verbal answers may influence the magnitude of heart rate changes to this task. In a more recent study, however, Linden (1987) compared cardiovascular responses to personally relevant speech to those seen with counting forward by increments of one. Based on the finding that counting did not significantly increase heart rate, it was concluded that physical exertion due to simple verbalization accounts for little of the reactivity seen with speech with affective content.

The impact of speech motor activity on heart rate has been indirectly touched upon in a study of the relationship between eye blink and cardiac activity (Schuri & von Cramon, 1981). Heart rate responses to two mental arithmetic tasks (i.e., involving either serial addition or subtraction) were measured while subjects performed each both verbally and non-verbally. Two sequences of presentation were employed. Significant increases in heart rate were evoked by both tasks and response conditions. However, significant differences in heart rate between vocalization and nonvocalization response conditions occurred only with a particular order of presentation of tasks and with serial addition only. The authors suggest that an improperly counterbalanced order of task presentation may have contributed to these inconsistent findings. Overall, since heart rate response to speech motor activity was not the primary focus of this study, the data, as it pertains to the present discussion, should be interpreted with caution.

A number of studies (Carroll, Turner & Rogers, 1987; Stoney, Langer & Gelling, 1986) have attempted to differentiate psychological and physical sources of heart rate reactivity through direct measurement of metabolic function. Heart rate and oxygen uptake with physical exercise were initially observed in order to predict metabolically justified increases in heart rate. When mental arithmetic and reaction time tasks were subsequently administered, excessive heart rate was seen over and above that predicted by the metabolic demands of these tasks. These studies have provided convincing support for the uncoupling of cardiac responses from metabolic demands when confronted with stressful tasks. However, the mouthpiece for expired gas collection employed in this paradigm precludes exploration of the influence on heart rate of verbalization as a response modality.

In summary, the underpinning of much of the interest in heart rate reactivity occurring with exposure to stressful tasks rests on the assumption that a relationship exists between such autonomic responses and psychological coping. Parametric inquiry has consistently supported an uncoupling of heart rate responses to stressful tasks from their purely physiologic demands. However, there are strands of evidence that suggest mental arithmetic, a commonly-employed laboratory task, may elicit heart rate reactivity due to both its psychological as well as physical characteristics. Thus, while a relationship does appear to exist, the degree to which psychophysiological responses in such settings are associated with psychological versus physical task demands remains ill-defined.

Baseline Measurement

The transformation of raw heart rate data into change scores is a common feature in the psychophysiological literature. These scores are calculated by subtracting a resting measure of heart rate from that seen during stressor presentation. As experimental interest is usually focused on transient responses to stress, a baseline heart rate measure is sought that represents a stable, individual level of resting heart rate from which valid and reliable change scores may be derived.

If measures of baseline heart rate do not correspond to a 'true' resting condition, as when heart rate is higher than normal resting levels, reactivity data might be affected in a number of ways. For example, subtracting an elevated baseline heart rate from heart rate measures might yield change scores of significantly reduced magnitudes compared to those derived from lower resting heart rates.

The manner in which elevated baselines might further compromise the sensitivity of the data is further outlined in the "Law of Initial Value" (LIV), a widely accepted corollary in the psychophysiology literature. Briefly, the LIV states that the prestimulus level of a physiological function will be related to response magnitude, where higher initial values will result in smaller responses to function-raising agents. The LIV may exert its influence on the data in a number of ways. Where subtle increases in heart rate are being examined, initial elevated levels of heart rate activity might result in significant attenuation of subsequent changes with stress. If response magnitudes are large, on the other hand, elevated baseline measures may result in response ceilings being encountered.

Scher and his colleagues (1985) have explored whether the LIV empirically predicts the relationship between baseline measures and heart rate reactivity. Employing a repeated measures design, subjects were exposed to a series of tasks requiring mental reversal of a digit string. Heart rate was recorded during pre-stimulus intervals and during stimulus presentations. While the within-subject correlations between pre-stimulus and stimulus heart rate provided some support for the LIV, a significant between-subject correlation opposite in sign to that predicted by the LIV was also revealed. These researchers concluded from their findings that while the LIV may predict cardiac responses in some individuals, important between-subject differences in reactivity also exist.

The influence that baseline measures may exert on indices of autonomic activity, therefore, has raised a number of methodological issues concerning the characteristics of baseline heart rate and how it might best be estimated. The short-term stability of baseline heart rate is one such issue. Heart rate adaptations to quietly sitting in the laboratory have been previously documented (Hastrup, 1986), with decreases in heart rate observed over longer periods of time. In spite of these observations, resting periods varying in time from a few seconds to 30 minutes appear in the research. With little apparent consensus regarding the optimal waiting period for obtaining a pre-stress resting heart rate, systematic examination of this issue is warranted.

A second related, yet unresolved question concerns the vulnerability of baseline heart rate to contextual factors. It has been argued that simply being in the laboratory attached to electrodes and anticipating a stressful event may be sufficient to elevate heart rate during pre-stress waiting periods (Obrist, 1981). In order to avoid the possible influence of anticipation on baseline measures, Obrist (1981) has contended that heart rate recorded when subjects are relaxed and not expecting stressful stimuli provides a more accurate representation of a 'true' resting state.

There are some strands of evidence to support this contention. Variation in pre-stress heart rate has been reported when a stressful task is anticipated, with response magnitude dependent on the degree to which subjects have been informed as to what to expect (Meyers & Craighead, 1978). Furthermore, individual differences may be accentuated under these circumstances, with the magnitude of elevation in pre-stress heart rate being greater in more reactive subjects (Light & Obrist, 1980 b). Two other studies, however, have failed to find pre-stress heart rate dependent on whether or not a stressful task was anticipated (Linden & Frankish, 1988; Drummond, 1985). Whitsett et al., (in press) on the other hand, have produced data in support of Obrist's (1981) position. However, these authors also suggest that simply sitting for long periods of time in the laboratory may also result in alterations in heart rate if subjects begin to ruminate over concerns in their lives through boredom. Hence, they suggest that presenting slides depicting peaceful scenery may circumvent this possibility.

Finally, another study (Kjellberg & Magnusson, 1979) has compared no-stress and pre-stress methods of baseline determination when either an "intake" (i.e., passive-coping) or a "rejection" (i.e., active-coping) task was subsequently presented. No differences between the two methods were found employing the former task type, but significant differences in baselines measures emerged employing the latter. This interesting finding hints that the impact of anticipation on baseline measures may be contingent on the type of task being presented.

In summary, questions concerning the waiting time required prior to measurement, and the anticipation of stress on the accurate determination of baseline heart rate have been raised. However, the data emerging from studies investigating these issues have been inconsistent. Thus, a number of further questions need to bc addressed to advance understanding of this area. First, in the absence of imminent stress, does some adaptation in heart rate occur over several minutes, and if so, what duration of time is required to achieve a stable resting heart rate measure? Second, are resting heart rates obtained in the laboratory with no stressors anticipated stable with repeated testing over several weeks? Third, how comparable are heart rates recorded immediately preceeding stressor presentation to those recorded at a separate no-stress session? Overall, the systematic analysis of the above issue could clarify whether the additional effort required to record no-stress baseline measures is justified.

Reliability of Measures of Heart Rate

A further methodological issue concerns the reliability of heart rate measures. The focus of much psychophysiological research has been the identification and alteration of persistent, individual cardiovascular responses that may contribute to cardiovascular pathogenesis. Several recent studies provide support for the stability of individual response patterns to different psychological stressors over various lengths of time (Seraganian et al., 1985; Foerster, 1985; Matthews, Rakaczky, Stoney & Manuck, 1987; Giordani, Manuck, & Farmer, 1981; Manuck & Garland, 1980; Robinson, Whitsett & Kaplan, 1987; Fahrenberg, Schneider & Safian, 1987). However, as previously noted, physical components of the stressful tasks employed may be reflected in measures of reactivity. Therefore, in repeated measures experiments, it is possible that observations of stability may to some degree reflect the consistent physical demands inherent in the particular task employed. As discussed above, where more precise distinctions between physical and psychological sources of reactivity have been attempted using direct oxygen uptake methods, tasks requiring verbalization could not be utilized (Allen, Obrist, Sherwood & Cromwell, 1986). Therefore, as many laboratory stressors involve verbal responses, further understanding of the stability of reactivity may emerge if heart rates with repeated exposure to stressful tasks are examined with the psychological versus physical task demands clearly differentiated.

<u>The Collection and Analysis of Heart Rate Data</u>

A final methodological issue of interest is the manner in which heart rate data are gathered and analysed. Heart rate measures have been generally reported in absolute beats per minute, or in change over baseline in beats per minute (Krantz & Manuck, 1984; Linden & Frankish, 1988). How the actual data are temporally factored to yield a measure of heart rate, however, varies greatly from study to study. For example, heart rate may be derived by counting heart beats in the last 10 seconds of each minute and multiplied by six to yield minute-by-minute measures. On the other hand, quite different data may be gathered if each beat is counted over entire minutes to ascertain minute-by-minute heart rates.

Some authors have suggested that a fine-grained approach to observing heart rate throughout the duration of a stressor may provide important data about coping with psychological stress (Jamieson & Lavoie, 1987; Sinyor et al., 1986). However, common practices of extrapolating mean heart rates from short durations of data collection and counting beats over entire minute periods are relatively insensitive to subtle fluctuations in heart rate reactivity over time. The development of computer-assisted data-logging apparatus now permits the measurement of individual reactivity characteristics derived fronm continuous beat-by-beat measures of heart rate. The amenability of heart rate to this precise scrutiny, therefore, favors further study of this index of reactivity.

<u>Purpose of the Present Study</u>

The interest in psychophysiological reactivity has for the most part been predicated on the assumption that changes in autonomic functioning in relation to psychologically stressful tasks may be related to behavioral processes. While the focus of recent research efforts has been to identify individual response patterns to stress, the inquiry has been conducted in the absence of a clear understanding of how different laboratory procedures might contribute to the data. The purpose of the three experiments making up the present study, therefore, was to examine systematically a number of prominent methodological issues in this area.

The first and second experiments addressed a number of aspects of baseline heart rate measurement. In Experiment I, heart rate was monitored while subjects sat quietly in the laboratory for approximately 24 minutes. One month following this session, subjects were asked to return to the laboratory for another session involving the same protocol. In this fashion, the stability of baseline heart rate gathered when no stress was anticipated could be assessed over both short as well as long intervals. If resting heart rate taken with no stressor imminent were a stable and robust individual feature, it was hypothesized that between sessions differences would be negligible. If, however, resting heart rates were vulnerable to fluctuations, then the present design would clarify whether these modulations occur over several minutes or over several weeks with repeated testing, whether these changes occurred in a systematic fashion, and what direction they might take.

In addition, Experiment II allowed exploration of whether baseline heart rate determined when stress was anticipated differs from baselines gathered when no stress was anticipated. Therfore, initial baseline heart rates gathered in Experiment I were compared to initial baseline heart rates observed in Experiment II. It was hypothesized that if anticipation of stress altered baseline heart rate, then the pattern and/or magnitude of baseline heart rate observed in Experiment II would be significantly different from that observed in Experiment I.

Experiment II also explored the relative importance of behavioral factors in reactivity to a stressful task compared with the contribution of the physical demands of the task. Two mental arithmetic tasks were presented during one laboratory session. One task involved verbalization of solutions to mental arithmetic problems, while in the other, solutions were required, but without response vocalization. All other features of the two tasks, including their frequency, difficulty and the motivation to solve the problems, were held constant. It was hypothesized that if vocalization contributed to heart rate during mental arithmetic, heart rate seen with the two tasks would differ significantly. Furthermore, this methodology would permit an evaluation of the impact on heart rate of only the psychological demands of mental arithmetic.

Experiment III explored the reliability of measures of heart rate reactivity. Two mental arithmetic tasks, one with verbalization of responses and one without, were presented on two occasions one month apart. In this manner, two specific issues could be addressed. First, the reliability of heart rate responses to mental arithmetic over a one month period could be assessed. Second, by using equivalent tasks with either verbal or non-verbal demands, the contribution of speech motor activity to the reliability of heart rate responses to mental arithmetic might be clarified.

The final issue addressed in the three experiments of this study involved data analysis practices. To a great extent, current data analysis protocols have involved averaging heart rate sampled at discrete intervals to provide a quantitative index of response. Expressed in this way, however, heart rate measures to stress may not be sufficiently sensitive to reveal potentially important response profile information. Thus, the present study employed fine-grained, computer-assisted measurement of inter-beat intervals. In this manner, heart rate responses seen in the context of the above paradigms could be both qualitatively and quantitatively evaluated.

Experiment I

Introduction

A number of questions have been raised concerning baseline measurement protocols in the psychophysiology literature (Linden & Frankish, 1988). Insufficient resting periods before baseline determination have been posited as resulting in higher heart rate measures as compared to measures taken after longer periods of time (Hastrup, 1986). As well, anticipation of stress may result in elevated baseline measures, especially when there is uncertainty surrounding the stressful task to be undertaken (Meyers & Craighead, 1978). While determination of baseline in a separate no-stress session has been hypothesized as resulting in more accurate measures of resting heart rate (Obrist, 1981), many features of such a baseline methodology have yet to be studied.

Experiment I sought to clarify a number of questions concerning baseline heart rate determined when no stressful task was anticipated. First, are heart rate measures gathered when subjects are required to sit quietly in the laboratory reliable with repeated data collection over a 10 minute interval; and if so, is a 10 minute resting period prior to data collection sufficient? Second, are heart rate measures gathered in this manner reliable with repeated testing over a one month period?

Employing a repeated measures design, subjects visited the laboratory twice, with a one month interval between sessions. Subjects expected and received no stressful stimuli in either session. Heart rate was recorded for 130 seconds twice per session, once after an initial 10 minute resting period, and again 10 minutes later.

Method

Subjects Eleven male students of Concordia University, ranging in age from 20 to 40 years were screened via a written questionnaire for the absence of any major medical problems (e.g., blood pressure greater than 150/95, history of heart disease, use of medication) contraindicating exposure to mild stress in the laboratory. Females were not used since reactivity may be influenced by cyclic hormonal changes (Hastrup & Light, 1984).

Apparatus and Stimuli The experimental room consisted of a temperature and humidity-controlled electronically-shielded chamber (305 x 335 cm, Spectrashield). A four-channel Beckman 511A Dynograph recorder and three Medi-Trace silver chloride disposable electrodes were used to record heart rate during the experimental sessions. The signal was processed through a Beckman (Type 9857) cardiotachometer coupler which transformed and recorded on one channel of the recorder the raw ECG signal into an analogue signal proportional to heart rate. This signal was subsequently fed by way of a Data Translation Analogue to Digital Converter (Model DT 2801) board directly into an expansion slot of an IBM PC computer (Model 5100). Incoming analog voltage signals are converted to a binary number 12 bits long, allowing 4096 different states (i.e., 2 raised by the power of 12) and a resolution of input differences in heart rate of .024% (ie., 100 x 1/4096). A custom software package permitted calibration and menu-driven sampling of analogue outputs of the Beckman at one-second intervals. The output of the analogue to digital convertor was sampled at one second intervals and stored in files containing digitized records of averaged heart rate on a beat-by-beat basis. An example of both the analogue output and digitized transformation of beat-by-beat heart rate is presented in Appendix 1. A Sony taperecorder (Model TC-630) and Sony headphones were employed to transmit the instructions.

<u>Procedure</u> Upon arrival to the laboratory, subjects were asked to read and sign an informed consent form which included a brief description of the study (see Appendix 2).

After entering the enclosed chamber, the subject was seated in an armchair in a curtained-off area. Three electrodes were attached to the chest for heart rate monitoring, and the polygraph calibrated. Taped instructions were then presented directing the subject to relax during the entire 24-minute protocol in either English or French, depending on the subject's mother tongue. A 10 minute interval ensued with continuous heart rate monitoring. Then, a 130-second data-logging trial followed. No further instructions were given; thus, the subject was unaware when data collection was in progress. This was followed by another 10 minute interval after which another 130-second data-logging trial occurred. After approximately four to five weeks, the subjects were called into the lab for a repeat session equivalent to the first.

Results

Heart rates over both 130-second sampling trials were collapsed by averaging heart rates over each 10 second period, thereby giving 13 periods of average heart rate per data-logging trial. Infrequently, errors in the data-logging procedure (i.e., less than 3 percent) resulted in less than a full complement of heart rate measures.

A graph depicting the average heart rate for each 10 second period for all subjects over all four trials is presented in Figure 1. (Individual heart rate data are presented in Appendix 3). Table 1 summarizes the mean, range and standard deviation of subject mean heart rate data over the two data-logging intervals conducted in each session. A 2 x 2 x 13 (Session x Trial x Period) repeated-measures ANOVA was performed using BMDP Statistical Software (1985), which also produces the Greenhouse-Geisser adjustment for violation of the assumption of sphericity in repeated measures analyses (Vasey & Thayer, 1987). The Session factor refers to the one month test-retest comparison of mean heart rate, the Trial factor being the comparison of means between the two data-logging intervals within each session, and the Period factor referring to the comparisons between heart rate means derived from each of the 13 10-second consecutive intervals of data-logging for each task. Significance at

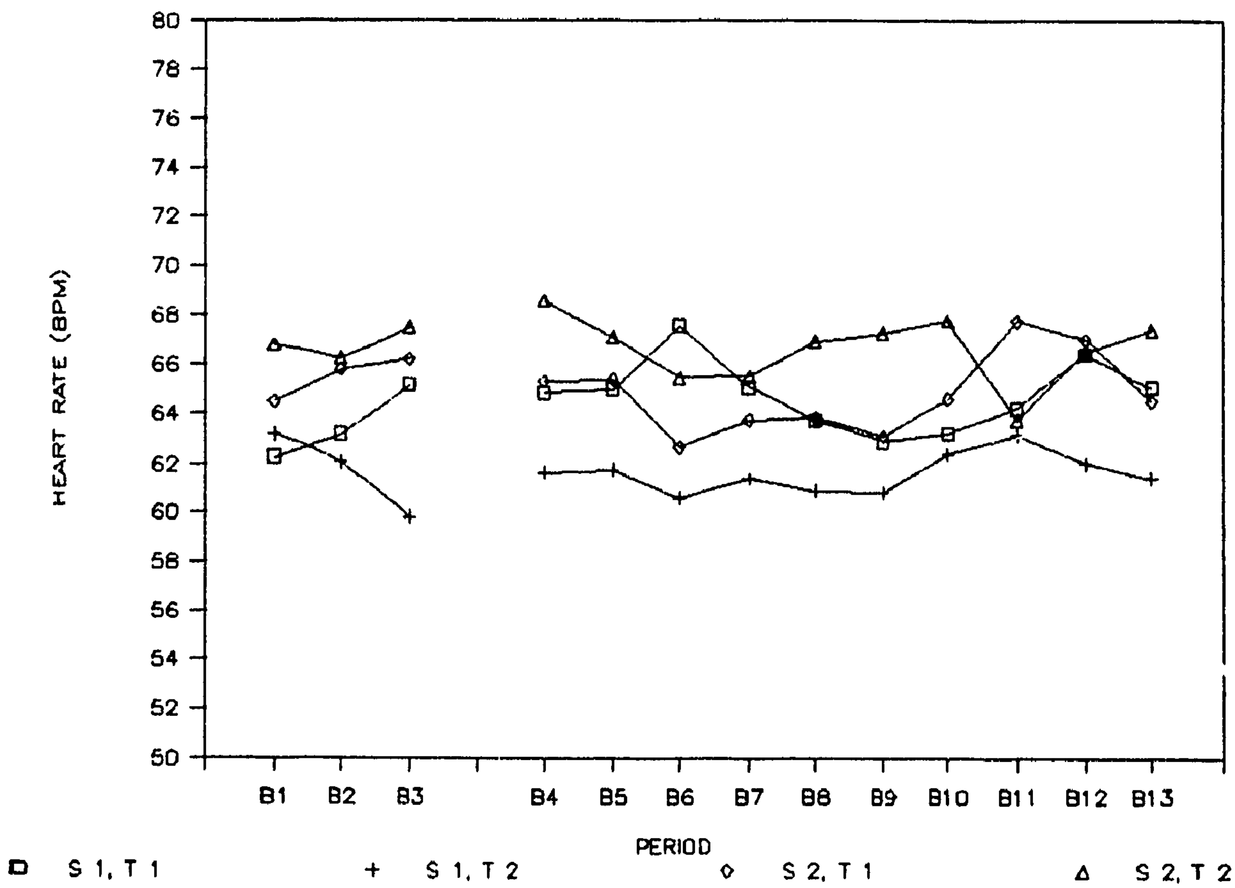


Figure 1. Resting heart rate over 13 10-s intervals (B1 - B3) for both trials in repeated sessions separated by one month. Symbol legend (S, T) connotes Session 1 or 2, and Trial 1 or 2.

Table 1

Mean, Range and Standard Deviation of Subject HR (BPM) for Trials 1 and 2 of Sessions 1 and 2.

	Mean	Range	Standard Deviation
Session 1			
Task 1	64.4	32.2	8.7
Task 2	61.6	30.0	8.4
Session 2			
Task 1	65.0	34.6	13.5
Task 2	66.7	37.4	10.5

$p < .05$ was not achieved for any of the main effects or interactions.

Another way of examining the reliability of this resting baseline heart rate data involved calculating the test-retest Pearson product correlations over short and long periods of time. Averaging the heart rates of the first three periods of both data-logging trials allowed the short-term test-retest reliability of heart rates over a 30-second interval after 10 and approximately 23 minutes of rest in the enclosed chamber for each of the two visits. The choice of these intervals follows from the commonly-employed protocol of allowing a several-minute rest period before stressors are presented and between repeated stressor presentations. The short-term test-retest Pearson product-moment correlations were .74 and .89 for the first and second sessions respectively, $p < .01$. Taking the average heart rate for both trials in each session, the one-month test-retest correlation was .65, $p <$ 05.

Discussion

The absence of either any significant main effects or interactions in Experiment I sheds light on heart rate adaptations to sitting quietly in the laboratory with beat-by-beat resolution. Exposure to the laboratory setting in the absence of anticipation of stressful tasks resulted in heart rate measures that were stable during the initial 130-second data-logging interval of Session 1, and again with repeated testing one month later in Session 2. Thus, a 10-minute waiting period prior to data collection was sufficient to produce stable heart rate measurements that did not fluctuate significantly over 130 seconds of data collection.

The absence of a significant Trials or Sessions main effect suggests that no further adaptation occurred in overall heart rate after an initial 10 minute waiting period, neither after a further 10 minutes of sitting quietly in the laboratory, nor after a month with repeated testing. The significant correlations between mean heart rate over both trials of each session supports the strong relationship between individual heart rate over a 10-minute period during the same session, and after repeated testing a month later.

Overall, these findings provide a consistent picture of the stability of no-stress baseline heart rate with repeated measurement over both short (i.e., several minutes) and long periods (i.e., one month) of time. Comparison of this baseline protocol and baseline determined immediately prior to stress presentation is presented below in Experiment II.

Experiment II

Introduction

Heart rate reactivity to mental arithmetic is frequently employed in the experimental context as an index of the psychophysiological processes involved in stress-coping. However, there is some evidence that reactivity to common laboratory stressors may to some degree be related not only to the psychological, but also the physical demands of these tasks (Linden, 1986; Brown et al., 1988). While attempts have been made to differentiate more clearly physical versus psychological determinants of reactivity to mental arithmetic (Turner & Carroll, 1985), to this author's knowledge no direct examination of the impact of verbal response modalities has yet been conducted.

The principle purpose of the second experiment was to compare reactivity elicited by two mental arithmetic tasks of equivalent difficulty, frequency, and cadence, where in one task, vocalization of solutions to the problems was required, while in the other, vocalization of responses was not. It was hypothesized that if the speech motor activity involved in generating the verbal responses contributed significantly to heart rate reactivity, a comparison of heart rate seen with both tasks might reveal significant quantitative and qualitative differences.

A further issue touched upon in this second experiment was the comparability of baseline measures taken when a stressor was expected with those taken in Experiment I when no stressor was expected. It has been contended (Obrist, 1981) that anticipation of a stressful event is sufficient to elevate resting baseline heart rate above that observed when no stress is expected. The identical laboratory setting and similar protocols employed in Experiments I and II presented an opportunity to pursue this issue. Thus, resting heart rate data with no anticipation gathered in Experiment I was compared to baseline heart rate data in the present experiment where a stressful task was expected.

Method

Subjects Fourteen male students were recruited in similar fashion as in Experiment I.

Apparatus and Stimuli The apparatus employed for this experiment was similar to Experiment I, with the following additions. Two tape-recorded mental arithmetic tasks were presented employing the tape recorder and headphones used in the previous experiment. Each task consisted of 11 problems, each requiring two serial arithmetic operations on integers (e.g., 14 x 6 - 17). All problems were designed to be of comparable complexity. Ten seconds were allotted for the presentation of the problem and a generated solution before the next problem was presented. A white, experimenter-controlled indicator light, situated three feet from the subject at eye-level, indicated whether verbal or non-verbal responses to the problems were to be made. A ten-point subjective rating scale (see Appendix 4) was used to evaluate the perceived difficulty of the two tasks.

Procedure The initial preparation of the subject was identical to that of Experiment I. Taped instructions (see Appendix 5) were then presented over headphones in either English or French directing the subject to mentally calculate the solution to each arithmetic equation, but to verbalize the response only if the white indicator light was illuminated before the next problem was presented. The subject was told that the indicator light would be illuminated for some problems but not for others.

After the instructions ended, a 10-minute resting interval ensued, followed by 30 seconds of baseline heart rate collection. Immediately after, the first stressor task was presented, and heart rate collection continued throughout the 110 seconds of the task. Another 10-minute resting period ensued, followed by a secon. baseline determination and collection of heart rate data during the second task. Immediately after completion of the the second task, the subjective rating questionnaire of task difficulty was administered.

In fact, in one task the white light was illuminated for the first 10 of the 11 problems presented, while in the other, the light was not illuminated for any of the 10 initial problems, but came on for the last one. These two protocols were counterbalanced between all subjects.

Since heart rate data were collected for only the first ten problems, effectively one task involved mental arithmetic with vocalization (verbal MA), while the other involved mental arithmetic without vocalization (nonverbal MA). In this manner, comparison of heart rate responses to mental arithmetic both with and without verbalized responses could be carried out.

The inclusion of the 11th problem in each task requiring a response opposite to the initial 10 questions was contrived to maintain motivation to solve all the problems presented. It was hoped that this protocol would maintain the impression that the light might be illuminated at any time, and thereby reduce the possibility of an expectancy of a no-light or all-lit condition in the second task. Furthermore, the instructions indicated that the final problems of each task would bear on the solutions of the first ten problems. Thus, regardless of whether subjects were not required to verbalize their solutions to the problems, they were nonetheless motivated to solve all problems.

Results

Heart rate was calculated by averaging the digitized heart rates for each 10-second period of data logging. Figure 2 depicts average heart rate over the three 10-second baseline periods preceding and ten 10-second periods during the performance of both tasks. Appendix 6 contains the individual heart rate data.

A 2 x 3, Task x Period ANOVA was performed on baseline data to determine whether the baseline heart rate measures between the two tasks were dissimilar. No significant main

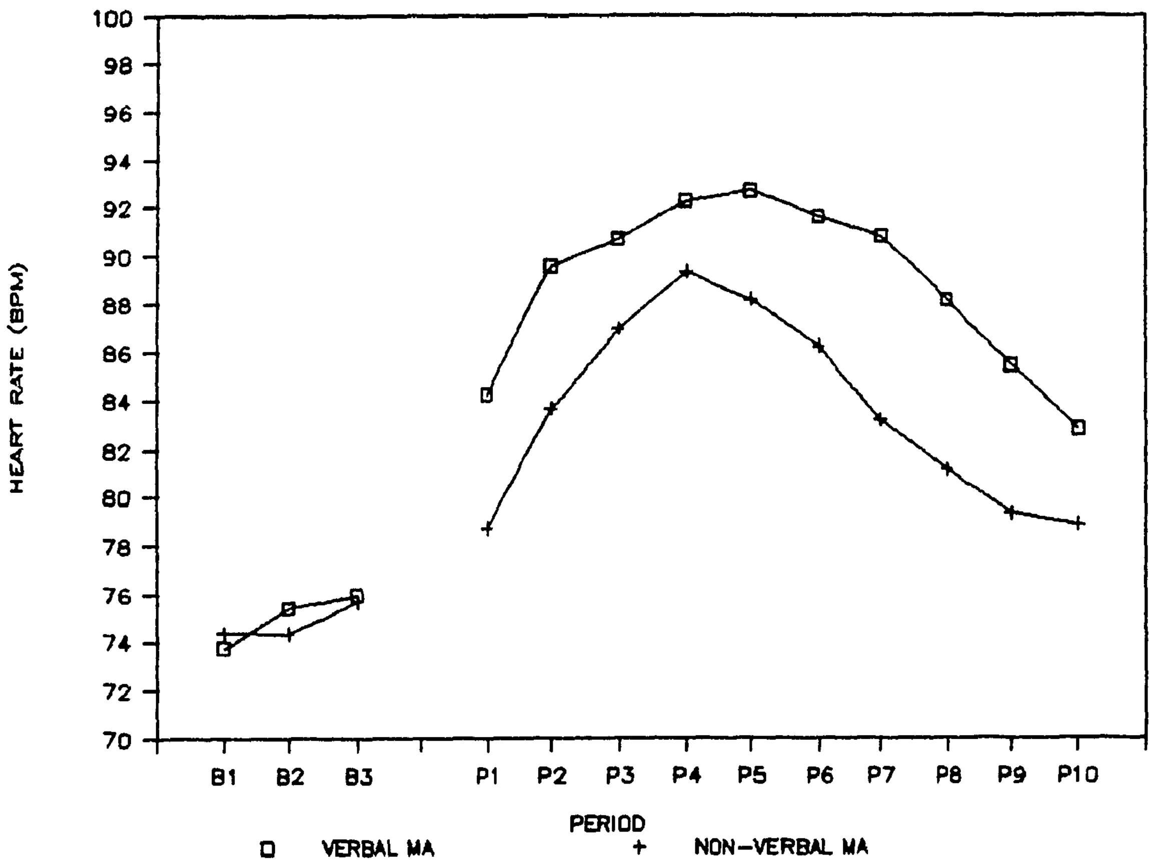


Figure 2. Heart rate at 10-s intervals during baseline (B1-B3) and during the performance of verbal and non-verbal MA (P1-P10).

effects or interactions were revealed between the baselines preceding the tasks. A Pearson-product correlation between the averages of the three baseline periods prior to each task indicated a high degree of correlation, with $r = .92$, $p < .01$. These analyses indicated that baseline heart rates prior to each task were similar, thus permitting the use of untransformed heart rates in subsequent analyses.

An analysis was undertaken to determine whether the tasks resulted in significant increases in heart rate over baseline levels. Employing the mean of three baseline heart rate periods and the ten periods over which the tasks were presented, a 2 x 11, Tasks x Period ANOVA was performed. Significant main effects of Task, $F(1,13) = 5.64$, $p < .05$, and Period, $F(10,130) = 16.15$, $p < .001$, were found. The Task x Period interaction did not attain significance.

Of specific interest were differences in heart rate between the mean baseline heart rate for each task and all the subsequent ten stress periods. Post hoc examination of the data employing the Tukey Honestly Significant Difference statistic revealed that to reject the null hypothesis (i.e., that mean period heart rates were not significantly different from each other) at the $p < .05$ level of confidence, a difference of 4.5 beats per minute was required. In both tasks it was found that heart rate was elevated significantly above baseline levels over all periods in which mental arithmetic was presented.

The 2 x 10, Task x Period ANOVA presented in Table 2

Table 2

Source Table for Task (Verbal, Non-Verbal MA) x Period, 2 x 10 ANOVA on Heart Rate.

Source	SS	DF	MS	F
Task	1960.5	1	1960.5	5.6*
Error	4575.5	13	352.0	
Period	3441.6	9	382.4	13.6**
Error	3296.3	117	28.2	
T x P	140.1	9	15.6	.7
Error	2813.5	117	24.0	

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

Note. Probabilities calculated using Greenhouse-Geisser degrees of freedom adjustments.

compared heart rates seen with the two tasks. Significant main effects of Task and Periods, $F(1,13) = 5.57$, $p < .04$, $F(9,117) = 13.57$, $p < .001$, respectively, were discovered, while no significant interaction emerged. This indicated that heart rate increases for all periods were significantly different in magnitude, with verbal mental arithmetic eliciting greater responses than non-verbal mental arithmetic.

Averaging heart rate over all ten periods of each task, as well as averaging across the three baseline measures for each task, allowed a comparison between overall mean heart rate during baseline and stress for each task. These data are presented in Table 3. This 2 x 2, Task x Period ANOVA revealed a significant Period main effect, $F(1,13) = 20.2$, $p < .001$, indicating that independent of task, heart rate increased significantly from baseline levels with mental arithmetic. However, no significant differences emerged between the two tasks when heart rate changes were expressed in this manner.

Taking mean heart rate for each task over all subjects also permitted the examination of the relationship of heart rate to verbal and non-verbal mental arithmetic. Table 4 presents the mean, range and standard deviation for these data. Figure 3 depicts a scatterplot of the average change scores for both tasks for individual subjects. The Pearson product-moment correlation between the two difference scores ($r = .88$, $p < .01$) indicates that the cardiac responses to

Table 3

Cell Means, Standard Errors and Source Table for Task x Period (Mean Task HR vs Mean Baseline HR) ANOVA.

	Baseline		Task	
	HR	St.Err	HR	St.Err
Verbal MA	75.0	±3.5	88.9	±4.8
Non-verbal MA	74.8	±3.6	83.6	±4.0

Source	SS	DF	MS	F
Task	107.9	1	107.9	4.5
Error	305.9	13	23.5	
Period	1784.9	1	1784.9	20.2***
Error	1147.9	13	88.3	
T x P	88.6	1	88.6	3.4
Error	338.9	13	26.1	

***$p < .001$

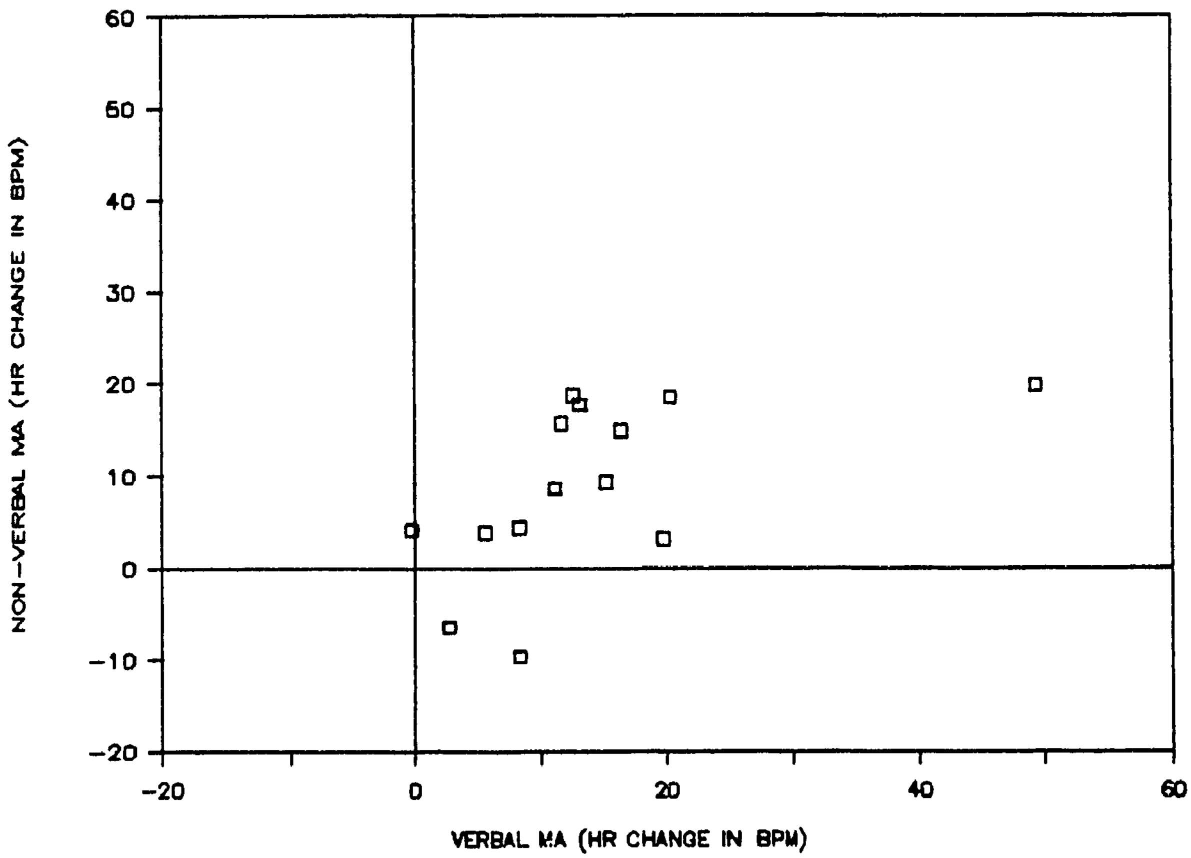


Figure 3. Scattergraph of HR change from baseline during the performance of verbal and non-verbal MA.

Table 4

Mean, Range and Standard Deviations of Subject HR Change Scores during Mental Arithmetic.

	Mean	Range	Standard Deviation
Verbal MA	13.8	49.6	11.4
Non-verbal MA	8.8	29.5	9.1

both tasks are significantly correlated despite their different response modalities.

The fine-grained data collection procedures employed in the present study allowed more detailed exploration of the relationship between heart rate responses to verbal and non-verbal mental arithmetic. Pearson product-moment correlations were calculated between mean heart rate over each 10 second period throughout the duration of both tasks. The result of these calculations are summarized in Table 5. Significant correlations ($p < .01$) between mean heart rate over all ten periods of both tasks were uncovered, with correlation coefficients ranging from .96 in period 1 to .69 in period 7. The mean subjective ratings of task difficulty for verbal and non-verbal mental arithmetic were 4.4 and 3.9, respectively, and were not significantly different.

In order to compare baseline heart rate measures gathered when a stress-inducing task is anticipated as opposed to when it is not, a mixed model, two-factor ANOVA was performed comparing the first three 10 second baseline periods of Session 1 in Experiment I with the first three 10 second baseline periods of Task 1 of Experiment II. BMDP 8V (BMDP Statistical Software, 1985) was employed to perform this analysis. The group factor A (i.e., one group anticipates a stressful task while the other does not) and period factor B (i.e., the three repeated 10-second periods making up the first baseline determination in both experiments) were fixed effects, while the subjects' factor

Table 5

Correlations Coefficients between HR Responses to Verbal and Non-verbal MA Over All Periods of the Tasks.

Period									
1	2	3	4	5	6	7	8	9	10
.96*	.86*	.86*	.91*	.86*	.75*	.69*	.76*	.79*	.82*

* $p < .01$

S were random and nested in A, but crossed in B. A significant group factor would be consistent with the hypothesis that anticipation of a stress-inducing task has a significant impact on baseline heart rate.

The source table emerging from this analysis is presented in Table 6. The mean heart rate of subjects anticipating a stressful task, at 77.5 beats/min was 14 beats greater than seen in subjects not expecting a stressful task. Significant main effects of both Groups, $F(1, 20) = 9.4$, $p < .001$, and Periods, $F(2, 40) = 3.7$, $p < .05$, were uncovered. No interactions proved significant. These findings are consistent with the interpretation that, on the average, the baseline heart rates of the subjects in Experiment II who anticipated stressful tasks were higher than those observed in Experiment I, where no stressful tasks were expected.

Non-Hypothesized Findings

Given the exploratory nature of this study and the observation that heart rate change from baseline increased for 12 of the 14 subjects with verbal and non-verbal mental arithmetic but actually decreased for the other two subjects (see Figure 3), additional analyses were carried out. That is, a re-examination of the data was undertaken by removing those two subjects whose mean heart rate response to mental arithmetic decreased from baseline levels. Figure 4 depicts the group mean heart rate responses to verbal and non-verbal arithmetic during baseline and task performance for the

Table 6

Design, Group Means and Standard Deviations and Source Table for Mixed Model ANOVA of Baseline Heart Rate of Groups Anticipating (MA) and Not Anticipating (No-MA) Mental Arithmetic.

		Symbol	Model: A, B, S(A)
Factors:	Group	A	Fixed
	Periods	B	Fixed
	Subjects	S	Random, nested in A

Group	No-MA (N=11)	MA (N=14)
Mean heart rate	63.5	74.9
Standard Deviation	8.6	11.5

Source [error]	SS	DF	MS	F
A [S(A)]	3225.6	1	3225.6	9.4**
B [SB(A)]	74.2	2	37.1	3.7*
S(A)	6884.5	20	344.2	
AB [SB(A)]	2.4	2	1.2	.12
SB(A)	405.9	40	10.1	

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

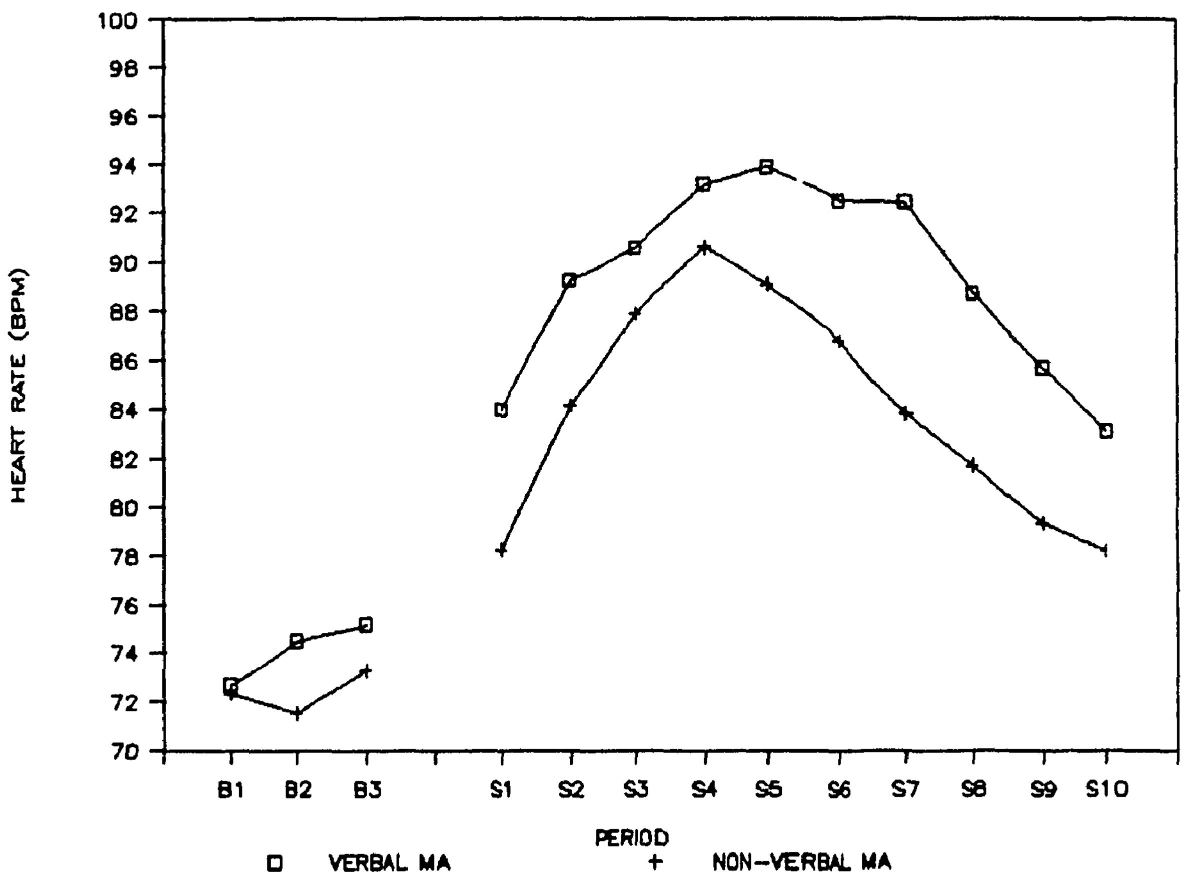


Figure 4. Heart rate at 10-s intervals during baseline (B1-B3) and during the performance of verbal and non-verbal MA (P1-P10), with subjects showing lower HR than baseline with task exposure omitted.

remaining 12 subjects. Table 7 provide descriptive statistics based on the data from those subjects. Unlike the ANOVA performed when all subjects were included, a 2 x 10, Task x Period ANOVA just failed to show a significant Task main effect. However, a simple one-way ANOVA performed on mean heart rate elicited by the two tasks over all periods failed to reveal a significant difference, consistent with the findings of the previous analvsis.

Table 7

Mean, Range and Standard Deviations of Group HR Change Scores during Mental Arithmetic Excluding Subjects (2) with Mean HR's Lower than Baseline HR.

	Mean	Range	Standard Deviation
Verbal MA	15.2	49.6	12.2
Non-verbal MA	11.6	16.7	6.6

Discussion

Experiment II clarified a number of issues related to heart rate responses to mental arithmetic with verbal and non-verbal task demands. Comparison of baseline heart rates with those elicited by both verbal or non-verbal mental arithmetic revealed that mental arithmetic succeeded in raising heart rate significantly over baseline levels for all subjects combined, whether heart rate was examined on a period-by-period basis, or whether it was averaged over the duration of the tasks. Furthermore, since heart rate was seen to be elevated above initial baseline levels (i.e., for 12 of the 14 subjects) independent of whether verbalized responses were required or not, the speech motor activity involved in mental arithmetic with verbal responses appears insufficient by itself to explain these increases.

The Task main effect uncovered in the analyses bears on the contribution of task response modalities on reactivity. This finding suggests that the speech motor activity required in verbalizing responses to mental arithmetic contributes significantly to reactivity throughout the duration of the task. In the absence of a significant interaction effect, verbalization of responses to mental arithmetic results in increases that are uniformly greater than mental arithmetic without verbalization throughout the 100-second duration of the tasks.

At the same time, however, heart rate to both tasks, expressed as an overall mean, was significantly correlated, in that 77 percent of the variance of one task may be predicted by the other. This suggests that both tasks, despite their different physical demands, elicited heart rate responses that were related in a systematic fashion. Thus, while the two tasks elicited significantly different magnitudes of heart rate reactivity, some common process seemed to be shared by both. These findings suggest that heart rate responses to the cognitive challenge of both tasks may underlie this relationship. The lack of a significant difference between the subjective ratings of task difficulty given the two tasks provides further support of this possibility.

Closer scrutiny of the scatterplot of average heart rate responses reveals that although both tasks elicited consistent overall patterns of reactivity, appreciable individual differences were still apparent. This was particularly true of responses to non-verbal mental arithmetic, where the heart rates of two subjects out of the total sample of fourteen subjects actually decreased from baseline.

This individuality in responding has been seen in prior work emerging from this laboratory (Brown et al., 1988) and raises the possibility that this pattern reflects a response characteristic of some proportion of the population to tasks of this nature. As such it is possible that other studies in this area may also encounter this response profile. Re-analysis of the data was undertaken in order to clarify what impact this phenomenon has on data of this kind.

It would appear that the impact of this response pattern is seen more on heart rate data elicited by tasks with less somatic involvement, where the trend appears towards raising mean heart rate and reducing the variability of the data through decreasing the standard deviation. Interestingly, the inclusion of these subjects contributed to the variance in some systematic way resulting in significant differences between heart rate responses to the two tasks. It would appear therefore that while some subjects' responses are in some fashion different from those of the other subjects, the between-task, within-subject relationship in the responses of these subjects are nonetheless consistent with those of the rest of the group.

Further fine-grained analysis of heart rate indicated that the profiles of cardiac activity elicited by both tasks were also significantly correlated. Over all the ten periods of the tasks, correlations between the two tasks ranged from .69 to .96. This trend suggests that a relationship between the heart rate responses may exist as a function of time over the duration of the tasks; that is, the degree to which heart rate during one task predicts that of the other decreases during the middle stages of the stressor presentation. This finding hints that individual differences in responding to the two tasks may be more pronounced during recovery after peak heart rates are attained. As the cognitive challenge of the two tasks was similar, it could be hypothesized that it is some variability due to speech motor activity involved in verbal mental arithmetic that may be emerging at this point in the tasks.

The between-subject comparisons of baseline measures obtained both when stress was anticipated and when it was not, afforded some insight into the potential impact of contextual factors in influencing baseline heart rate measurement. The group of subjects of Experiement I sat quietly in the laboratory with no further task demand asked of them while those in Experiment II anticipated the presentation of a stressful task. Other than this difference, both groups experienced an equivalent experimental protocol with heart rate data gathered after precisely the same waiting resting period (i.e., 10 minutes). The finding that the group in Experiment I had a mean resting heart rate significantly lower than the group of subjects in Experiment II provides some support for those authors who advocate the assessment of resting heart rate in the absence of imminent stress, insofar as lower heart rate in an experimental laboratory context is considered a more accurate reflection of the normal resting condition.

It could be argued that the between-subject design of this study may have been confounded by subject differences independent of the influence of anticipation. For example, as the present research was conducted over several months, changing seasonal patterns of physical activity may have been reflected in group differences in fitness. Thus, the subjects participating in Experiment I may have been aerobically fitter than those recruited for Experiment II. Higher levels of fitness are known to result in significantly lower resting heart rates (Mathews & Fox, 1976). On the other hand, the source table emerging from analysis of these data revealed the absence of significant between-subject and error variances. Thus, with respect to the implications of this statistical analysis, individual subject variation does not appear to have significantly influenced the data.

Overall, as a consequence of the findings emerging from Experiments I and II, it was decided, therefore, to repeat and extend Experiment II by using a within-subject and repeated measures design (Experiment III). This was done so as to gain empirical data for further evaluating the effect of individual subject differences in heart rate responses to mental arithmetic, and to consider more precisely the effect of the purely physical components of the experimental task in contrast to the mental.

Experiment III

Introduction

An assumption that underlies much of the study of heart rate responses to stressful laboratory tasks is that such autonomic activity reflects reliable individual behavioral processes. Several studies have produced data supporting the reliability of heart rate reactivity over time (Seraganian et al., 1985; Robinson et al., 1987). However, the findings of Experiment II, as well as from other research (Brown et al., 1988), suggest that the physical components of stressful laboratory tasks may significantly contribute to reactivity. It is possible, therefore, that observations of stable heart rate measures seen with repeated exposure to laboratory stressors may to some degree reflect the consistent physical components of those tasks.

The purpose of Experiment III was to determine if, with physical sources of reactivity to mental arithmetic parcelled out, heart rate continued to be altered in a systematic fashion with repeated exposure to this task. Employing a paradigm similar to that of Experiment II, subjects were exposed to mental arithmetic both with and without verbalization of responses. After approximately one month, subjects returned for an equivalent repeat session. In this manner, a within-subject comparison between the stability of heart rate measures elicited by mental arithmetic both with and without the speech motor activity of verbalized responding could be undertaken.

Method

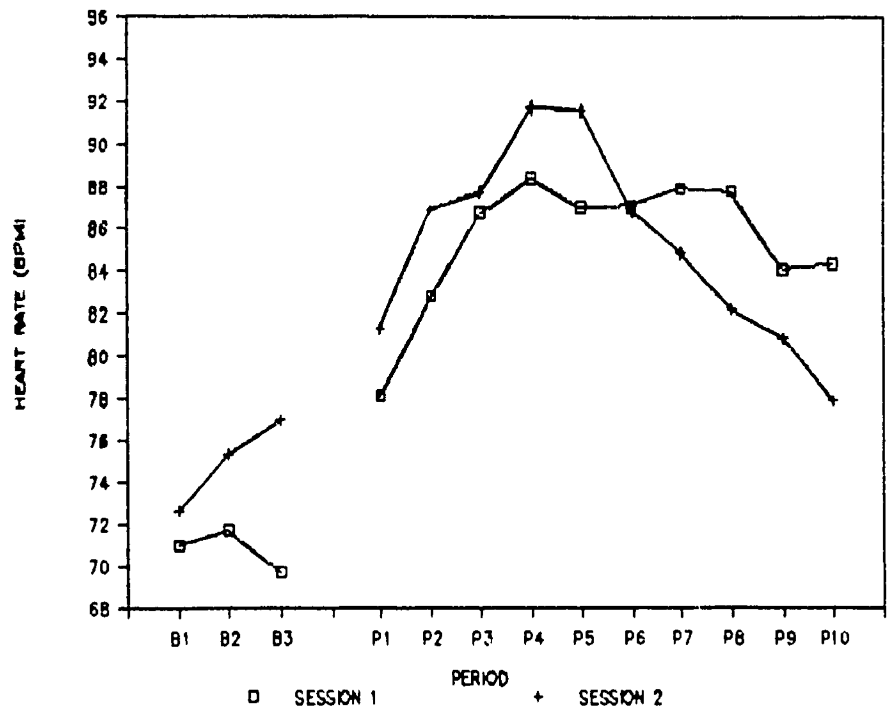
Subjects Eight males were recruited as in Experiment 2.

Procedure Subjects visited the laboratory twice, each visit separated by a four to five week period. Each session employed a protocol similar to that of Experiment 2. Subjects were exposed to mental arithmetic both with and without verbalized responses in counterbalanced order over the two sessions, with an equal number of subjects receiving each type first. The tasks were the same as those used in Experiment 2, with the same tasks repeated for each session. Thus, all subjects were required to respond verbally to both mental arithmetic tasks over the course of the study.

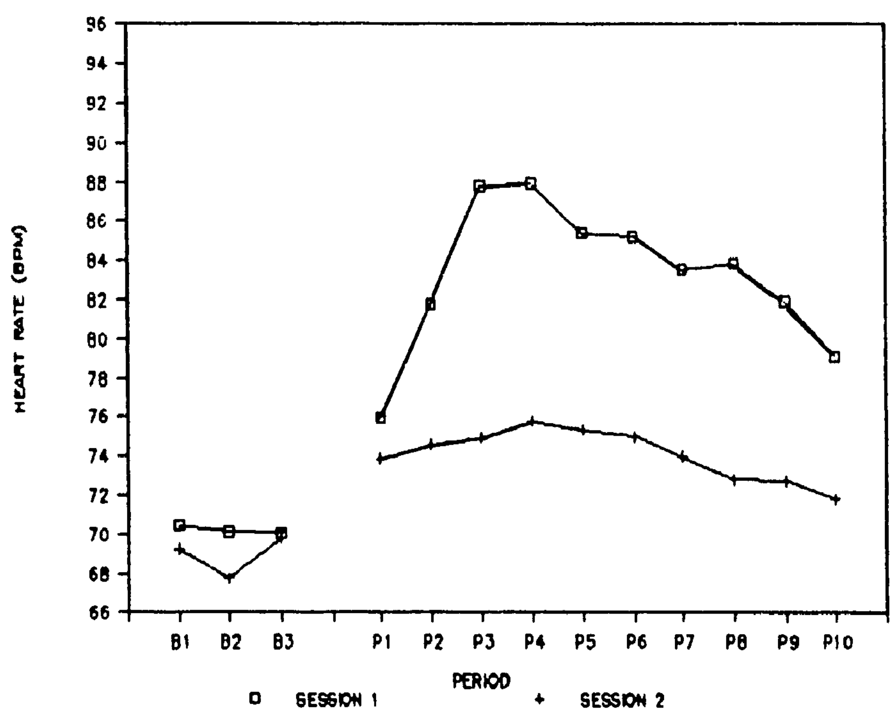
Results

Figures 5 (a) and 5 (b) depict heart rate responses to repeated verbal and non-verbal mental arithmetic tasks respectively over approximately a one month period, while Figure 6 combines these figures on one graph for another comparative perspective. Table 8 lists mean heart rate responses at baseline and during both verbal and non-verbal mental arithmetic. Appendix 7 contains individual heart rate data.

A 4 x 3, Task x Period ANOVA was performed on all baseline heart rates preceding the two tasks of both sessions. This analysis failed to reveal any significant differences between any of the baseline heart rates. Therefore, subsequent comparisons between the tasks could be made employing absolute heart rate data.



<u>Figure</u> <u>5</u> <u>a</u>. Heart rate at 10-s intervals during baseline (B1-B3) and during the performance of verbal MA (P1-P10) in Sessions 1 and 2, one month apart.



<u>Figure</u> <u>5</u> <u>b</u>. Heart rate at 10-s intervals during baseline (B1-B3) and during the performance of non-verbal MA (P1-P10) in Sessions 1 and 2, one month apart.

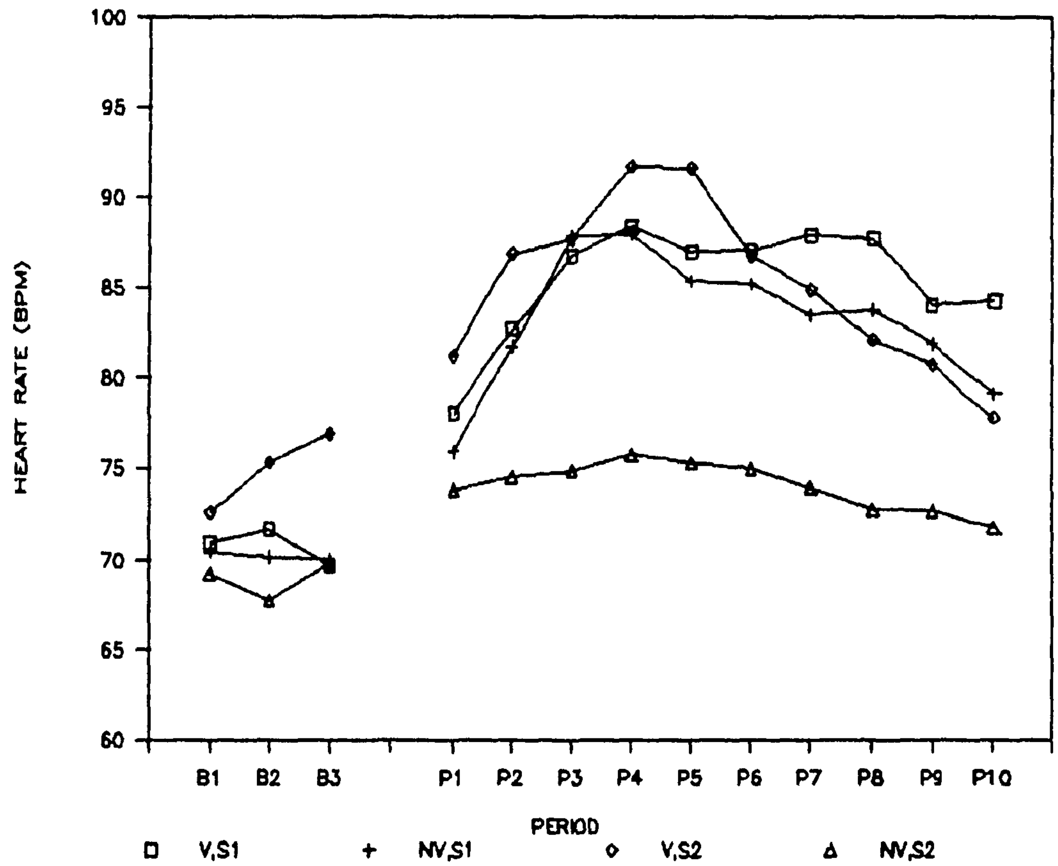


Figure 6. Heart rate at 10-s intervals during baseline (B1-B3) and during the performance of verbal and non-verbal MA (V, NV respectively) in Sessions 1 and 2 (S1, S2 respectively), one month apart.

Table 8

Cell Means and Standard Errors of HR to Verbal and Non-verbal MA.

	Baseline		Task	
Session 1	HR	St.Err	HR	St.Err
Verbal MA	70.5	±4.1	85.4	±6.8
Nonverbal MA	70.2	±4.2	83.2	±5.1
Session 2				
Verbal MA	74.6	±4.0	85.1	±4.4
Nonverbal MA	68.9	±3.2	74.1	±3.7

In order to determine whether all four tasks (i.e., two verbal, two non-verbal mental arithmetic tasks) succeeded in eliciting significant change in heart rate over preceding baseline measures, a 4 x 2, Task x Period ANOVA was performed on mean heart rate observed during all baselines and mental arithmetic tasks. The results of this analysis are presented in Table 9. The significant Period main effect, $F(3,21) = 4.1$, $p < .05$, indicated that across all tasks, mean heart rate with mental arithmetic, whether verbal or non-verbal in demand, increased over mean resting baseline levels. The significant Task main effect, $F(1,7) = 38.6$, $p < .001$, indicated that some tasks resulted in magnitudes of heart rate change that differed significantly from others.

Since the above analysis focused on ascertaining changes from baseline without distinguishing the impact of the type of task (i.e., verbal vs non-verbal mental arithmetic), a 2 x 2, Session x Task ANOVA was performed on the mean heart rate responses over all periods of each of the tasks. The source table for this analysis is presented in Table 10.

A significant main effect of Tasks, $F(1,7) = 9.31$, $p < .05$, was revealed, as was a significant Task x Session interaction, $F(1,7) = 10.42$, $p < .05$. Further clarification of the interaction was done employing Tukey's Honestly Significant Difference (Keppel, 1982). A difference of 6.9 between the cell means was required to indicate significance

Table 9

Source Table for Task x Period (Mean Task HR vs Mean Baseline HR) ANOVA.

Source	SS	DF	MS	*F*
Task	643.1	3	214.4	4.1*
Error	1093.7	21	52.1	
Period	1847.6	1	1847.6	38.6**
Error	335.2	7	47.9	
T x P	207.8	3	69.3	2.9
Error	497.8	21	23.7	

*p < .05
**p < .001

Note. Probabilities calculated using Greenhouse-Geisser degrees of freedom adjustments.

Table 10

Source Table for Session x Task ANOVA on Mean HR with Verbal and Non-verbal MA with Repeated Sessions.

Source	SS	DF	MS	F
Session	177.8	1	177.8	1.8
Error	2633.1	7	376.2	
Task	350.1	1	350.1	9.3*
Error	236.3	7	37.6	
S x T	158.9	1	158.9	10.4*
Error	106.7	7	15.2	

*$p < .05$

at the $p < .05$ level of error. Comparison of heart rates with repeated exposure to verbal mental arithmetic separated by a month period yielded no significant differences, while variations in heart rate responses to repeated exposure to non-verbal mental arithmetic were significant.

To permit more fine-grained scrutiny of patterns of heart rate responding to these tasks over a one month period, a 2 x 2 x 10, Session x Task x Period ANOVA was performed. The results of this analysis are presented in Table 11. The source table reveals Task and Period main effects as significant, $F(1,7) = 9.3$ and $F(9,63) = 5.7$, $p < .05$ respectively, while only the Session x Task interaction, $F(1,7) = 10.4$, attained significance at $p < .05$. Employing the Tukey statistic, these findings indicated that, similar to the 2 x 2 Session x Task ANOVA performed above, significant differences between heart rate responses with non-verbal mental arithmetic were seen from Session 1 to Session 2, over each individual 10 second period of the task.

Figures 7 and 8 depict scatterplots of individual heart rate change scores for both repeated verbal and non-verbal tasks administered approximately one month apart, while Table 12 summarizes the data with the mean, range and standard deviation statistics of these scores. The apparent lack of a relationship between individual change scores over a month period is corroborated by correlations of $r = .21$ and $.26$ for verbal and nonverbal mental arithmetic

Table 11

Source Table for Session x Task x Period ANOVA on HR with Verbal and Non-verbal MA with Repeated Sessions.

Source	SS	DF	MS	F
Session	1777.6	1	1777.6	1.8
Error	6778.3	7	968.3	
Task	3500.7	1	3500.7	9.3*
Error	2633.1	7	376.2	
S x T	1588.7	1	1588.7	10.4*
Error	1066.9	7	152.4	
Period	2329.7	9	258.9	5.7*
Error	2882.4	63	45.8	
S x P	530.3	9	58.9	2.5
Error	1483.1	63	23.5	
T x P	129.0	9	14.3	0.7
Error	1397.5	63	22.2	
S x T x P	424.2	9	47.1	1.1
Error	2663.9	63	42.3	

*$p < .05$

Note. Probabilities calculated using Greenhouse-Geisser degrees of freedom adjustments.

respectively, both not significant at the $p < .05$ level.

The mean subjective ratings of task difficulty for verbal mental arithmetic were 4.8 and 4.3 for Sessions 1 and 2 respectively, and 3.9 and 3.8 for non-verbal mental arithmetic. Figure 9 depicts these means for both types of task over the two sessions. A 2 x 2, Session x Task repeated measures ANOVA was subsequently performed, and a summary of this analysis is presented in Table 13. A significant Task main effect, $F(1,7) = 7.63$, $p < .05$, was found, indicating that subjects rated the verbal mental arithmetic as more difficult than the non-verbal task.

Further examination of subjective ratings were conducted employing correlational analyses. Subjective rating versus individual heart rates to the tasks failed to reveal any significant relationships, whether within or across tasks. However, the subjective ratings of verbal and non-verbal mental arithmetic tasks within each session were significantly correlated at $r = .86$ and $.92$, $p = .01$, for sessions 1 and 2 respectively. Furthermore, subjective ratings of repeated verbal mental arithmetic tasks over a one month period were significantly correlated at .82, while non-verbal tasks were correlated at .84, $p < .01$.

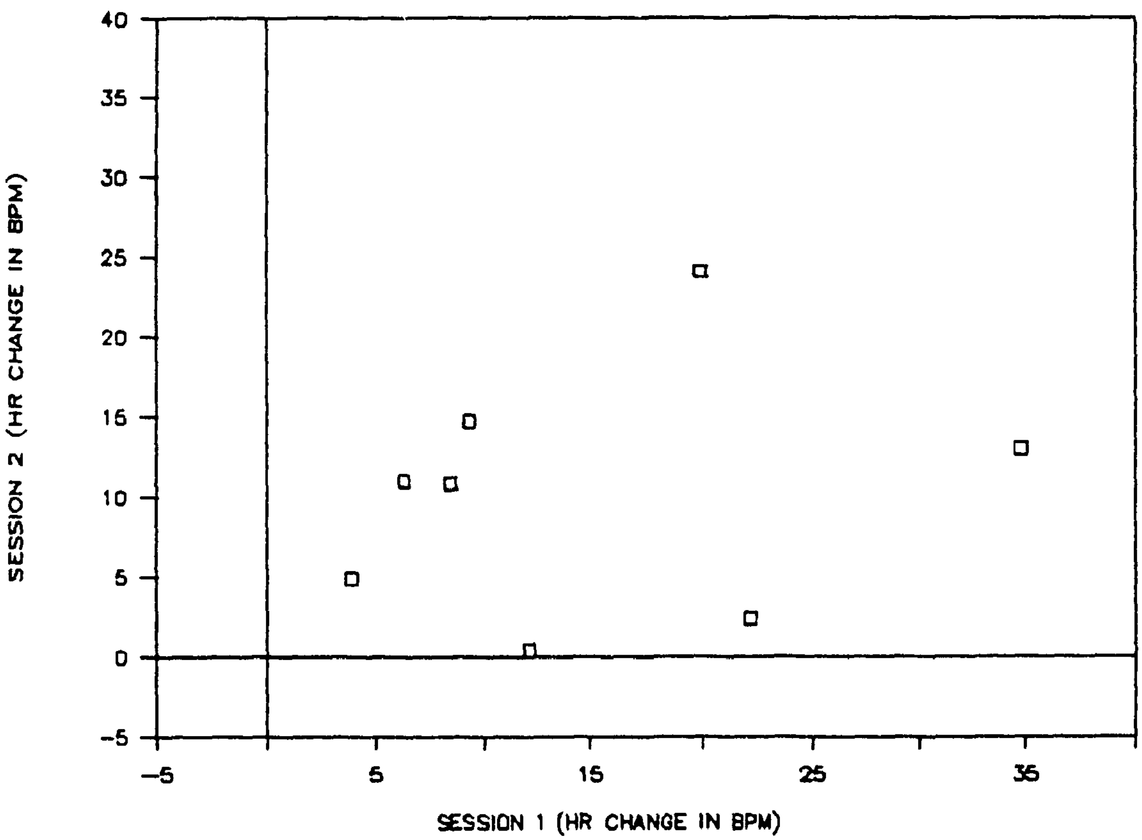


Figure 7. Scattergraph of HR change from baseline during the performance of verbal MA, Session 1 vs Session 2, one month apart.

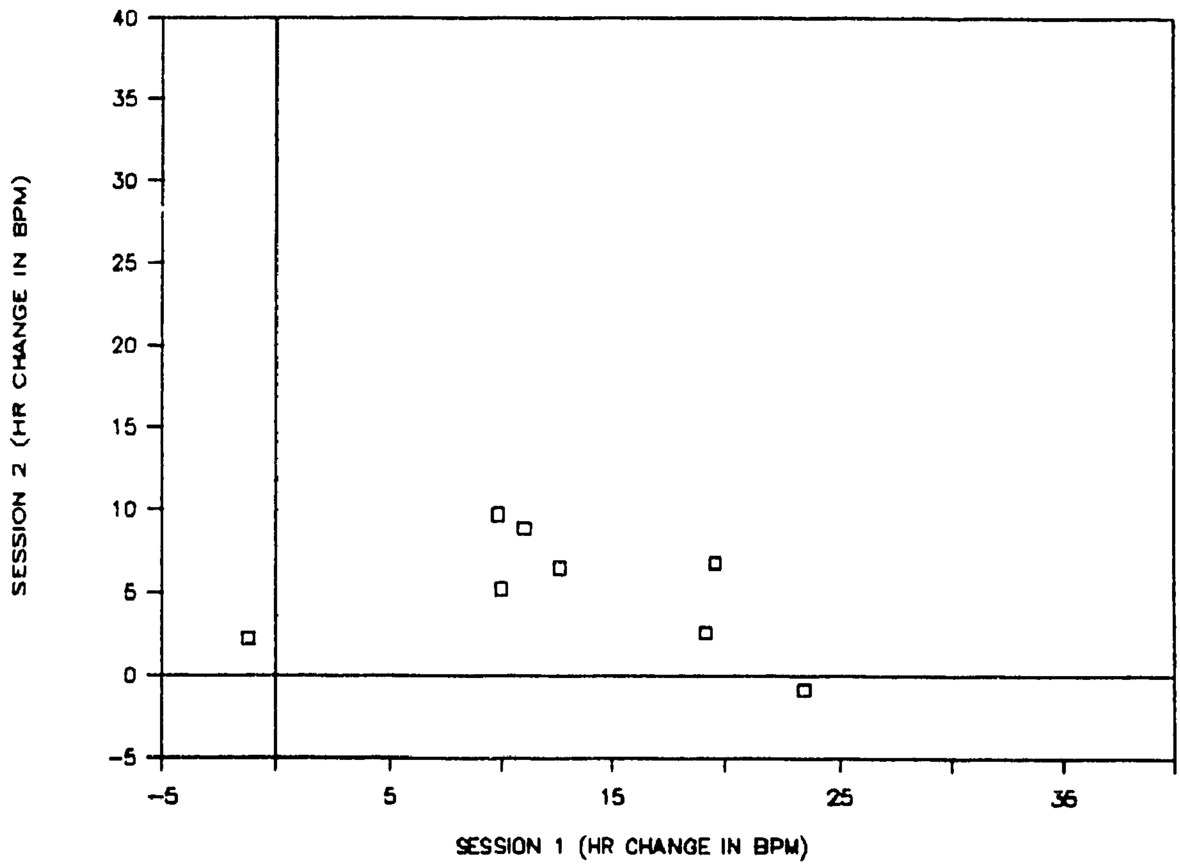


Figure 8. Scattergraph of HR change from baseline during the performance of non-verbal MA, Session 1 vs Session 2, one month apart.

Table 12

Mean, Range and Standard Deviation of Subject HR Change Scores during Verbal and Non-verbal Mental Arithmetic with Repeated Sessions.

	Mean	Range	Standard Deviation
Session 1			
Verbal MA	14.6	30.8	9.7
Non-verbal MA	7.2	24.8	7.2
Session 2			
Verbal MA	10.2	23.7	7.1
Non-verbal MA	5.1	10.5	3.4

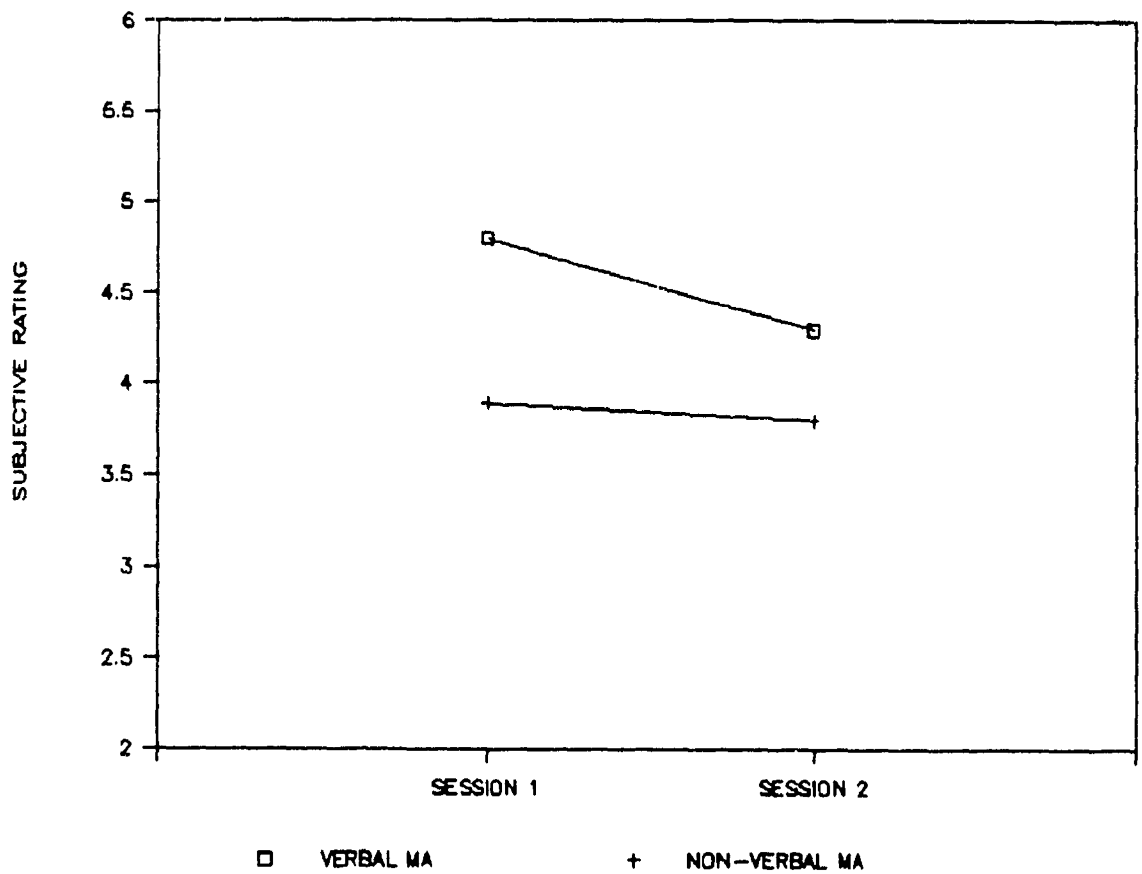


Figure 9. Subjective ratings of task difficulty of verbal and non-verbal MA over Sessions 1 and 2.

Table 13

Mean Subjective Ratings of Task Difficulty and Source Table for Session x Task ANOVA

	Verbal MA	Non-verbal MA
Session 1	4.8	3.9
Session 2	4.3	3.8

Source	SS	DF	MS	F
Session	.8	1	.8	.4
Error	12.5	7	1.8	
Task	3.8	1	3.8	7.6*
Error	3.5	7	.5	
S x T	.3	1	.3	.3
Error	7.0	7	1.0	

*$p < .05$

Note. Probabilities calculated using Greenhouse-Geisser degrees of freedom adjustments.

Discussion

Mental arithmetic, both with and without verbalization of responses, produced significant increases in heart rate. This corroborates the findings of Experiment 2, and further suggests that mental arithmetic tasks elicit reliable increases in heart rate by way of task characteristics other than simply the physical effort of responding verbally.

The statistical significance of these increases was maintained with repeated exposure to the same tasks a month later. That is, though subjects were exposed to two sessions in the laboratory, and were required to perform the stress-inducing tasks four times over the course of the experiment, heart rate remained significantly affected. This suggests that the reactivity seen with these tasks is not attributable to novelty alone.

At the same time, while mental arithmetic with verbalization produced reliable levels of cardiac increases with repeated administration over a one month period, there was a significant decline in heart rate with non-verbal mental arithmetic from the first to second session. It would appear that differences between non-verbal and verbal mental arithmetic tasks might be related to the long-term stability of heart rate responses.

These dissimilar patterns of stability in the context of tasks that vary in their physical demands raises a number of possibilities concerning the precise determinants of reactivity. One explanation for these findings is that verbalization of the responses to mental arithmetic might be contributing significantly to the response stability with this stressor task. If this is indeed the case, the stability of heart rate responses seen in this study as well as others (Robinson et al., 1987; Seraganian et al., 1985) may reflect the consistent physical demands of repeated administrations of the same stressor. When this consistent source of response is minimized as in the present experiment, other patterns of cardiac adaptation emerge over time.

Alternatively, the removal of verbalization of responses may somehow reduce the psychological potency of mental arithmetic, rendering it more vulnerable to habituation. The subjective ratings of non-verbal mental arithmetic, 3.9 and 3.8 for sessions 1 and 2 respectively, did in fact indicate that the subjects in this study perceived the non-verbal mental arithmetic as significantly less challenging than verbal arithmetic, rated at 4.8 and 4.3 for sessions 1 and 2 respectively. These ratings, however, unlike the heart rate responses, were stable. Therefore, at least in terms of the degree of affective disturbance these tasks evoke, no habituation with repeated exposure to the tasks occurred.

Nonetheless, it is possible that repeated exposure to the tasks allowed subjects to anticipate their demands. In the case of non-verbal mental arithmetic, the reduced expectancy of having to produce cogent solutions discernable to the experimenter may be what is reflected in the attenuation of responses over the two sessions. This in turn suggests that neither affective impact nor the cognitive effort of these tasks are sufficient to maintain stable magnitudes of heart rate responding.

The relationship of subjective ratings of task difficulty to heart rate is not clear. Whereas ratings of difficulty for verbal and non-verbal mental arithmetic tasks remained stable over a one month period, heart rate responses to non-verbal arithmetic were seen to decline significantly. Furthermore, heart rates between the verbal and non-verbal tasks in Session 1 were indistinguishable, despite the different subjective ratings given the two tasks. Direct examination of the correlation between individual heart rate responses and subjective ratings also failed to uncover any systematic relationship. Finally, subjective ratings of the difficulty of verbal and non-verbal tasks were highly correlated within each session while highly variable patterns of cardiac responding were observed. Overall, based on the findings of the present experiment, the relationship between subjective perceptions of task difficulty and heart rate responses appears to be both important and complex.

While average heart rate responses to verbal mental arithmetic remained stable over time, the correlational analyses indicated that the responses to either verbal and non-verbal versions of mental arithmetic were not systematically related to responses to the same tasks one month later. This suggests that either subject or task factors may be emerging with repeated exposure to these tasks over time.

General Discussion

In this series of experiments, a number of basic issues underlying a common psychophysiological paradigm were put under empirical scrutiny. Experiment II, by examining the contribution to heart rate of verbalization of responses to mental arithmetic addressed the assumption that autonomic responses to stressful laboratory tasks provide a valid reflection of individual styles of behavioral coping. It has long been argued that if excessive cardiac adjustments are an important behavioral characteristic, an individual would be expected to react in similar ways to various challenges (Light, 1981; Obrist, 1981). However, while some studies have found significant correlations between the cardiac responses to various tasks (Light, 1981; Manuck & Garland, 1980), others have not (Carroll, Turner & Rogers, 1987; Carroll, Turner & Hellawell, 1986).

In a recent re-examination of the data emerging from this literature, Turner (1988) has posited that inter-task correlations are seen to improve as a function of two factors: 1) when the tasks are similar in their physical demands; and 2) when the predicted heart rate reactivity due to the physical effort of different stressful tasks is statistically accounted for. The concerns for the degree to which inter-task consistency may depend on methodological factors, such as the choice of stressors rather than individual differences, are supported by the present findings. The physical effort expended in verbalizing responses to mental arithmetic appears to contribute significantly to measures of heart rate reactivity.

While this finding is consistent with prior work from this laboratory, important differences exist that warrant commentary. In the Brown et al. (1988) study, simple verbalization resulted in significant increases in heart rate over baseline levels in five of ten 10-second periods of the task. The present study, in contrast, revealed that non-verbal mental arithmetic resulted in significant increases uniformly throughout the duration of the task. Thus, the demands of mental arithmetic appear to be a more consistent elicitor of increased heart rate activity than simple verbalization.

Both the present results as well as those of Brown et al. (1988) contradict previous arguments that speech contributes little to reactivity (Linden, in press). However, differences in data reduction methodologies may be posited to explicate the discrepant findings. Heart rate in Linden's (1987) study was expressed as an overall average figure over the duration of each task. In the present study, heart rate reactivity expressed as an overall average measure also failed to reveal significant differences between verbal and non-verbal mental arithmetic tasks. However, differences did emerge when the fine-grained reactivity profiles were scrutinized. Given the variability of fine-grained techniques of heart rate measurement and the rudimentary nature of our knowledge of the nature of reactivity, continued reliance on gross response measures that may ignore subtle but important information appears unwise.

Another fundamental issue addressed in Experiment III of this study concerns the reliability of autonomic responses to stress. The present findings indicated that while heart rate response to verbal mental arithmetic was stable over a period of a month, mental arithmetic without verbalization resulted in a significant attenuation in responding. Furthermore, this effect occurred although the subjective experience of task challenge did not change.

The reliability of heart rate responses to stressful tasks has been well documented (Drummond, 1985; McKinney et al., 1985; Robinson et al., 1987). Therefore, in part, the findings of Experiment III adds to a growing literature supporting the notion of stability of heart rate responses to stress. The failure to see stable responses to non-verbal mental arithmetic, on the other hand, presents a number of interesting possibilities. Experiments II and III examined the stability of heart rate to stress while attempting to exert greater control over the potential contribution of physical task demands. The studies supporting stability have involved tasks requiring some physical action, such as verbalization or hand gestures, to meet their cognitive challenges. By removing that portion of heart rate responding attributable to physical sources, more accurate observations of the processes involved in reactivity to stressful tasks may have emerged.

The present findings suggest that verbalization may interact in some manner with the cognitive challenge of the task to produce a more robust pattern of autonomic responding. Several psychological features of laboratory stressors have been posited to contribute to their potency, including apprehension, emotional disturbance, cognitive activity, and social context (Krantz & Manuck, 1984). In this case, it may be the social implications of verbal responding to cognitively challenging tasks in close proximity to an experimenter that may be contributing to consistent magnitudes of heart rate reactivity.

The findings of Experiments II and III suggest a number of areas for future inquiry. First, the results indicate that unravelling the precise determinants of reactivity to stressful tasks is possible. Further research might continue to clarify the precise manner in which different laboratory tasks evoke their characteristic patterns of autonomic responding. With such an approach, several commonly employed stressful tasks might be compared for their relative potency in producing true psychological (i.e., versus physically-mediated) reactivity. Second, the design and standardization of a battery of laboratory stressors could be undertaken in which the magnitude of response attributable to their specific physical and psychological characteristics are clearly known. Overall, knowledge of the manner in which different stressors elicit reactivity, or alternately, the use of such a standardized battery would permit more valid interpretations of psychophysiological phenomena and enhance the integration of data emerging from the many laboratories engaged in this area.

Several characteristics of baseline heart rate measurement were clarified in Experiment I. First, heart rates measured when subjects sat quietly in the laboratory with no anticipation of stress appear to be markedly stable. The absence of significant fluctuations in heart rate under these conditions was found over several minutes within a single session, as well as over a month period with repeated sessions. This finding suggests that as a method of baseline determination, this procedure does not appear to be vulnerable to significant habituation over either short intervals of time lasting several minutes, or over longer periods of up to a month.

In addition, fine-grained analysis revealed several other important characteristics of baseline determined in this fashion. Heart rate seen in the first 10-second period after an initial 10 minute rest period remains stable over all subsequent measurements. This suggests that resting periods preceding data collection need not last longer than 10 minutes in order for a stable baseline measure to be established.

Hastrup (1986) has posited that a 15 minute waiting period might be necesssary before a stable baseline heart rate lower than 70 beats per minute could be seen. The data emerging from this study, however, suggest that highly stable baseline heart rates lower than 70 beats per minute may be observed after only 10 minutes of rest. Thus, it would appear that the act of simply sitting in the laboratory does not yield enduring elevations in cardiac activity.

The comparison between baselines obtained in Experiments I and II provides qualified support for the contention that pre-stress baselines are more susceptible to increases in heart rate (Obrist, 1981; Light & Obrist, 1980 b). In Experiment II, baseline heart rate gathered prior to stress yielded average measures 10 beats higher than those seen in Experiment I in the absence of stress. This appears to corroborate the findings of Whitsett et al. (in press) and Kjellberg & Magnusson (1979) where pre-stress baselines produced significantly higher heart rates than when no stress was presented.

It might be asked whether the present findings justify the extra effort and additional session required to acquire this baseline measure. Baseline measures taken when stress is to be presented were also seen to be stable over short and long durations in Experiments II and III. These findings correspond to those of other authors studying the stability of pre-stress baseline heart rate (Fahrenburg et al., 1987; McKinney et al., 1985). As well, correlation coefficients of individual baseline heart rate over short and long durations are of approximately the same magnitude whether stress is expected or not. Based on the stability of baseline measures, there may be little advantage to one method over another.

While the LIV states that initial resting heart rate predicts subsequent acute measures of reactivity, this principle enjoys only equivocal support. For example, patterns of reactivity are often opposite to that predicted by the LIV, and have led one author (Jamieson, 1988) to suggest that observations of support for the LIV are attributable to either unreliable measurement and/or artifacts of the scale of measurement. Other authors suggest that the discrepancy in the findings for the LIV may arise from data based on either within-subject as opposed to a between-subject methodologies, and have suggested that for the present, the LIV be degraded from a methodological rule to the status of an empirical generalization to be differentially tested (Furedy & Scher, 1988).

Overall, based on the present findings, future research into baseline protocols might focus on two areas of inquiry: 1) the two baseline protocols could be compared employing a counterbalanced repeated measures within-subject design; and 2) on-going investigation into the parametric and organismic significance of the LIV as related to heart rate measures. Further clarification of these areas might provide insight into the appropriateness and comparability of the two baseline methodologies.

In addition to clarification of the impact of certain laboratory protocols upon heart rate reactivity, the present findings also hold broader conceptual and theoretical implications for the study of psychophysiological phenomena. It has been hypothesized that the study of psychophysiologic responses to challenges in the laboratory may provide important information regarding the role of psychological stress in the development of coronary heart disease (CHD) (Krantz & Manuck, 1984). However, there are a number of critical assumptions that underlie the interpretation of observations derived from the laboratory.

One such assumption is that measures of cardiovascular adjustment seen in the laboratory generalize to daily life: that is, the examination of acute responses to challenges in the laboratory predict those seen in the normal functioning of the individual. The methodological issues involved in baseline determination explored in Experiment I may be seen in light of the assumption that a 'true' baseline measure represents a resting, lowest level of cardiac activity. While obtaining the lowest resting heart rate is thought to optimize the response resolution when stress is subsequently presented, contriving the situation to accomplish this may be at the expense of the generalizability of this paradigm.

Two models have been posited (Manuck & Krantz, 1984) to describe the relationship between responses in the laboratory with those seen in real-life: the 'recurrent activation' model, and the 'prevailing state' model. The former hypothesizes that acute responses in the laboratory accurately reflect those seen in real life. Resting baseline levels of cardiac activity are seen as the normal state of the organism, interspersed with transient acute reactions to challenges in either the laboratory or daily life. Establishing a low resting level of cardiac activity, therefore, is essential for subsequent observation of responses to stress.

Alternatively, others (Manuck & Krantz, 1984) have argued that resting baseline measurements gathered in this fashion bear little resemblance to real life but rather are an artifact of the relatively stimulus-free laboratory environment. The 'prevailing state' model posits that acute responses to stress in the laboratory are more accurate in describing the normal state of the individual in the environment. As such, absolute measures of cardiac activity are more appropriate in describing individual responses to stress in daily life than the acute changes studied in the recurrent activation model.

There has been little empirical exploration of the manner in which laboratory observations of reactivity generalize to a real life setting (Manuck & Krantz, 1986). In the absence of clarification of which model, if either, more accurately reflects the functioning of the individual outside the laboratory, the relative value of a baseline determination in a more or less stimulus-free environment remains speculative at present. Future research might be directed towards telemetric monitoring of heart rate responding during normal waking life to obtain a better understanding of its relationship to data gathered in the controlled environment of the laboratory.

Another assumption involves the importance of stress-induced alterations in cardiovascular functioning in the development of CHD. Several mechanisms linking acute psychophysiological reactivity and the development of CHD have been hypothesized, and include neuroendocrine mediation of changes in the vascular tissue (Herd, 1986), and atherosclerotic lesion formation through haemodynamic processes (Clarkson, Manuck & Kaplan, 1986).

One interesting area of recent exploration concerns the role of myocardial ischemia in the development of CHD. The frequent occurences of myocardia ischemia in coronary patients, particularly in response to both psychological and physical stress, have been cited as a link between psychophysiological responses and CHD. A recent study (Rozanski et al., 1988) employed radionuclide ventriculography in order to explore the impact of a variety of physical and mental stressors in producing acute ischemic episodes. Evidence of ischemia was found to occur more frequently in coronary patients than in controls with the relatively small increases in heart rate accompanying public speaking, mental arithmetic and Stroop colour-word tasks.

Some authors have pointed out that the stressors employed in the above study involved verbalization. Citing the literature supporting the increases in cardiovascular activity and the occurence of cardiac anomalies seen with simple speech, Rosch and his colleagues (1988) further suggest that until the precise precipitants of cardiovascular reactivity are understood, the significance of psychological stress in the development of CHD remains unclear.

The results of Experiment II bear on this issue. In that experiment, mental arithmetic both with and without verbalization produced significant increases in mean heart rate over resting baseline levels. These increases, approximately 14 bpm and 10 bpm with verbal and non-verbal mental arithmetic respectively, are similar in magnitude to the 10 bpm observed in response to mental arithmetic by Rozanski and his colleagues (1988) and that were also accompanied by ischemic episodes. It is therefore probable that the increases in heart rate seen in the present study with non-verbal mental arithmetic would yield similar evidence of potentially pathogenic activity in high-risk individuals.

It appears then, contrary to the caveats of Rosch et al. (1988), that psychological stress 'suffered in silence' is sufficient in itself to produce significant alterations in cardiac activity. Given the potential importance to heart health of cardiovascular adaptations to mental stress, greater attention is warranted to identify which aspects of psychological stressors employed in the laboratory potentiate specific mechanisms of cardiovascular activity.

References

Allen, M.T., Obrist, P.A., Sherwood, A., and Cromwell, M.D. (1986). Two year stability of cardiovascular responses to reaction time and cold pressor tasks. Psychophysiology, 23, 423. (From Abstracts from the Twenty-sixth Annual Meeting of the Society of Psychophysiology Research, 1986, 412-496).

Astrand, P. and Rhyming, L. (1954). A nomogram for calculation of aerobic capacity (physical fitness) from pulse rate during submaximal work. Journal of Applied Physiology, 7, 218-221.

Berne, R.M. and Levy, M.N. (1977). Cardiovascular Physiology. The C.V. Mosby Co.: Saint Louis.

Brown, T.G., Szabo, A., and Seraganian, P. (1988). Physical versus psychological determinants of heart rate reactivity to a mental stressor. Psychophysiology, 25, 532-537.

Bunnell, D.E. (1982). Autonomic myocardial influences as a factor determining inter-task consistency of heart rate reactivity. Pychophysiology, 19, 442-448.

Carroll, D., Turner, J.R., and Hellawell, J.C. (1986). Heart rate and oxygen consumption during active psychological challenge: The effects of level of difficulty. Psychophysiology, 23, 174-181.

Carroll, D., Turner, J.R. and Rogers, S. (1987). Heart rate and oxygen consumption during mental arithmetic, a video game, and graded static exercise. Psychophysiology, 24, 112-118.

Clarkson, T.B, Manuck, S.B, and Kaplan, J.R. (1986). Potential role of cardiovascular reactivity in artherogenesis. In K.A. Matthews, S.M. Weiss, T. Detre, T.M. Dembroski, B. Falkner, S.B. Manuck, and R. B. Williams Jr. (Eds.), Handbook of stress, reactivity, and cardiovascular disease (pp. 35-48). New York: Wiley-Interscience.

Drummond, P.D. (1985). Cardiovascular reactivity in borderline hypertensives during behavioral and orthostatic stress. Psychophysiology, 22, 621-627.

Fahrenberg, J., Schneider, H.J. and Safian, P. (1987). Psychophysiological assessments in a repeated-measurement design extending over a one-year interval: Trends and stability. Biological Psychology, 24, 49-66.

Fredrickson, M., Danielssons, T., Engel, B.T., Frisk-Homberg, M., Strom, G. and Sundin, O. (1985). Autonomic nervous system function and essential hypertension: Individual response specificity with and without beta-adrenergic blockade. Psychophysiology, 22, 167-174.

Friedmann, E., Thomas, S.A., Kulick-Ciuffo, D., Lynch, J.J., and Suginohara, M. (1982). The effects of normal speech on blood pressure. Psychosomatic Medicine, 44, 334-343.

Foerster, F. (1985). Psychophysiological response specificities: A replication over a 12-month period. Biological Psychology, 21, 169-182.

Furedy, J.J. and Scher, H. (1989). The law of initial values: Differentiated testing versus enshrinement as a methodological rule. Psychophysiology, 1, 120-122.

Giordani, B., Manuck, S.B., and Farmer, J.K. (1981). Stability of behaviorally induced heart rate changes in children after one week. Child Development, 52, 533-537.

Hassett, J. (1978). A primer of psychophysiology. San Francisco: W.H. Freeman and Co.

Hastrup, J.L. (1986). Duration of initial heart rate assessment in Psychophysiology: Current practices and implications. Psychophysiology, 23, 15-18.

Hastrup, J. and Light, K. (1984). Sex differences in cardiovascular stress responses: Modulation as a function of menstrual cycle phases. Journal of Psychosomatic Research, 28, 475-483.

Herd, J.A. (1986). Neuroendocrine mechanisms in coronary heart disease. In K.A. Matthews, S.M. Weiss, T. Detre, T.M. Dembroski, B. Falkner, S.B. Manuck, and R. B. Williams Jr. (Eds.), Handbook of stress, reactivity, and cardiovascular disease (pp. 49-70). New York: Wiley-Interscience.

Houston, B.K. (1986). Psychological variables and cardiovascular and neuroendocrine reactivity. In K.A. Matthews, S.M. Weiss, T. Detre, T.M. Dembroski, B. Falkner, S.B. Manuck, and R. B. Williams Jr. (Eds.), Handbook of stress, reactivity, and cardiovascular disease (pp. 207-229). New York: Wiley-Interscience.

Jamieson, J.. (1988). The law of initial values: Influences of unreliability and skewness. Psychophysiology, 4, 432.

Jamieson, J.L. and Lavoie, N. (1987). Type A behavior. aerobic power and cardiovascular recovery from psychosocial stressor. Health Psychology, 6, 361-371.

Keppel, G. (1982). Design & Analysis: A researcher's handbook (Second edition). New Jersey: Prentice-Hall.

Kjellberg, A. and Magnusson, E. (1979). Physiological response patterns during 'intake' and 'rejection' tasks. Biological Psychology, 9, 63-76.

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

Krantz, D.S., Manuck, S.B., and Wing, R.R. (1986). Psychological stressors and task variables as elicitors of reactivity. In K.A. Matthews, S.M. Weiss, T. Detre, T.M. Dembroski, B. Falkner, S.B. Manuck, and R. B. Williams Jr. (Eds.), Handbook of stress, reactivity, and cardiovascular disease (pp. 461-474). New York: Wiley-Interscience.

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

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

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

Light, K.C. and Obrist, P.A. (1983). Task difficulty, heart rate reactivity and cardiovascular responses to an appetitive reaction time task. Psychophysiology, 20, 301-312.

Linden, W. (1985). Cardiovascular response as a function of predisposition, coping behavior and stimulus type. Psychosomatic Medicine, 29, 611-620.

Linden, W. (1986). Mental stress versus somatic effort: A microanalysis of cardiovascular responsivity. Psychophysiology, 23, 448. (From Abstracts from the Twenty-sixth Annual Meeting of the Society of Psychophysiology Research, 1986, 412-496).

Linden, W. (1987). A microanalysis of autonomic activity during human speech. Pychosomatic Medicine, 6, 562-578.

Linden, W. and Frankish, J. (1988). Expectancy and type of activity: Effects on pre-stress cardiovascular adaptation. Biological Psychology, 227, 227-235.

Long, J.M., Lynch, J.J., Machiran, N.M., Thomas, S.A., and Malinow, K.L. (1982). The effect of status on blood pressure during verbal communication. Journal of Behavioral Medicine, 5, 165-172.

Lynch, J.J. (1985). Language of the heart: The body's response to human dialogue. New York: Basic Books.

Lynch, J.J., Thomas, S.A., Long, J.M., Malinow, K.L., Friedmann, E., and Katcher, A.H. (1982). Blood pressure changes while talking. Israel Journal of Medical Sciences, 18, 575-579.

Manuck, S.B. and Garland, F.N. (1980). Stability of individual differences in cardiovascular reactivity: A thirteen month follow-up. Physiology and Behavior, 21, 621-624.

Manuck, S.B. and Krantz, D.S. (1984). Psychophysiologic reactivity in coronary heart disease. Behavioral Medicine Update, 6, 11-15.

Manuck, S.B. and Krantz, D.S. (1986). Reactivity, Coronary heart disease, and essential hypertension. In K.A. Matthews, S.M. Weiss, T. Detre, T.M. Dembroski, B. Falkner, S.B. Manuck, and R. B. Williams Jr. (Eds.), Handbook of stress, reactivity, and cardiovascular disease (pp. 11-34). New York: Wiley-Interscience.

Mathews, D.K. and Fox, E.L. (1976). The physiological basis of physical education and athletics (2nd edition). Philadelphia: Saunders.

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

Matthews, K.A. (1986). Summary, Conclusions and Implications. In K.A. Matthews, S.M. Weiss, T. Detre, T.M. Dembroski, B. Falkner, S.B. Manuck, and R. B. Williams Jr. (Eds.), Handbook of stress, reactivity, and cardiovascular disease (pp. 461-474). New York: Wiley-Interscience.

Matthews, K.A., Rakaczky, C.J., Stoney, C.M. and Manuck, S.B. (1987). Are cardiovascular responses to behavioral stressors a stable individual difference variable in childhood. Psychophysiology, 4, 464-473.

McKinney, M.E., Miner, M.H., Ruddell, H., McIlvain, H.E., Witte, H., Buell, J.C. and Eliot, R.S. (1985). The standardized mental stress test protocol: Test-retest reliability and comparison with ambulatory blood pressure monitoring. Psychophysiology, 22, 453-463.

Meyers, A.W. and Craighead, W.E. (1978). Adaptation periods in clinical psychophysiology research. Behavior Therapy, 9, 355-362.

Myrtek, M., and Spital, S. (1986). Psychophysiological response to single, double, and triple stressors. Psychophysiology, 23, 663-671.

Nadell, E.R. (1985). Physiological adaptations to aerobic training. American Scientist, 73, 324-343.

Obrist, P.A. (1976). The cardiovascular-behavioral interaction as it appears today. Psychophysiology, 13, 95-107.

Obrist, P.A. (1981). Cardiovascular Physiology. New York: Plenum Press.

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

Psychophysiology, 15, 102-115.

Obrist, P.A., Lawler, J.E., Howard, J.L., Smithson, K.W., Martin, P.L. and Manning, J. (1974). Sympathetic influences on cardiac rate and contractility during acute stress in humans. Psychophysiology, 11, 405-427.

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

Robinson, J.W., Whitsett, S.F., and Kaplan, B.J. (1987). The stability of physiological reactivity over multiple sessions. Biological Psychology, 24, 129-139.

Rosch, P.J., Katcher, A.H., Malinow, K.L. and Elkes, J. (1988). Speaking stress, and the induction of myocardial ischemia. The New England Journal of Medicine, 13, 867-868.

Rozanski, A., Bairey, C.N., Krantz, D.S., Friedman, J., Resser, K.J., Morrell, M., Hilton-Chalfen, S., Hestrin, L., Bietendorf, J., and Berman, D.S. (1988). Mental stress and the induction of silent myocardial ischemia in patients with coronary artery disease. The New England Journal of Medicine, 16, 1005-1012.

Scher, H., Furedy, J.J., and Heslegrave, R.J. (1985). Individual differences in phasic cardiac reactivity to psychological stress and the Law of Initial Value. Psychophysiology, 22, 345-348.

Schneiderman, N. and Pickering, T.G. (1986). Cardiovascular measures of physiologic reactivity.In K.A. Matthews, S.M.

Weiss, T. Detre, T.M. Dembroski, B. Falkner, S.B. Manuck, and R. B. Williams Jr. (Eds.), Handbook of stress, reactivity, and cardiovascular disease (pp. 145-186). New York: Wiley-Interscience.

Schuri, U. and von Cramon, D. (1981). Heart rate and blink rate responses during mental arithmetic with and without continuous verbalization of results. Psychophysiology, 18, 650-653.

Seraganian, P., Hanley, J.A., Hollander, B.J., Roskies, E., Smilga, C., Martin, N., Collu, R., and Oseason, R. (1985). Exaggerated psychophysiological reactivity: Issues in quantification and reliability. Journal of Psychosomatic Research, 29, 393-405.

Sherwood, A., Allen, M.T., Obrist, P.A. and Langer, A.W. (1986). Evaluation of beta-adrenergic influences on cardiovascular and metabolic adjustments to physical and psychological stress. Psychophysiology, 23, 89-104.

Sinyor, D., Golden, M., Steinert, Y., and Seraganian, P. (1986). Experimental manipulation of aerobic fitness and the response to psychosocial stress: Heart rate and self-report measures. Psychosomatic Medicine, 48, 324-337.

Stoney, C.M., Langer, A.W. and Gelling, P.D. (1986). The effects of menstrual cycle phase on cardiovascular and pulmonary responses to behavioral and exercise stress. Psychophysiology, 23, 393-402.

Thomas, S.A., Lynch, J.J., Friedmann, E., Suginohara, M., Hall, P.S. and Peterson, C. (1984). Blood pressure and heart rate changes in children when they read aloud in school. Public Health Reports, 99, 77-84.

Turner, J. R. and Carroll, D. (1985). Heart rate and oxygen consumption during mental arithmetic, a video game, and graded exercise: Further evidence of metabolically-exaggerated cardiac adjustment? Psychophysiology, 22, 261-267.

Turner, J.R. (1988). Inter-task consistency: An integrative re-evaluation. Psychophysiology, 25, 235-238.

Vasey, M. and Thayer, J.T. (1987). The continuing problem of false positives in repeated measures ANOVA in psychophysiology: A multivariate solution. Psychophysiology, 4, 479-486.

Weiss, S.M. (1986). Introduction and overview. In K.A. Matthews, S.M. Weiss, T. Detre, T.M. Dembroski, B. Falkner, S.B. Manuck, and R. B. Williams Jr. (Eds.), Handbook of stress, reactivity, and cardiovascular disease (pp. 1-10). New York: Wiley-Interscience.

Whitsett, S.F., Robinson, J.W., and Kaplan. (In press). A comparison of three approaches for the determination of baseline levels of physiologic activity. International Journal of Psychophysiology.

Appendix 1

Sample of Heart Rate Transformed to Beat-By-Beat Analogue and Digital Outputs.

1955	89
1970	87
1982	85
1986	84
1977	86
1990	84
1994	83
1986	84
1966	88
1943	91
1938	92
1916	96
1921	95
1953	90
1990	84
1993	83
1966	88
1952	90
1927	94
1911	96
1914	96
1933	93
1996	83
2023	79
2019	79
2017	80
1988	84
1968	87
1947	91
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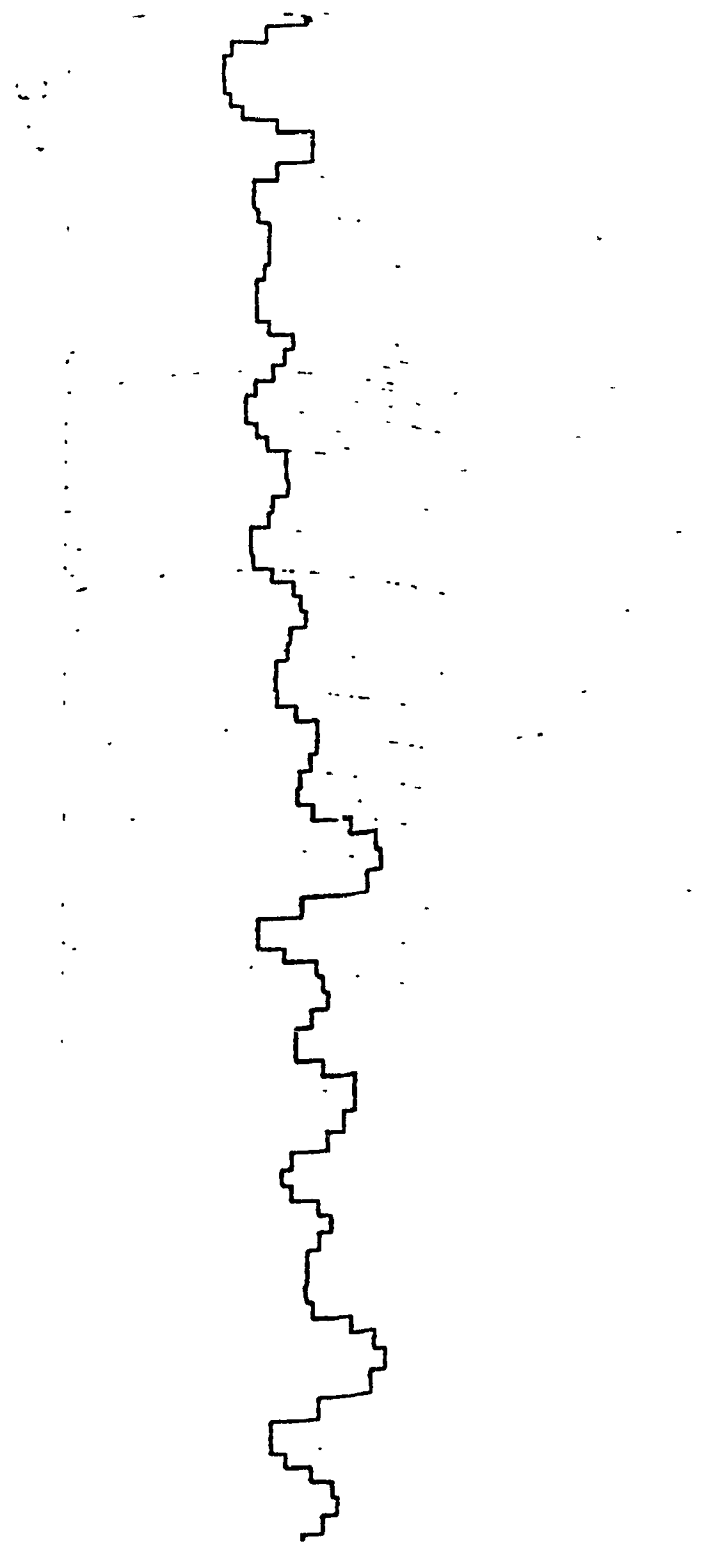
1966	88
1975	86
1976	86
1981	85
1978	86



Appendix 2

Informed Consent Form

This phase of our psychophysiological research requires subjects to come into the laboratory on two separate occasions, one month apart. Therefore, someone will contact you to make another appointment in a few weeks.

You are asked to simply sit quietly in the enclosed chamber for approximately 20 minutes. During this time, your heart rate will be monitored using electrodes stuck to the surface of your chest. Nothing more is asked of you than to simply relax, and to move as little as comfortably possible. The experimenter will tell you when the session is over. If at any time, for whatever reason, you wish to terminate the session, indicate this to the experimenter and the session will be aborted immediately.

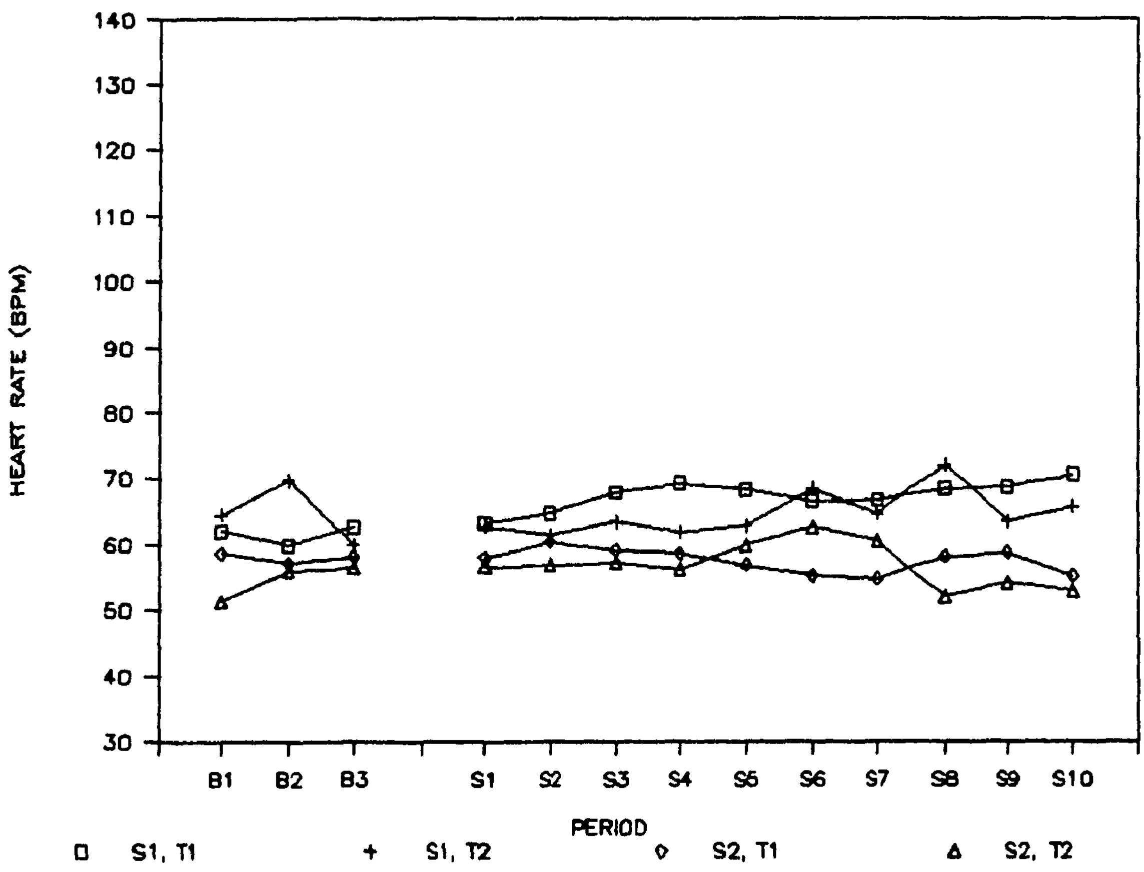
If you consent to participate and do not suffer any medical conditions that might contradict your involvement (i.e., any medical history of heart disease, hypertension, dizziness, chest pains, currently taking medications of any kind), please sign your name below.

Signature:__________________________; Date:______________

Age:___________________; Tel.:___________________________.

Appendix 3

Case by case heart rate data for all subjects (N = 11) employed in Experiment 1, with interbeat intervals over ten second intervals averaged over 13 periods (i.e., B1 to B3, S1 to S10). S1 and S2 refers to first or second repeated sessions respectively one month apart, while T1 and T2 refers to first or second data-logging trial respectively within each session.


140
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60
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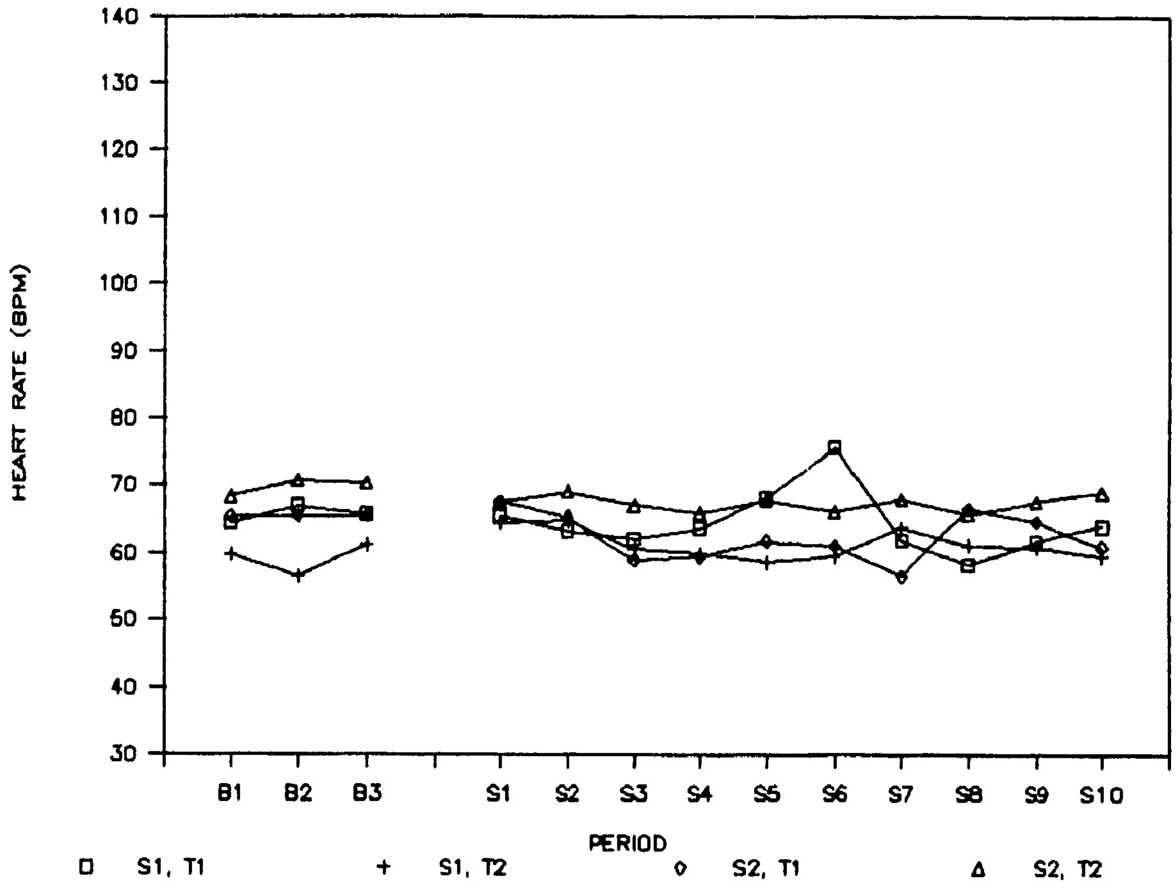
40
30
HEART RATE (BPM)
B1 B2 B3 S1 S2 S3 S4 S5 S6 S7 S8 S9 S10
PERIOD
□ S1, T1
+ S1, T2
◇ S2, T1
△ S2, T2

Subject I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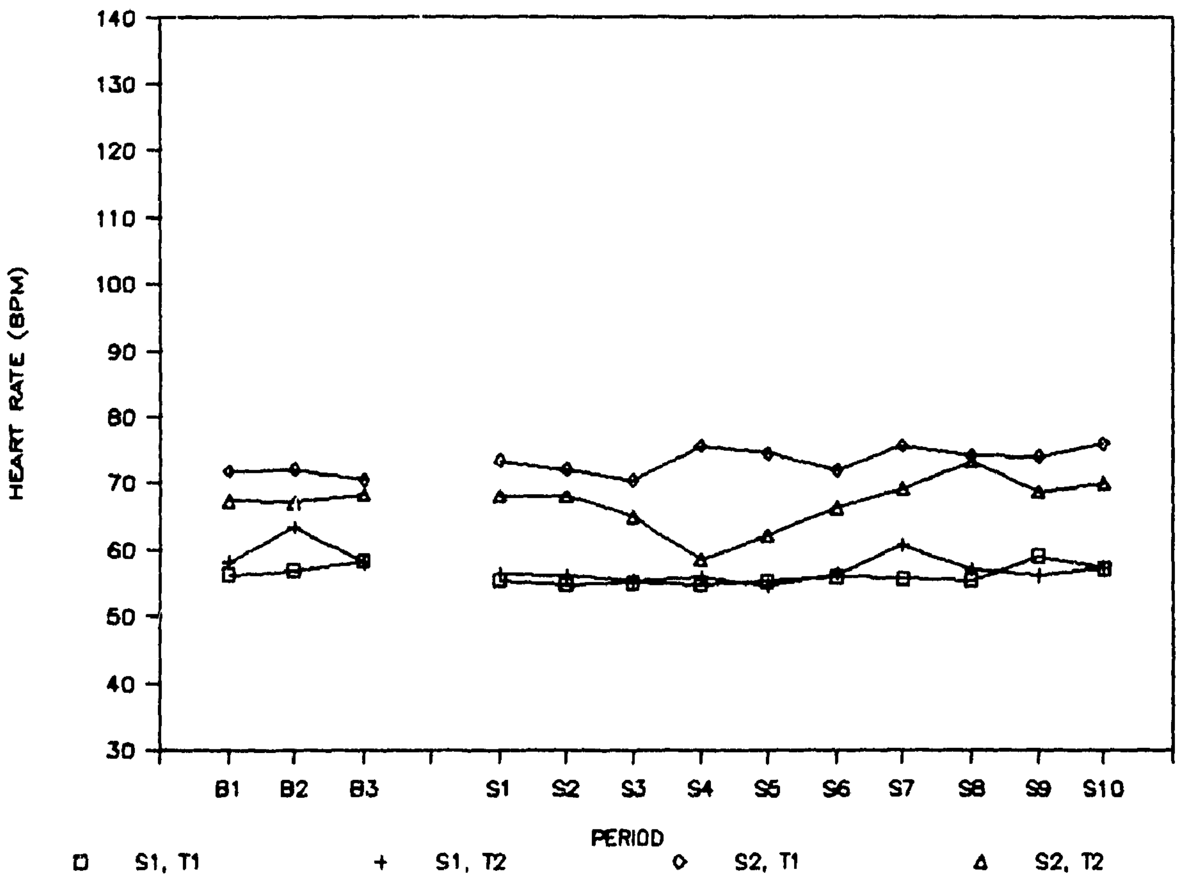
30
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
S1, T1
S1, T2
S2, T1
S2, T2

Subject 2



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60
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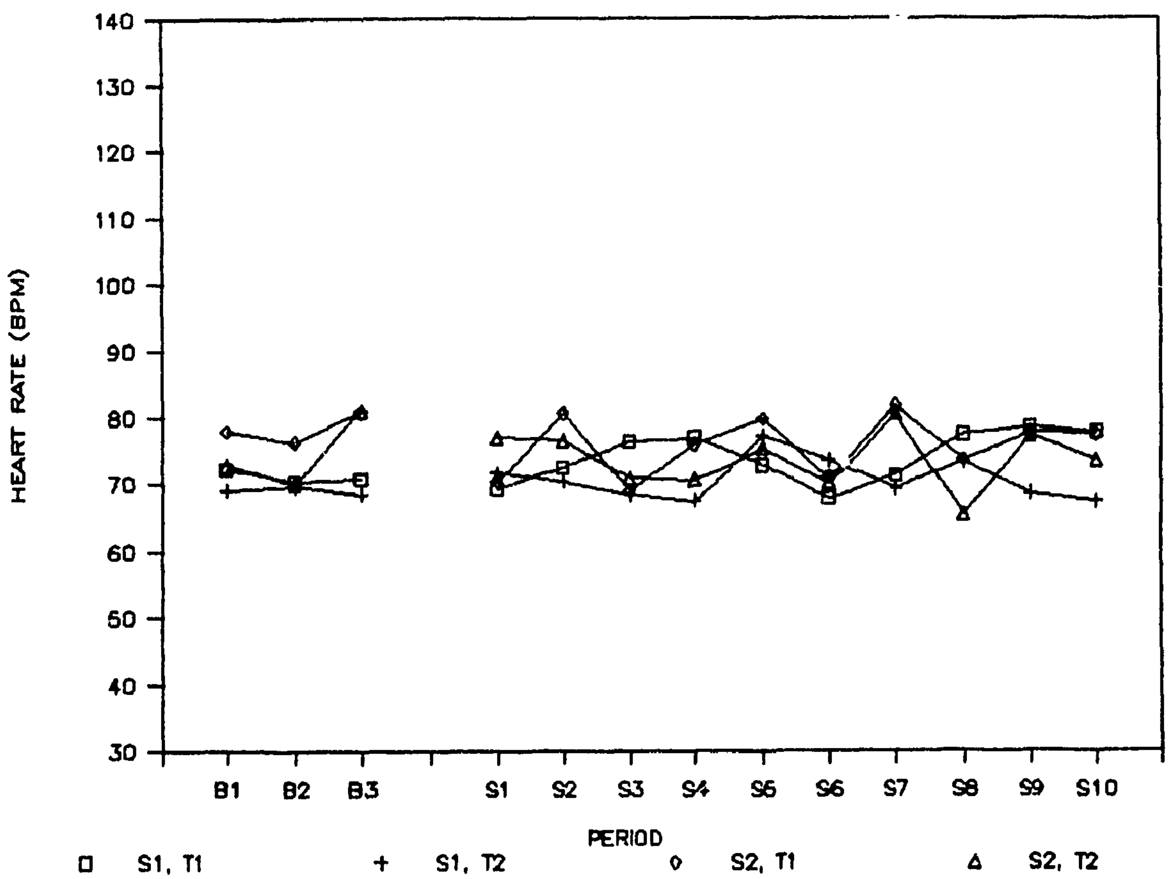
30
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
S1, T1
S1, T2
S2, T1
S2, T2

Subject 3



HEART RATE (BPM)
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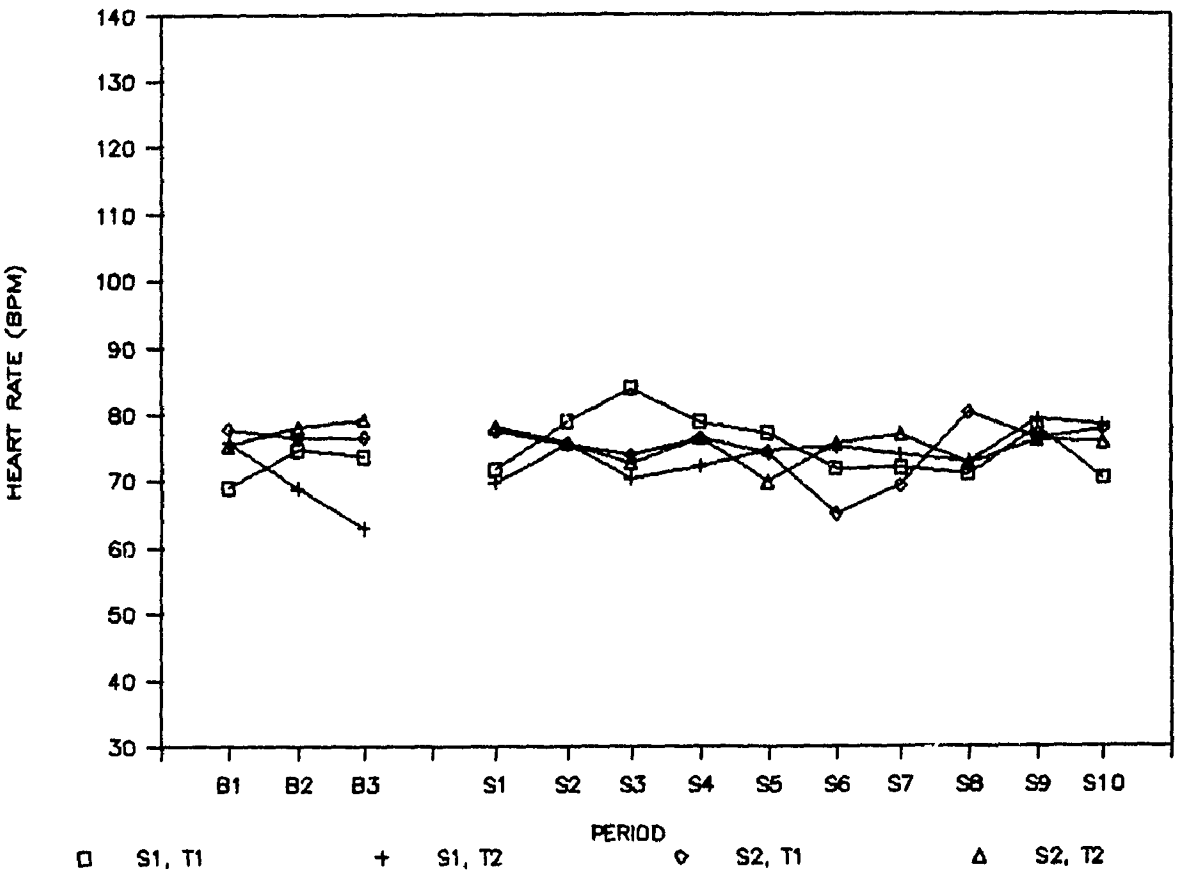
60
50
40
30
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
S1, T1
S1, T2
S2, T1
S2, T2

Subject 4



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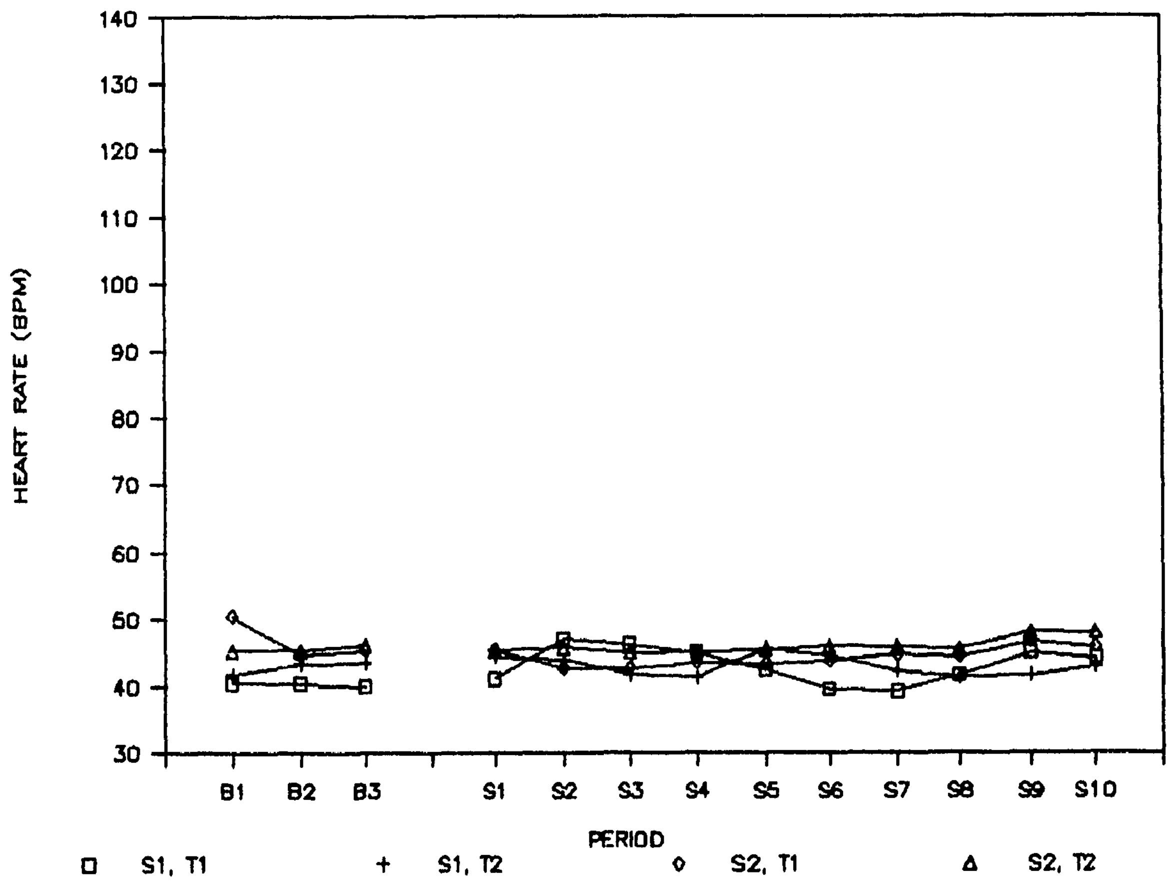
30
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
S1, T1
S1, T2
S2, T1
S2, T2

Subject 5



HEART RATE (BPM)
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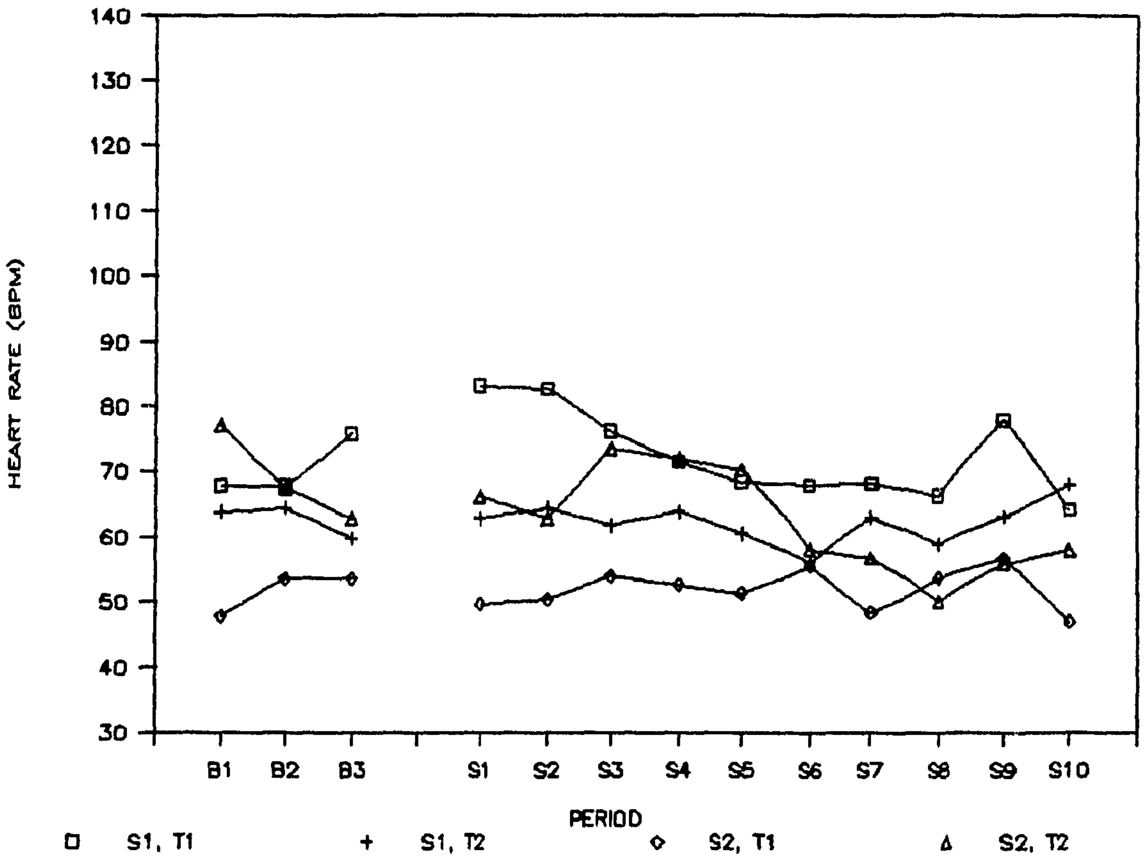
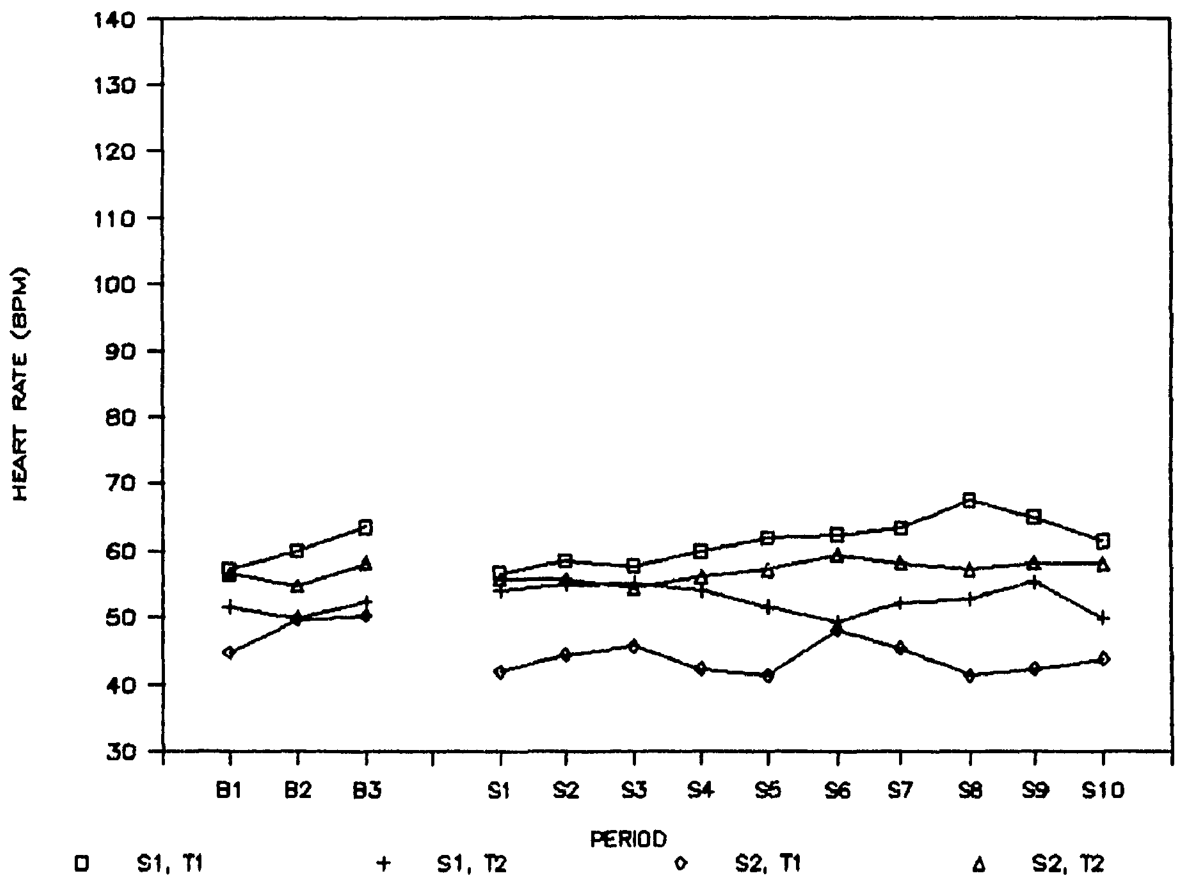
60
50
40
30
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
S1, T1
S1, T2
S2, T1
S2, T2

Subject 6


HEART RATE (BPM)
140
130
120
110
100
90
80
70
60
50
40
30
B1 B2 B3 S1 S2 S3 S4 S5 S6 S7 S8 S9 S10
PERIOD
S1, T1
S1, T2
S2, T1
S2, T2

Subject 7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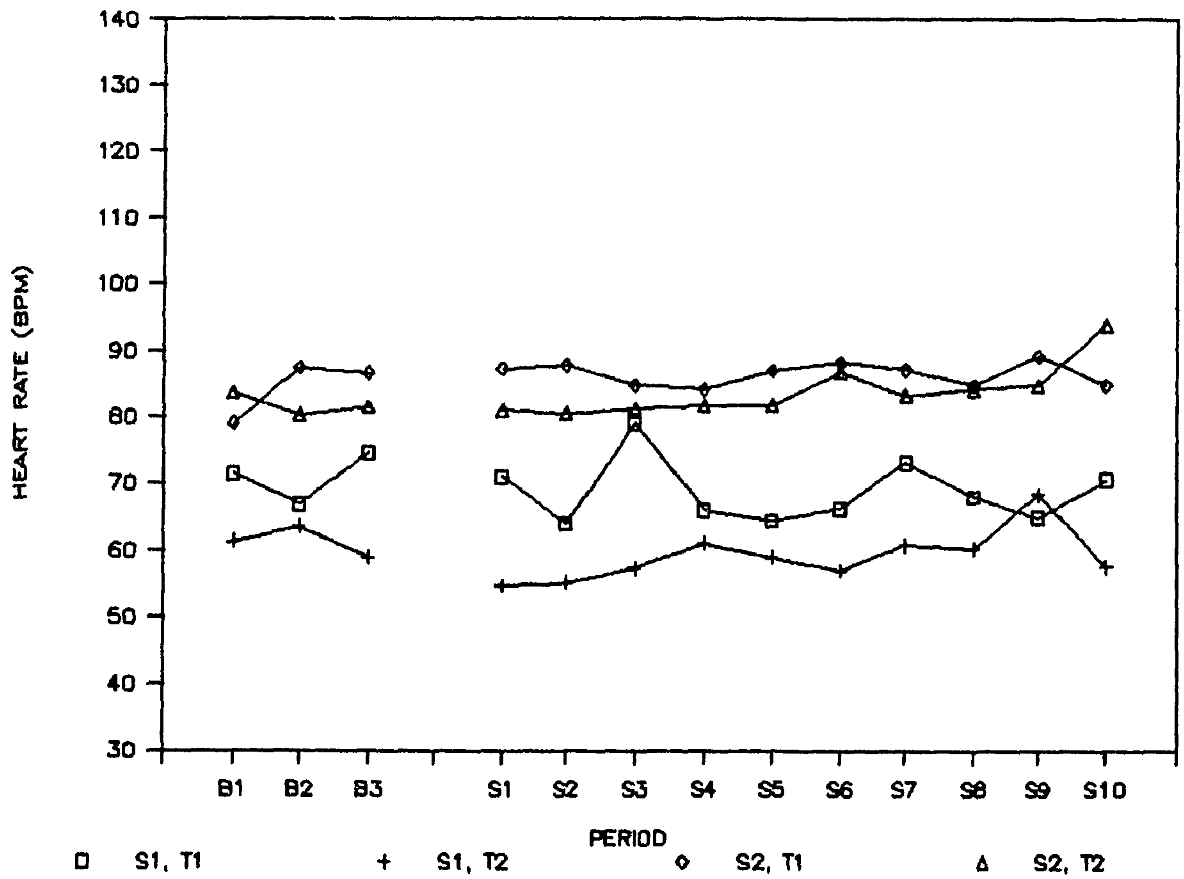
30
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
S1, T1
S1, T2
S2, T1
S2, T2

Subject 8



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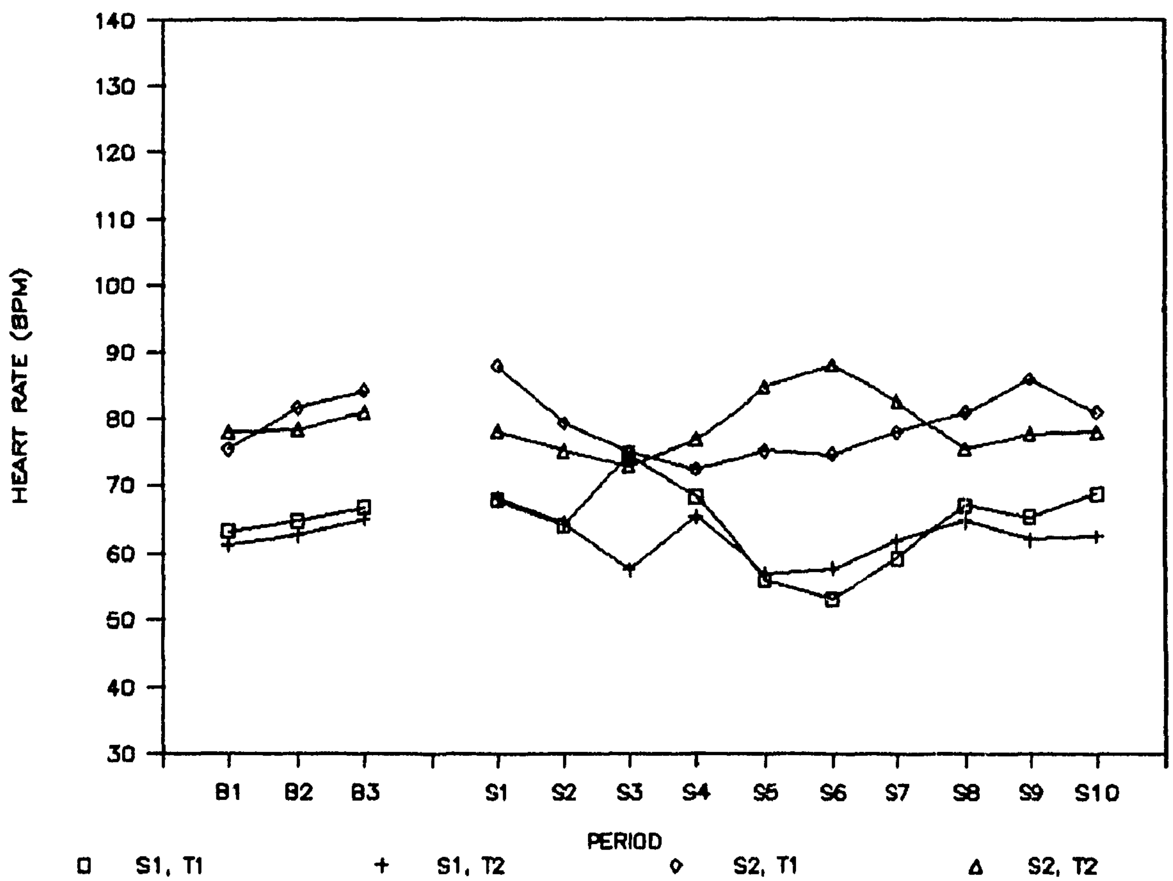
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
S1, T1
S1, T2
S2, T1
S2, T2

Subject 9


HEART RATE (BPM)
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90
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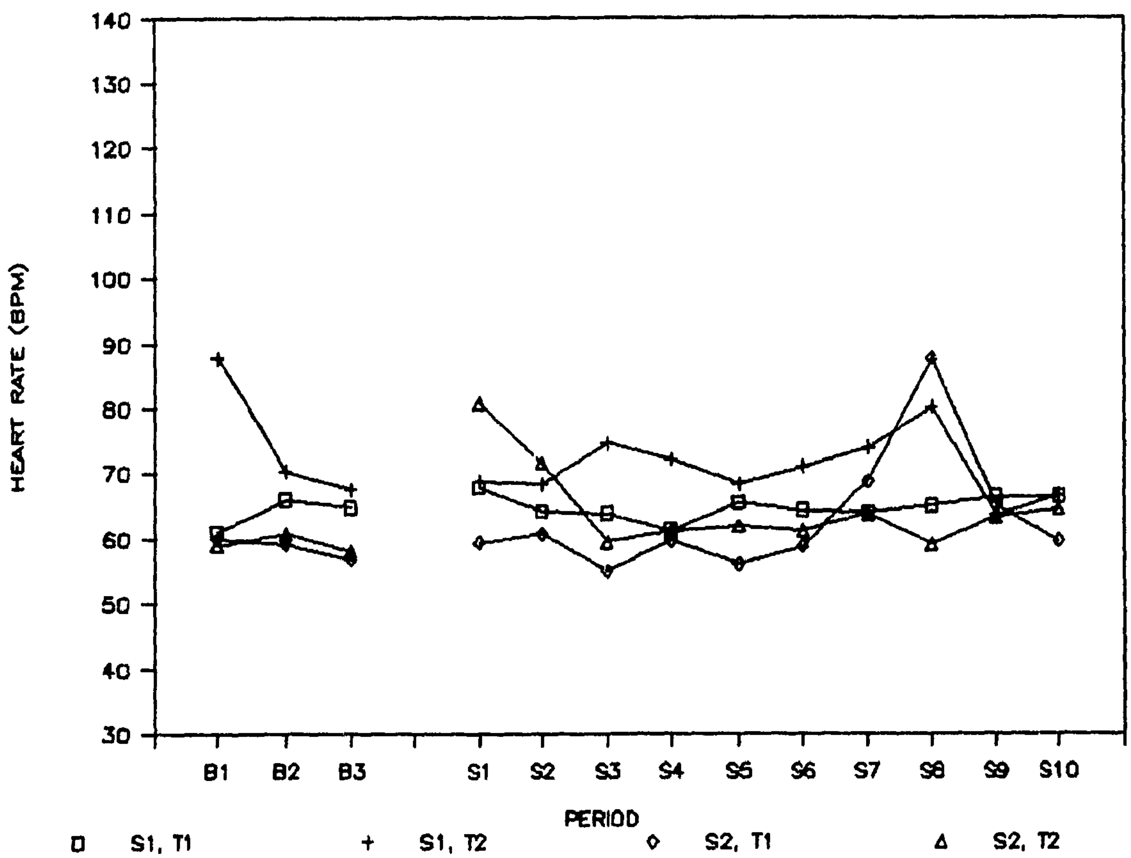
40
30
B1 B2 B3 S1 S2 S3 S4 S5 S6 S7 S8 S9 S10
PERIOD
S1, T1
S1, T2
S2, T1
S2, T2

Subject 10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30
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
S1, T1
S1, T2
S2, T1
S2, T2

Subject 11

Appendix 4

Subjective Rating Scale

We would like to get some indication of how challenging you found the two tasks on a scale of 1 to 10, where 1 is not challenging, and 10 is very challenging. Please circle one of the numbers for each task.

Task 1

1	2	3	4	5	6	7	8	9	10
not challenging									very challenging

Task 2

1	2	3	4	5	6	7	8	9	10
not challenging									very challenging

Appendix 5

You will be presented two tasks that test your ability to answer a series of mathematical questions accurately and quickly. Some questions will require you to verbalize your responses, others will not. When the white light flickers on, please give your verbal response to the preceding question. If the white light does not go on, do not verbalize your response. There will be a several minute waiting period before the first task, and another several minute waiting period between the two tasks.

Sample Questions

1. 9 x 7 - 6

2. 27 / 3 x 4

3. 3 x 8 - 5

4. 22 x 4 - 85

5. 12 x 6 + 9

6. 38 / 2 x 3

7. 72 / 8 x 4

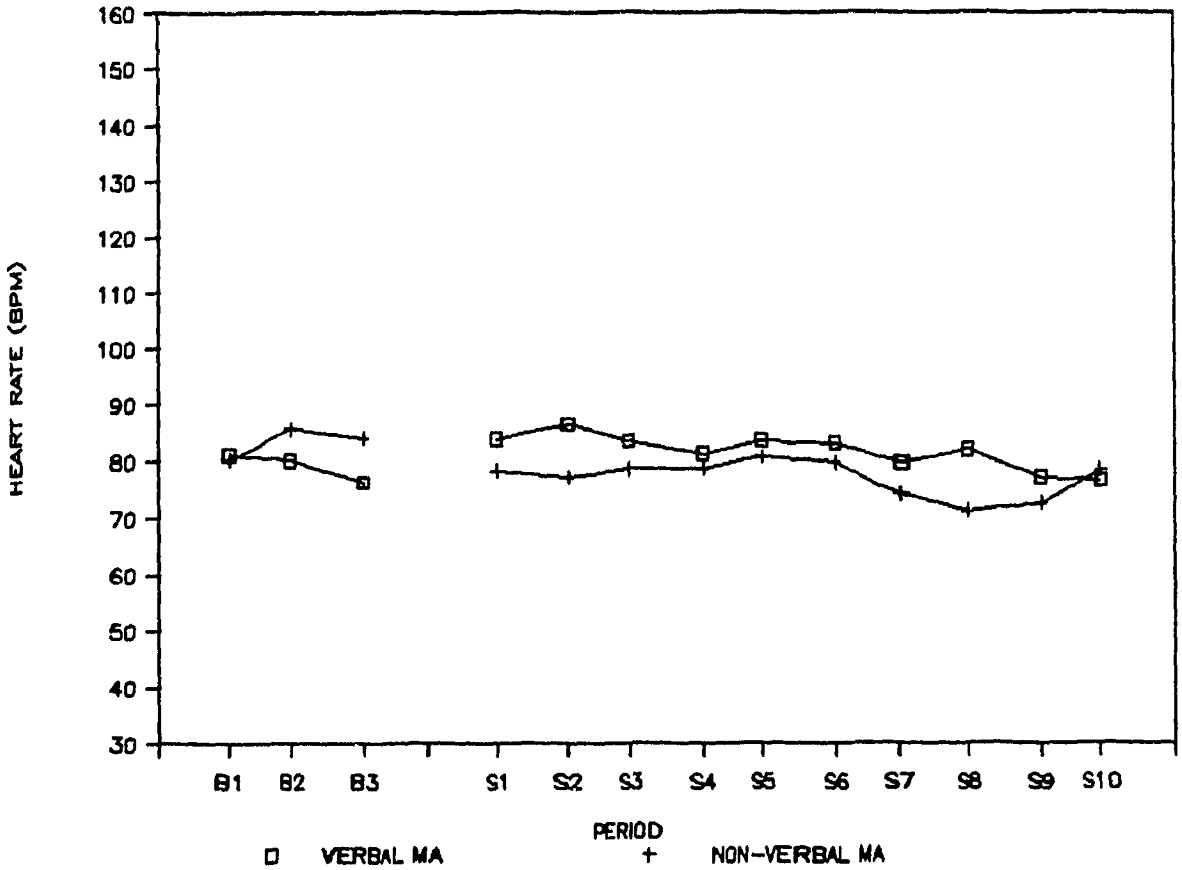
8. 6 x 2 - 3

9. 11 x 4 - 2

10. 5 x 4 + 7

Appendix 6

Case by case heart rate data for all subjects (N = 14) employed in Experiment 2, with interbeat intervals over ten second intervals averaged over 13 periods (i.e., B1 to B3 for baseline heart rate, and S1 to S10 for heart rate with exposure to verbal and non-verbal mental arithmetic).



160
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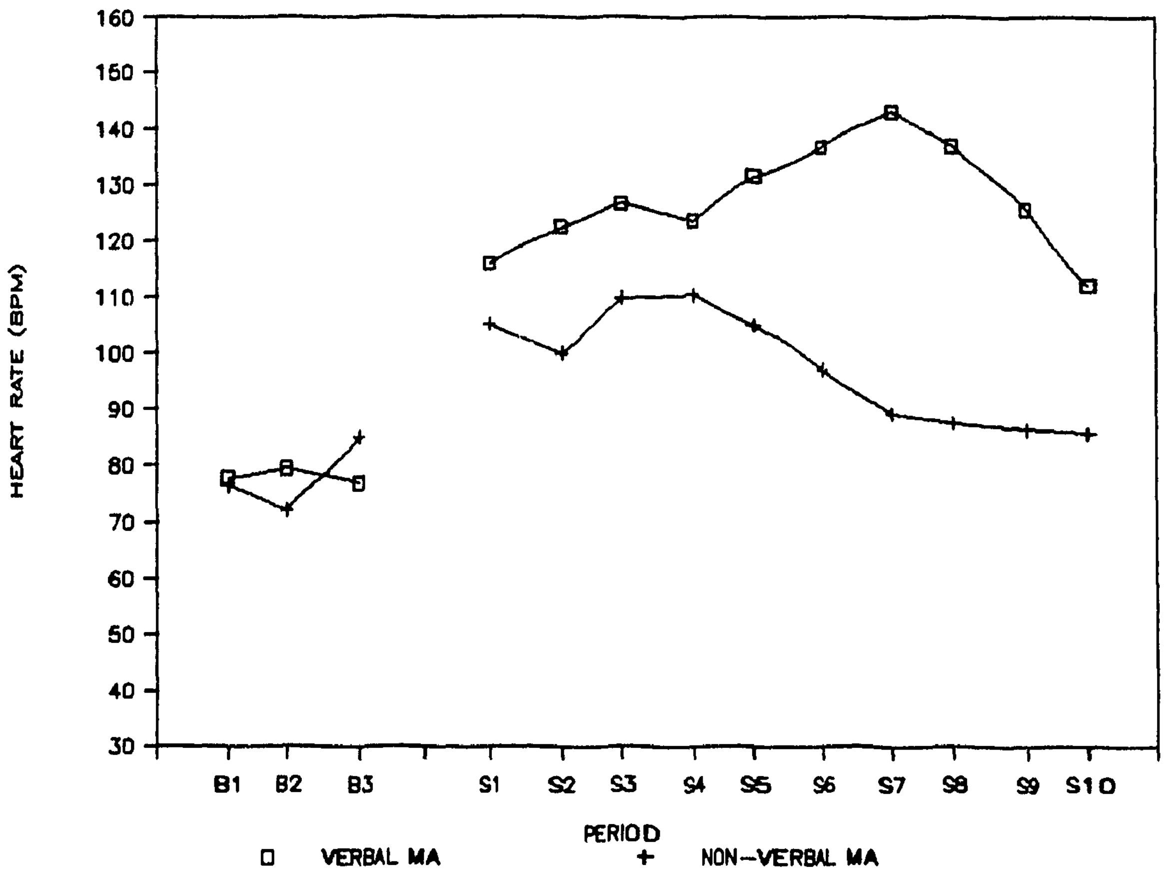
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
VERBAL MA
NON-VERBAL MA

Subject 1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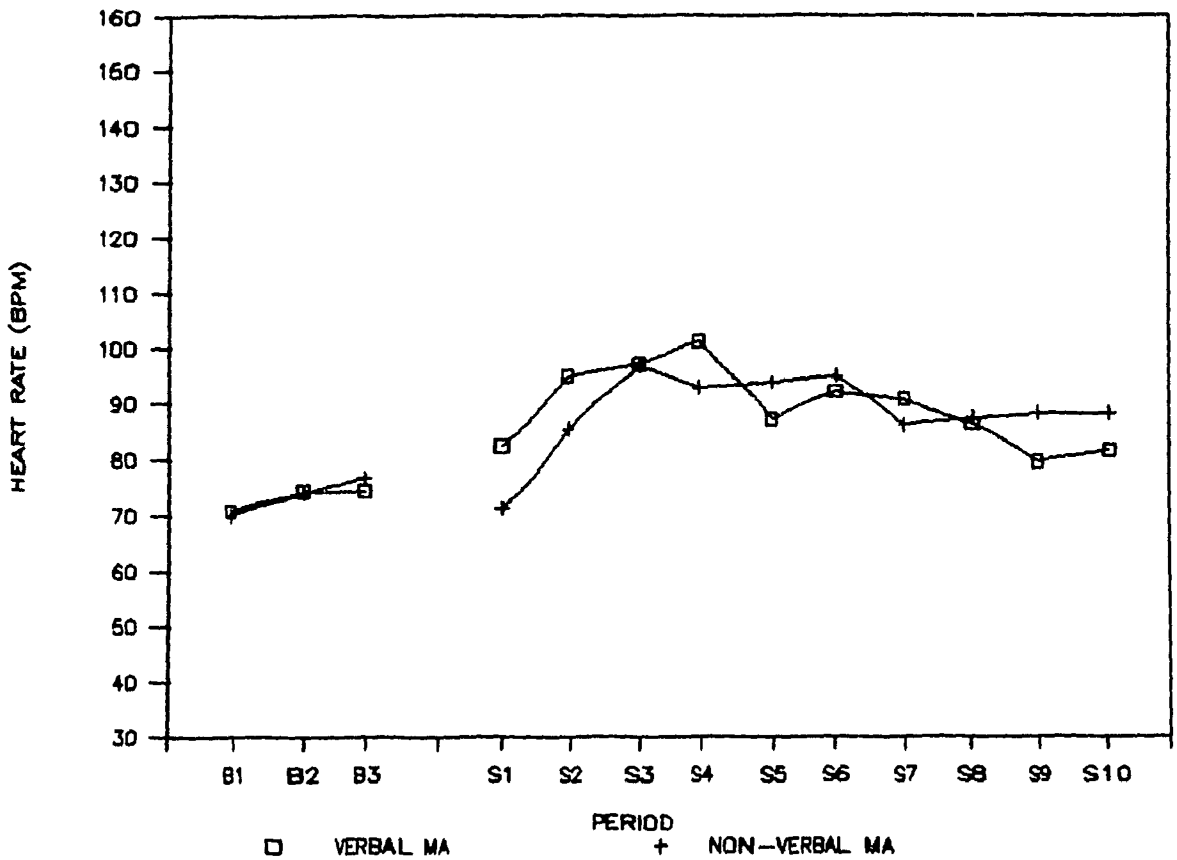
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
VERBAL MA
NON-VERBAL MA

Subject 2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HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
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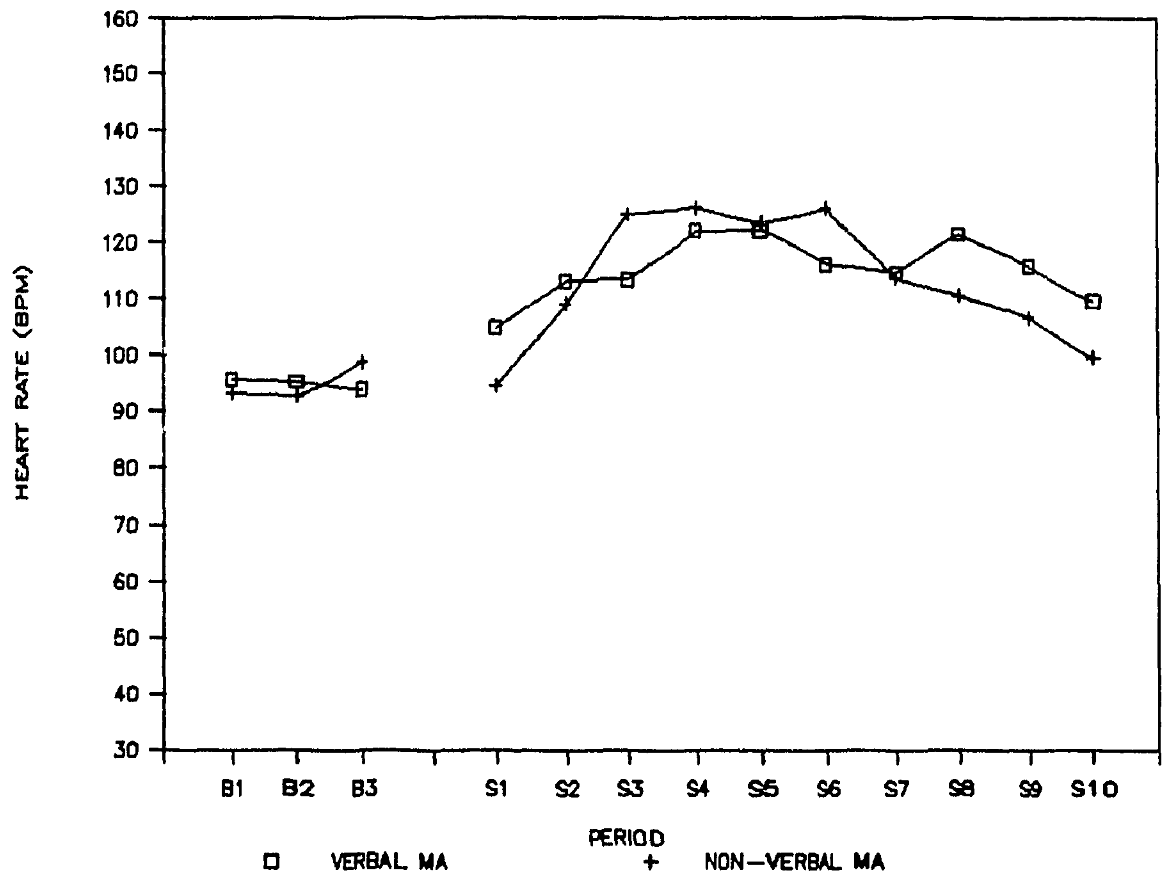
S7
S8
S9
S10
PERIOD
VERBAL MA
NON-VERBAL MA

Subject 3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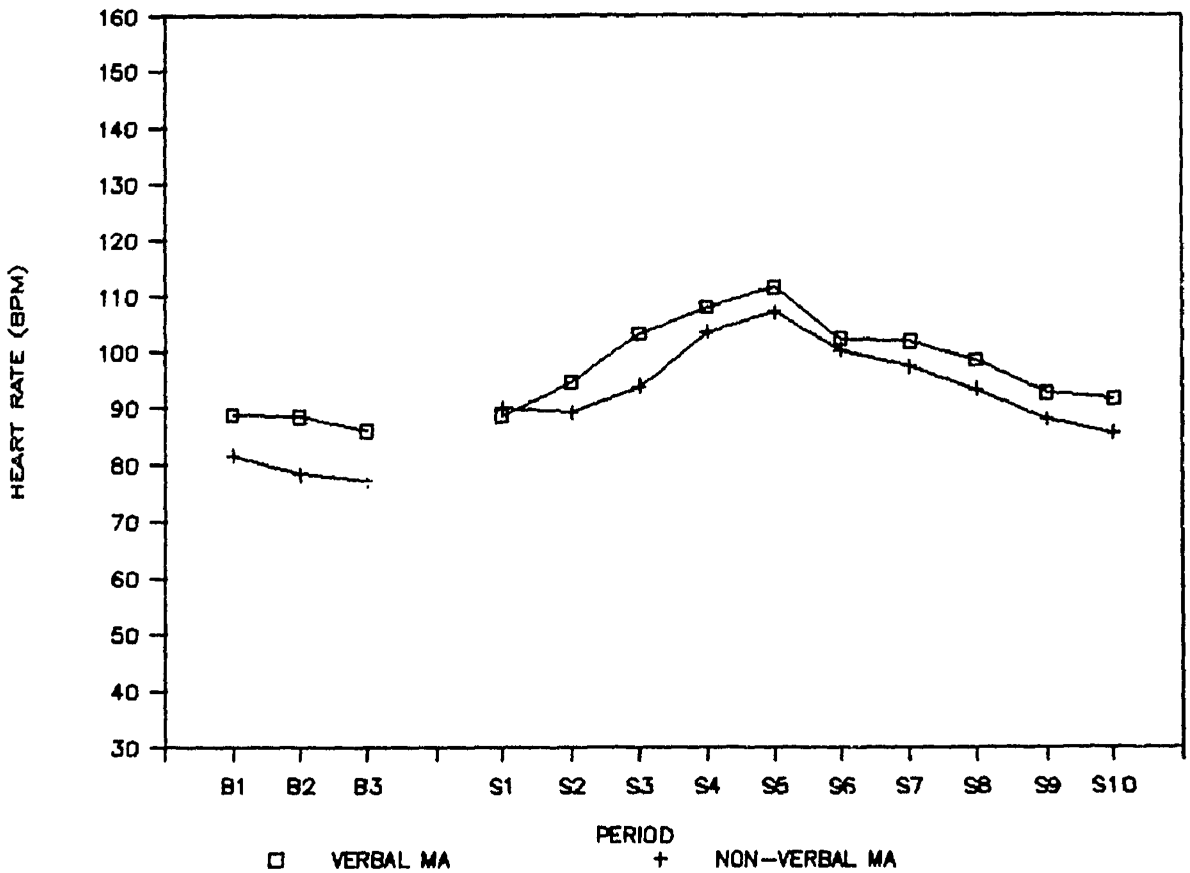
40
30
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
VERBAL MA
NON-VERBAL MA

Subject 4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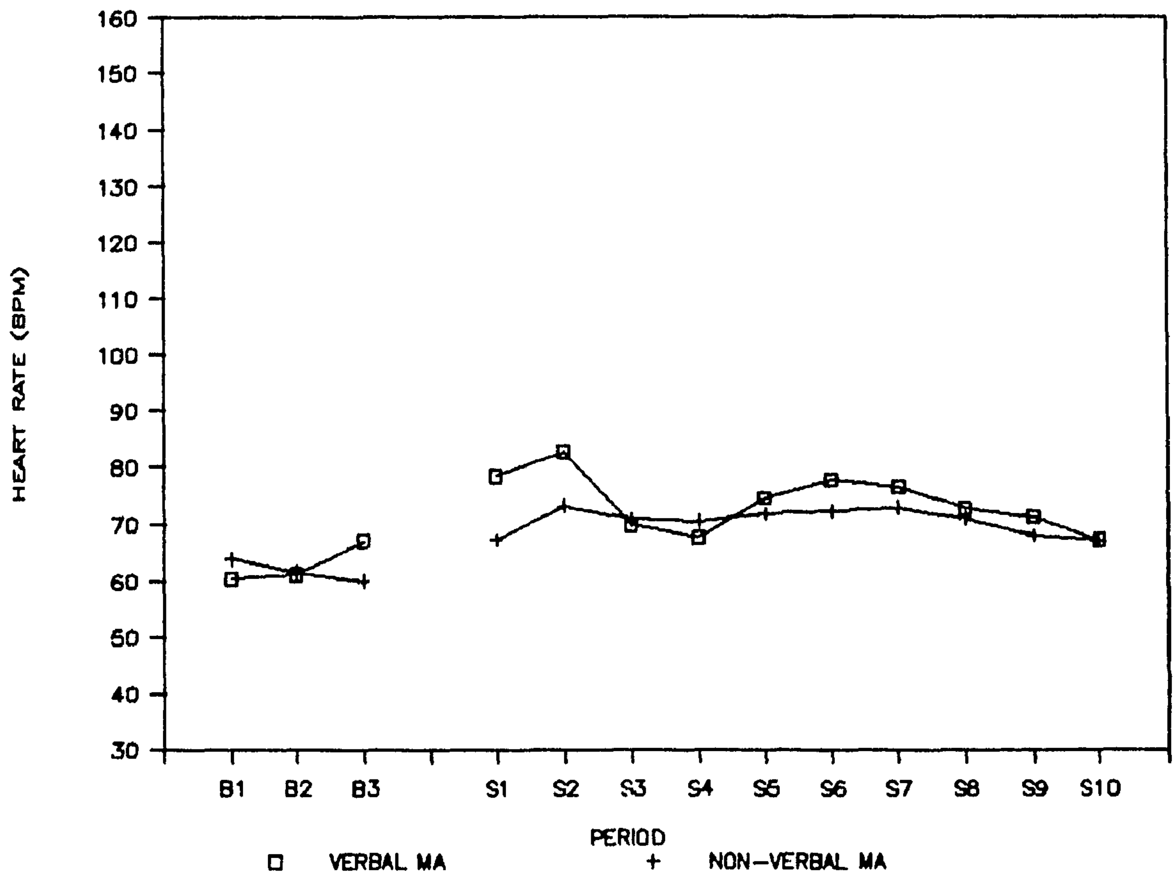
30
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
VERBAL MA
NON-VERBAL MA

Subject 5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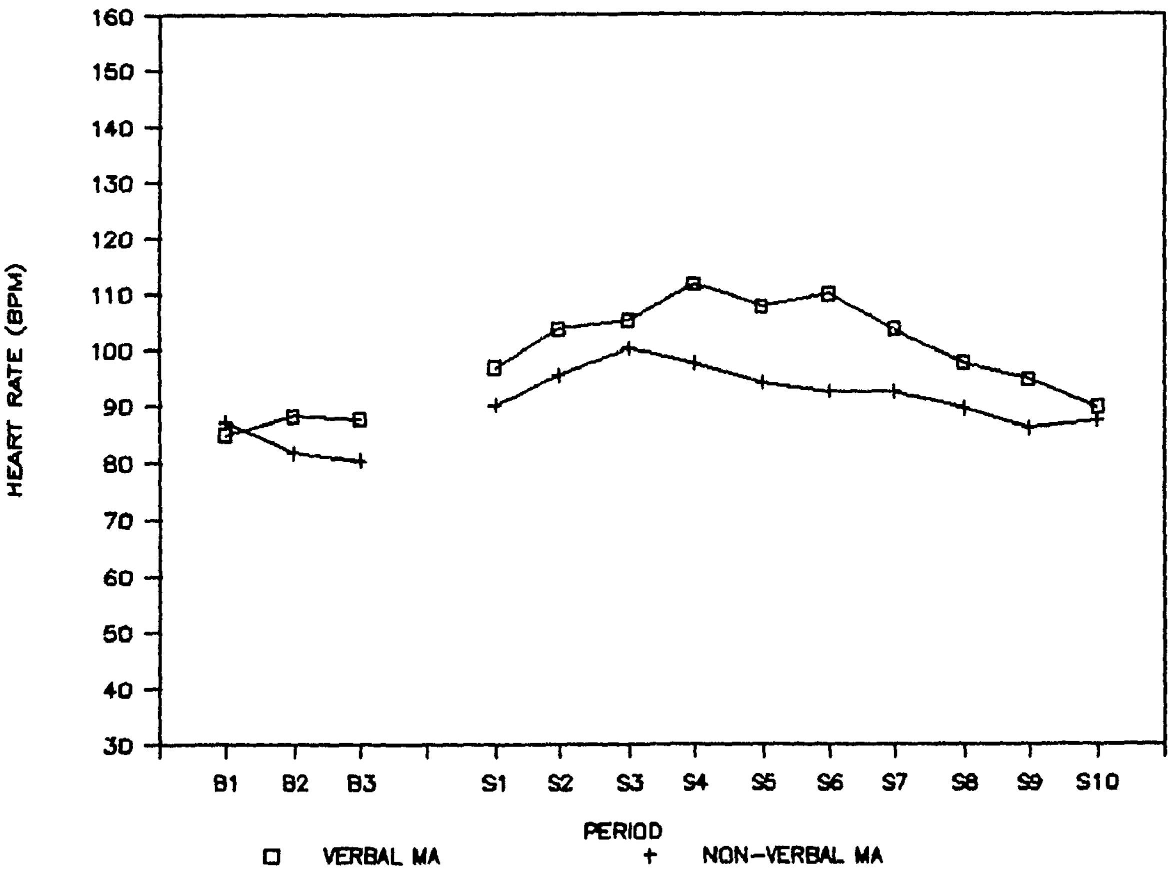
HEART RATE (BPM)
B1 B2 B3 S1 S2 S3 S4 S5 S6 S7 S8 S9 S10
PERIOD
VERBAL MA
NON-VERBAL MA

Subject 6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HEART RATE (BPM)
B1
B2
B3
S1
S2
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S4
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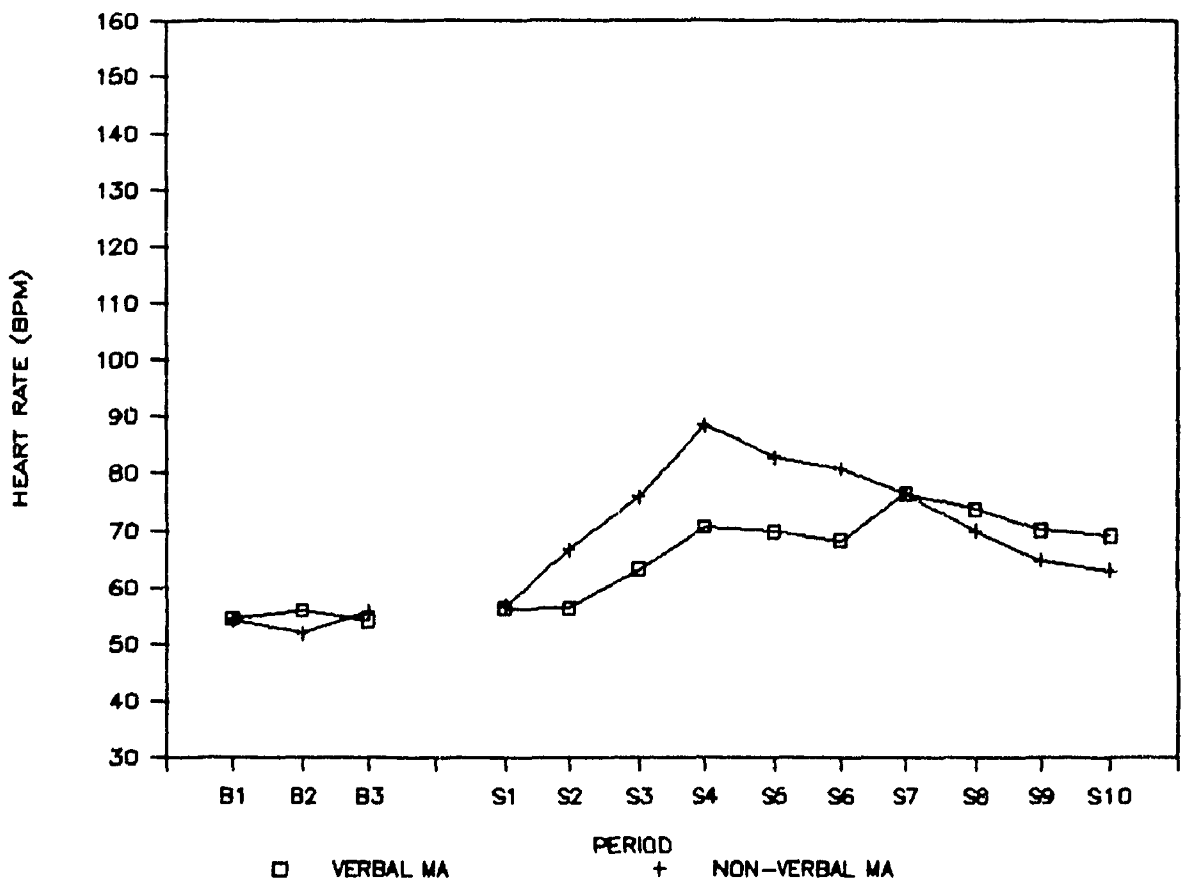
S7
S8
S9
S10
PERIOD
VERBAL MA
NON-VERBAL MA

Subject 7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HEART RATE (BPM)
B1
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B3
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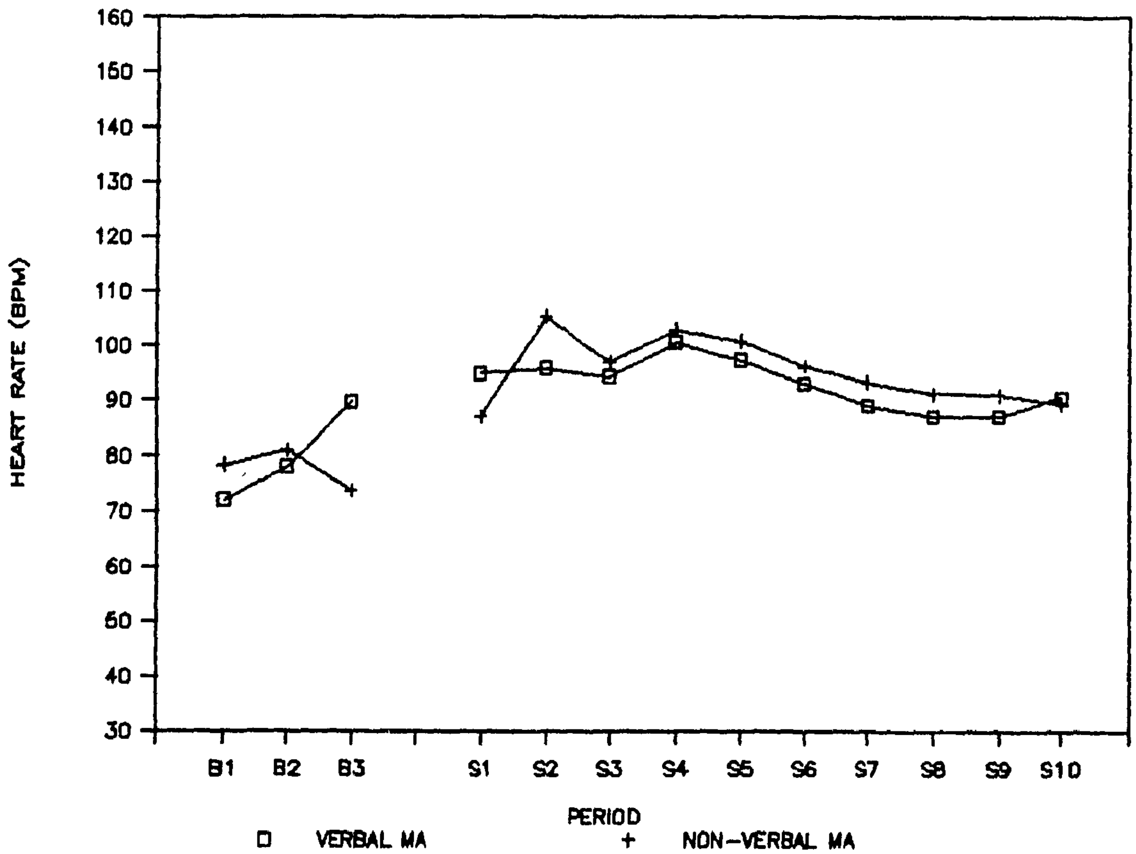
S9
S10
PERIOD
VERBAL MA
NON-VERBAL MA

Subject 8



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HEART RATE (BPM)
B1
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B3
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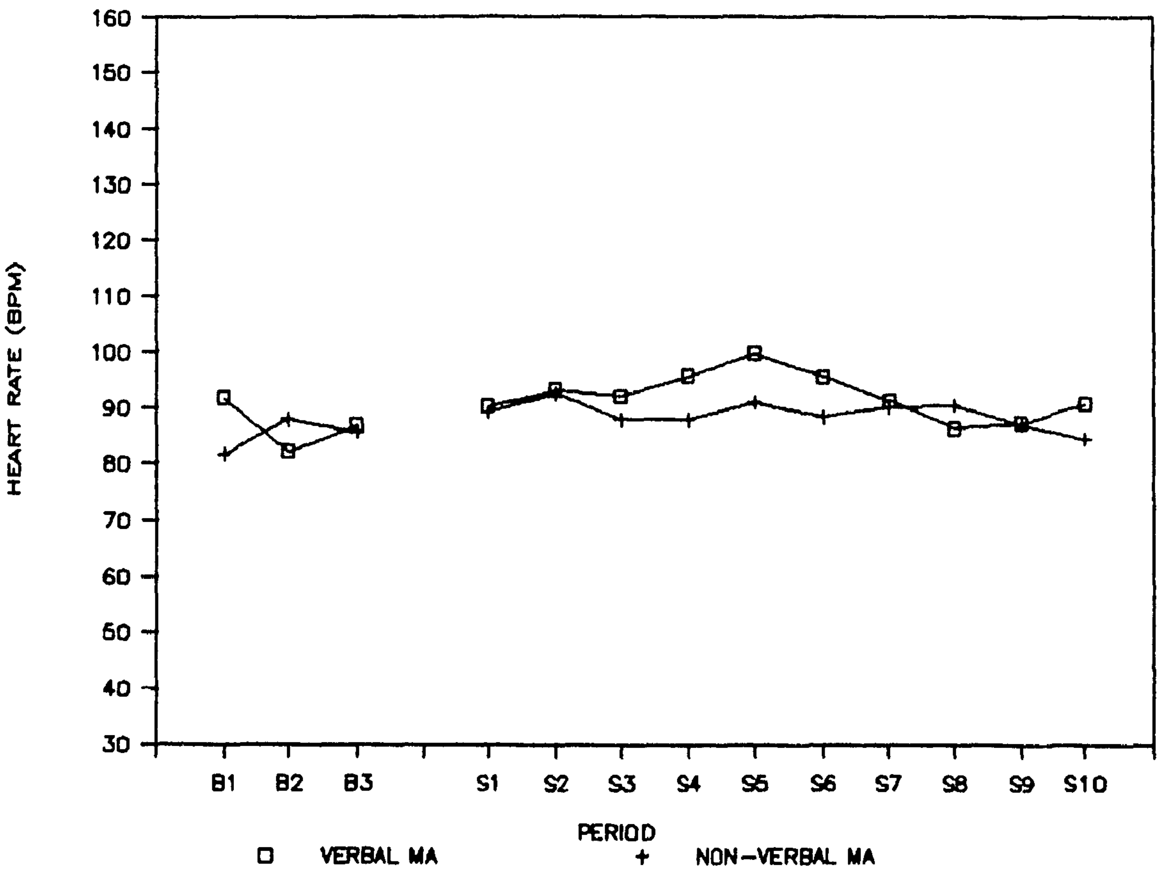
S10
PERIOD
VERBAL MA
NON-VERBAL MA

Subject 9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HEART RATE (BPM)
B1
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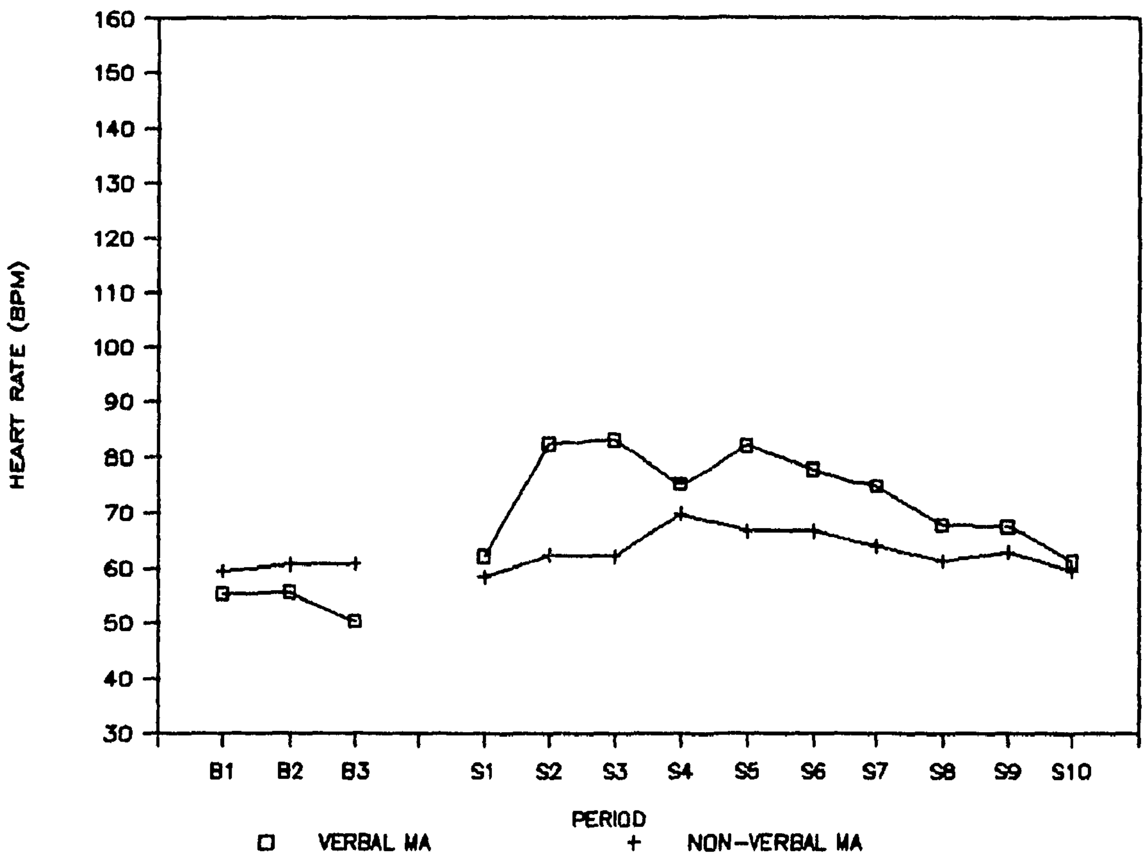
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
VERBAL MA
+ NON-VERBAL MA

Subject 10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HEART RATE (BPM)
B1
B2
B3
S1
S2
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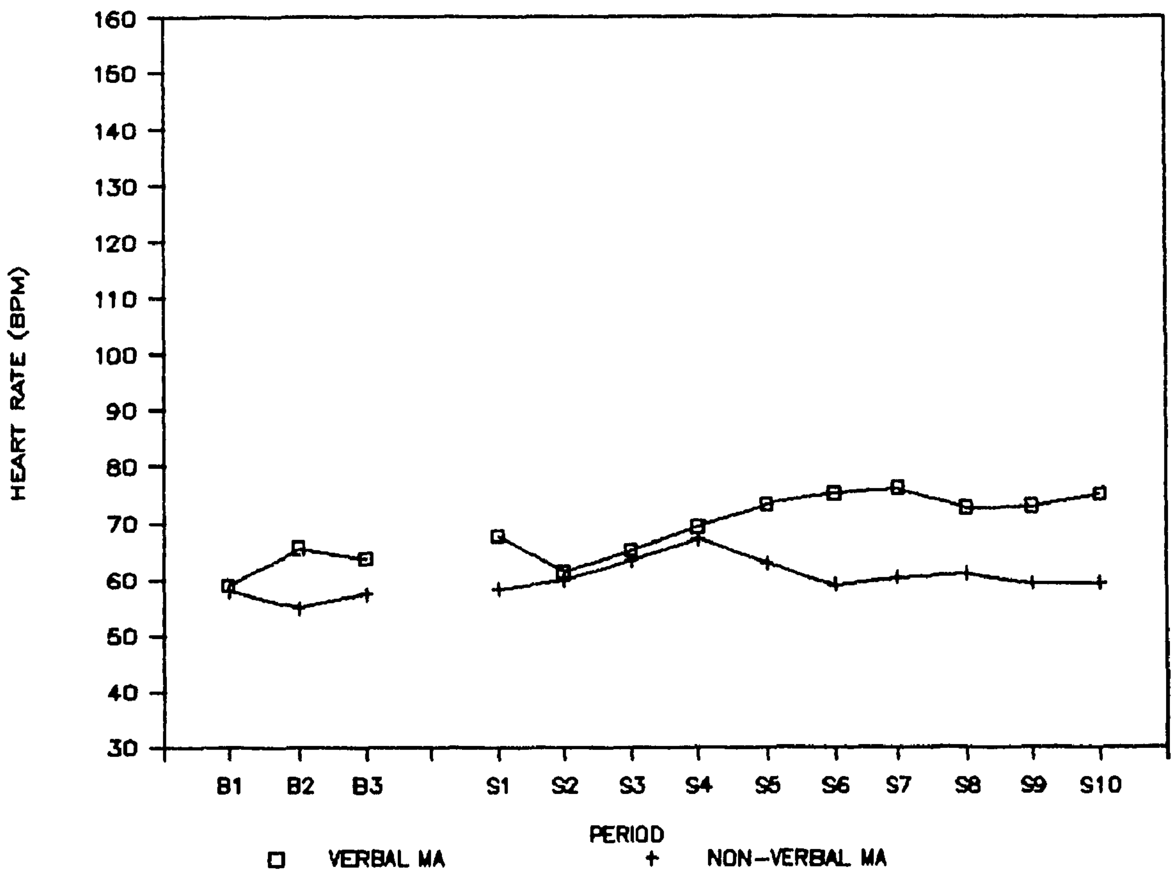
S7
S8
S9
S10
PERIOD
VERBAL MA
NON-VERBAL MA

Subject 11


HEART RATE (BPM)
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B1
B2
B3
S1
S2
S3
S4
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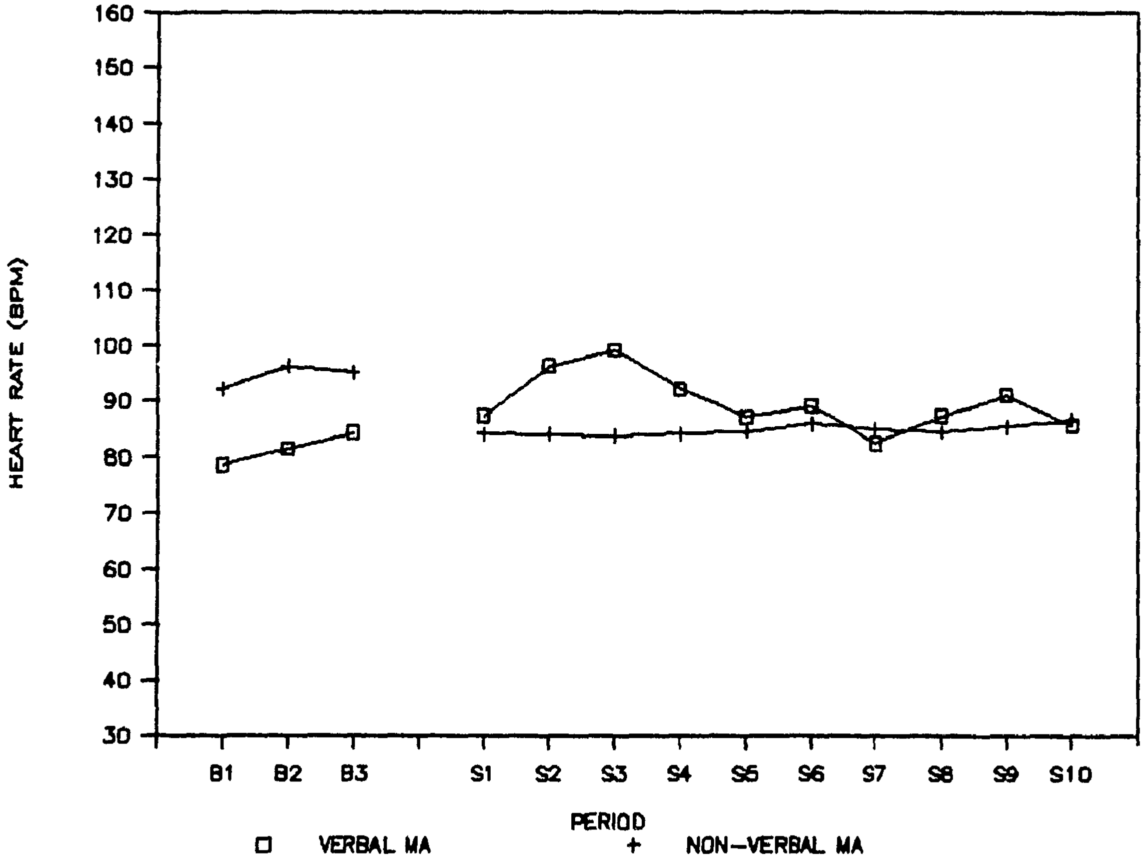
S6
S7
S8
S9
S10
PERIOD
VERBAL MA
NON-VERBAL MA

Subject 12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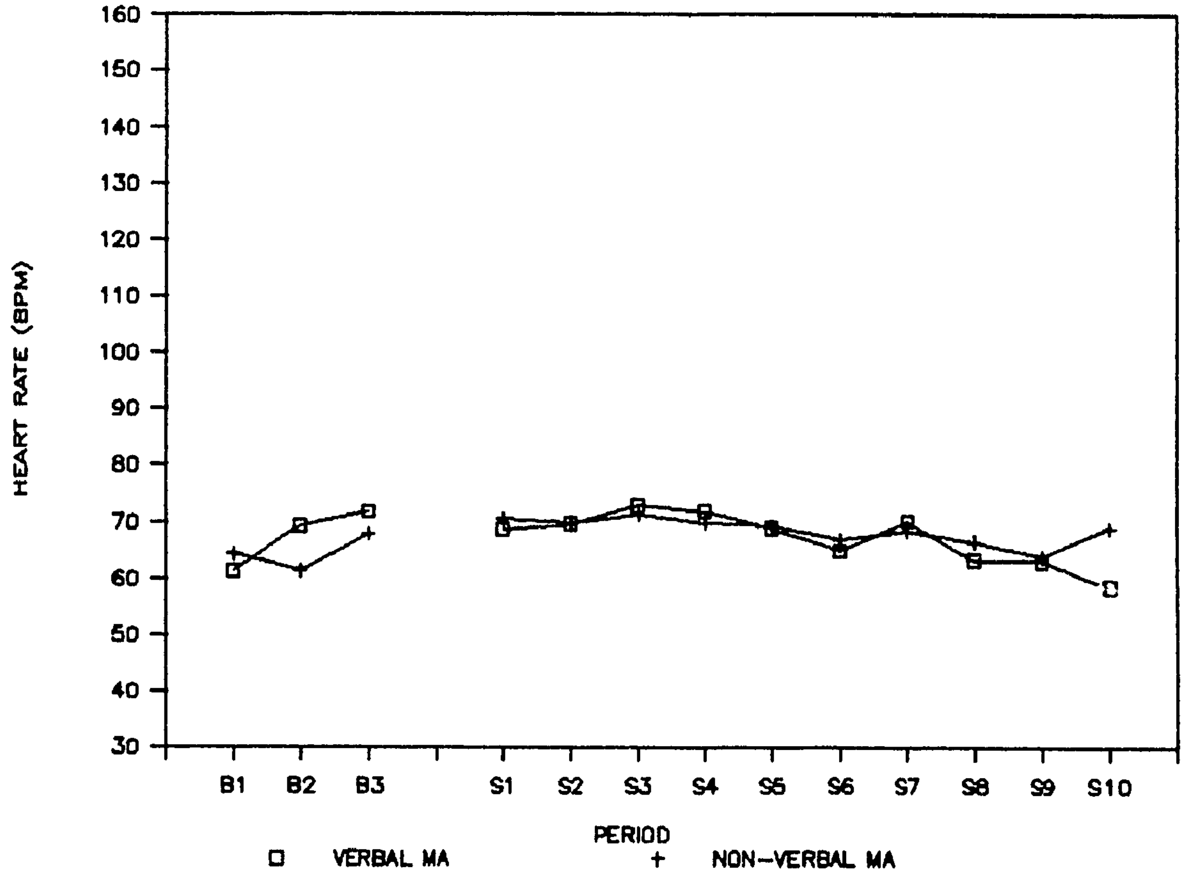
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
VERBAL MA
+ NON-VERBAL MA

Subject 13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HEART RATE (BPM)
B1
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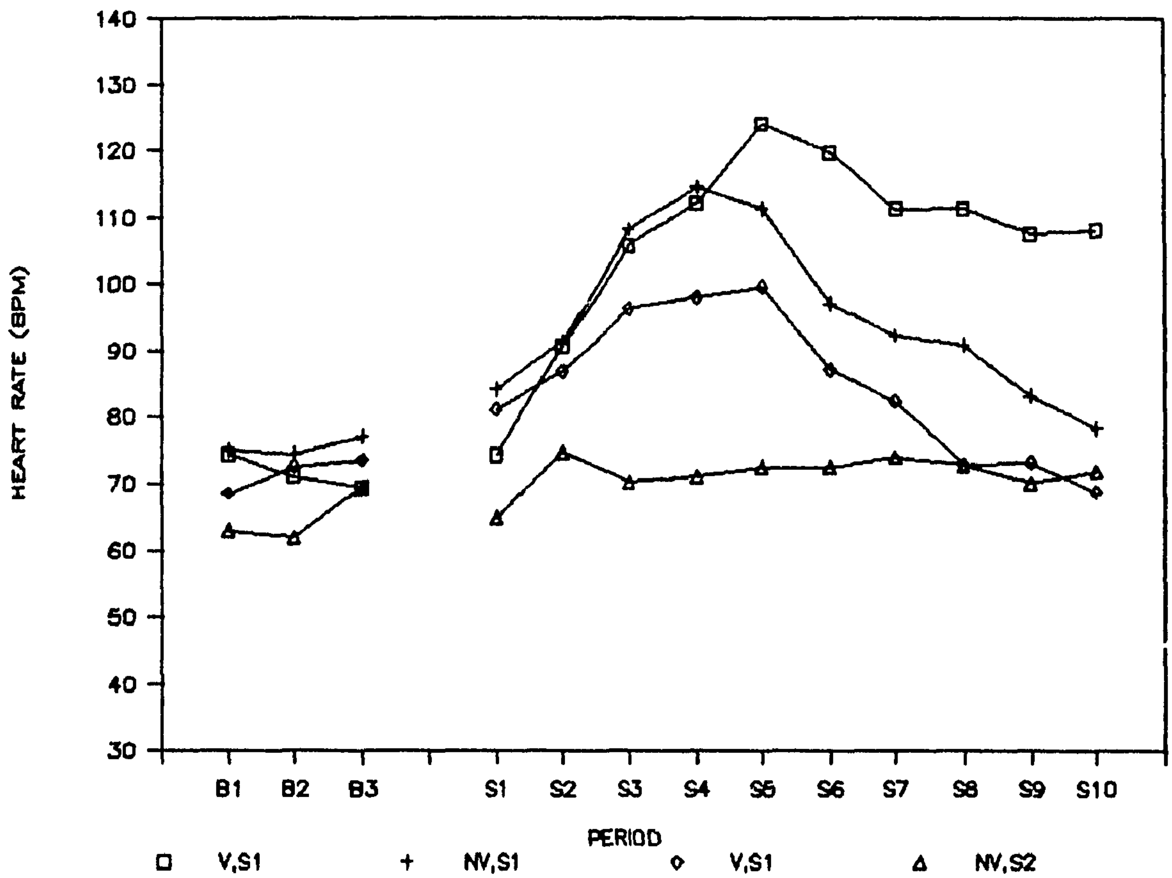
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
VERBAL MA
NON-VERBAL MA

Subject 14

Appendix 7

Case by case heart rate data for all subjects (N = 8) employed in Experiment 3, with interbeat intervals over ten second intervals averaged over 13 periods (i.e., B1 to B3 for baseline heart rate, and S1 to S10 for heart rate with exposure to verbal and non-verbal mental arithmetic). Data for both sessions separated by a one-month interval involving exposure to both verbal and non-verbal mental arithmetic are depicted.



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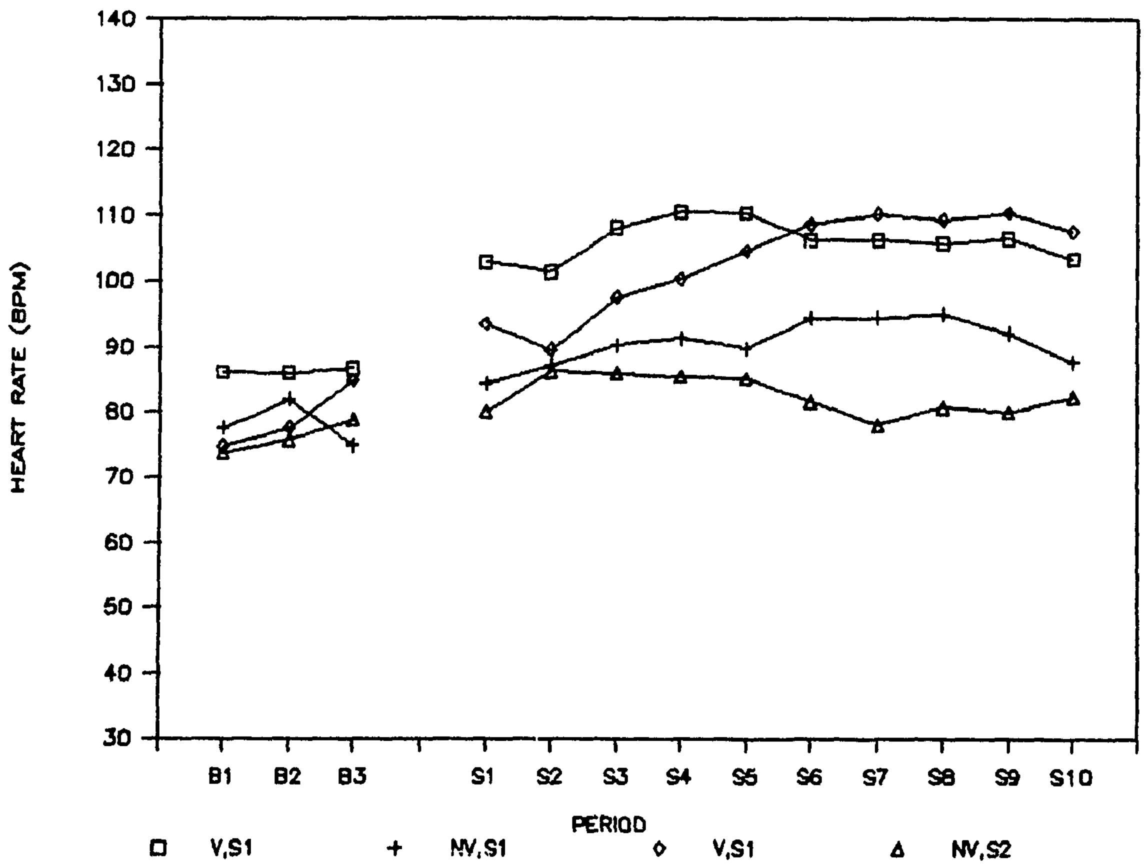
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
V,S1
NV,S1
V,S1
NV,S2

Subject 1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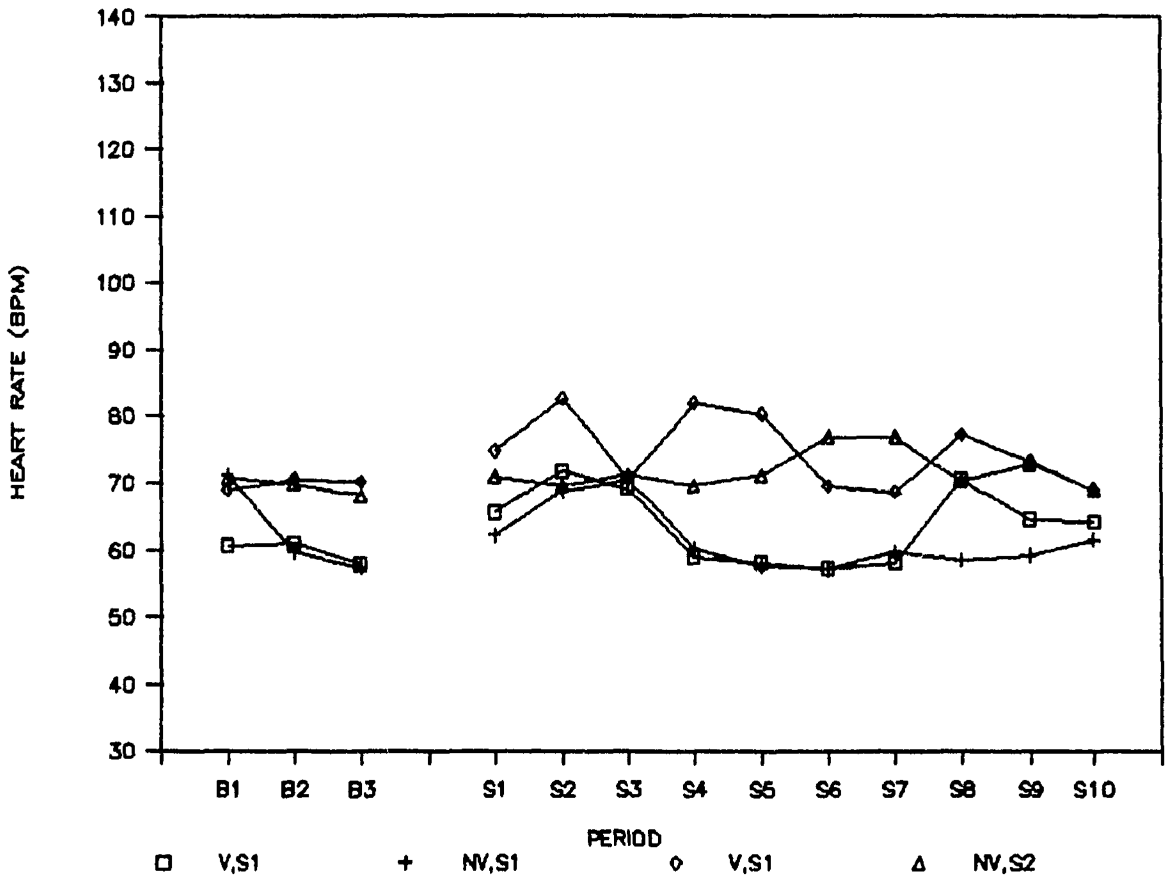
HEART RATE (BPM)
B1 B2 B3 S1 S2 S3 S4 S5 S6 S7 S8 S9 S10
PERIOD
V,S1
NV,S1
V,S1
NV,S2

Subject 2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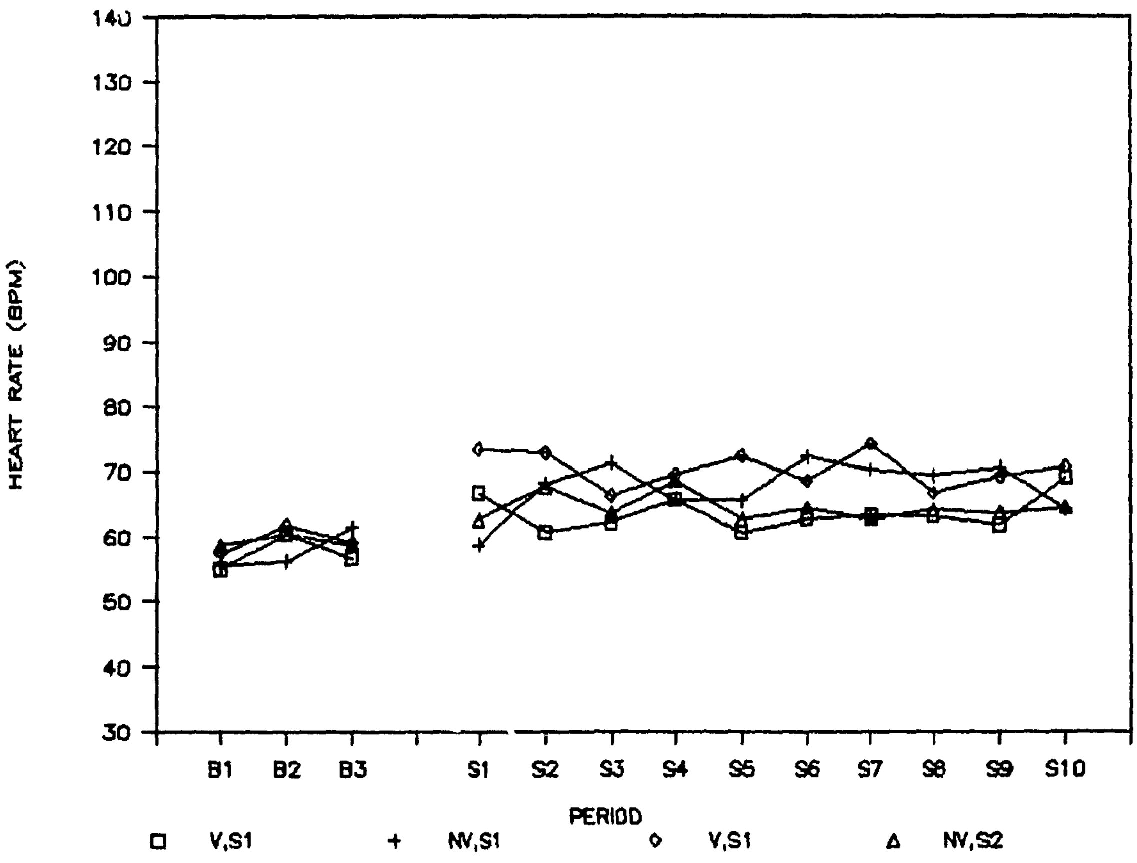
40
30
HEART RATE (BPM)
B1 B2 B3 S1 S2 S3 S4 S5 S6 S7 S8 S9 S10
PERIOD
V,S1
NV,S1
V,S1
NV,S2

Subject 3



140
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70
60
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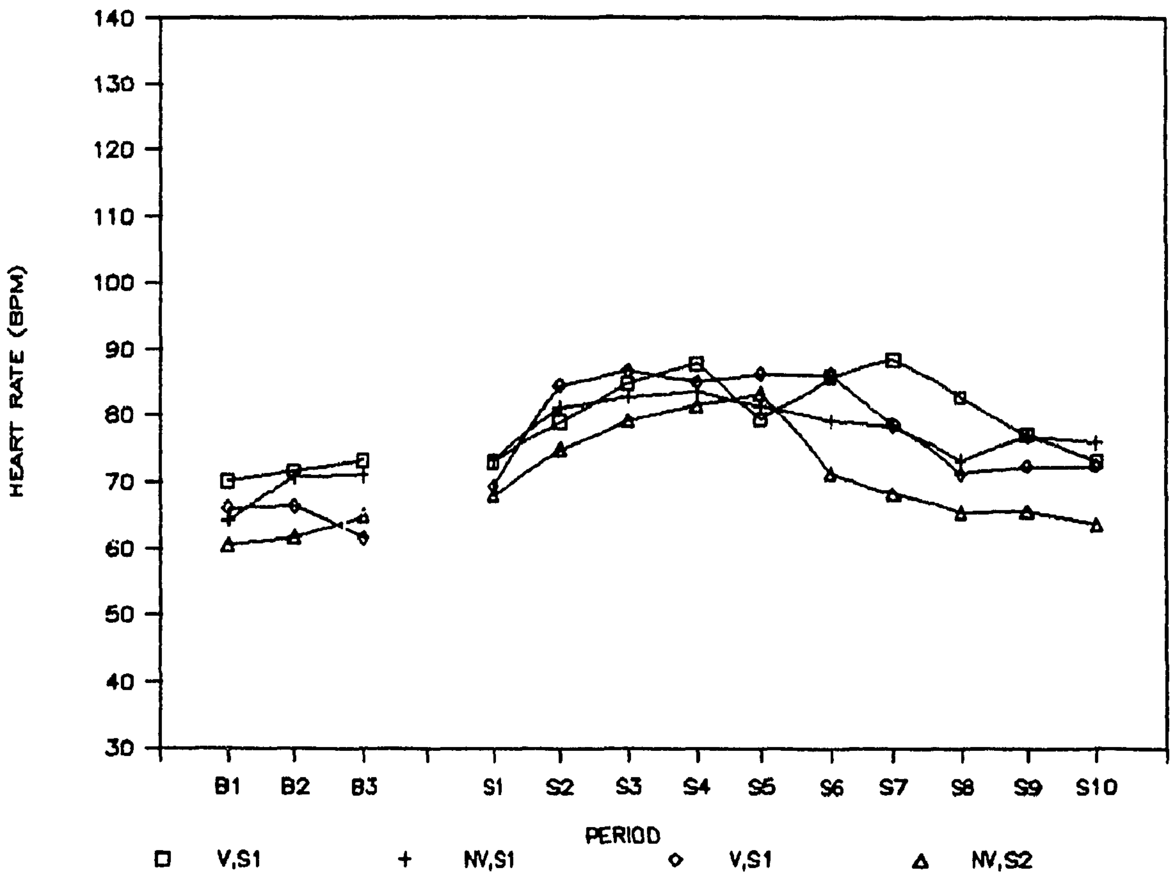
40
30
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
V,S1
NV,S1
V,S1
NV,S2

Subject 4


140
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70
60
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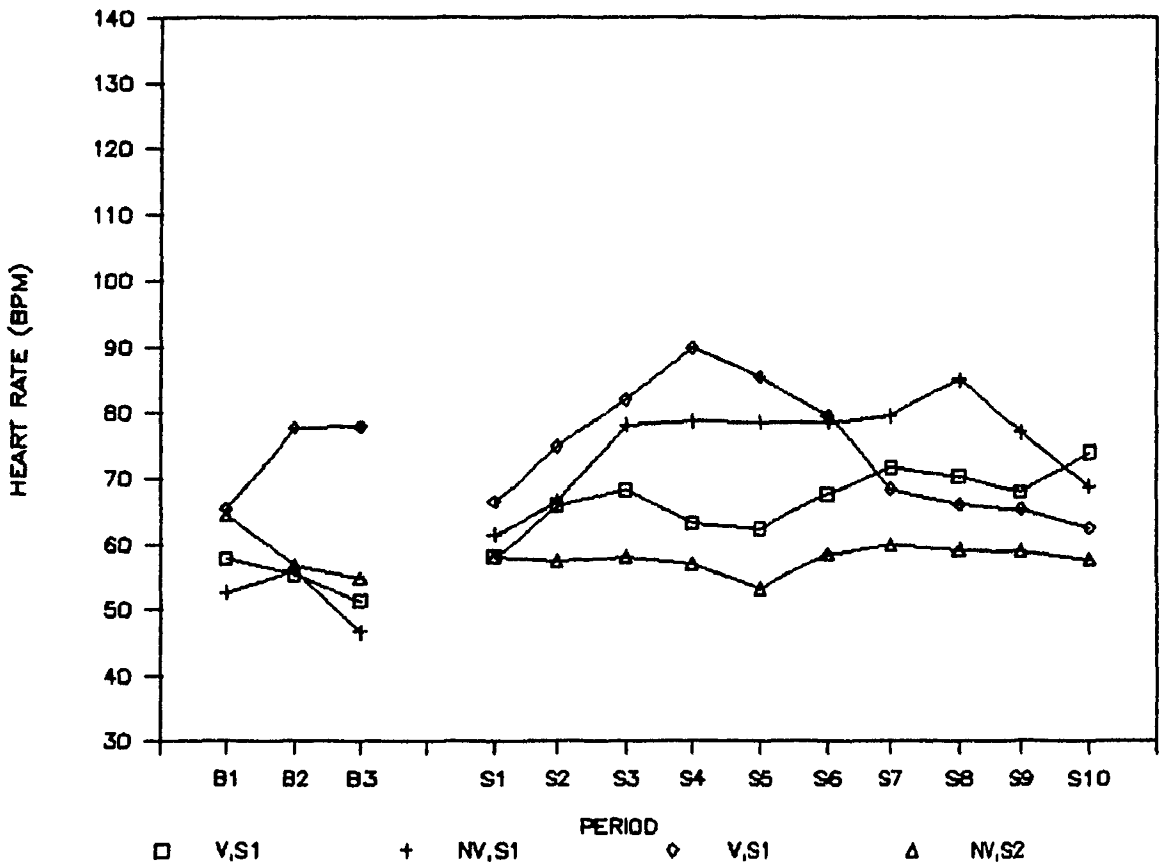
30
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
V,S1
NV,S1
V,S1
NV,S2

Subject 5


140
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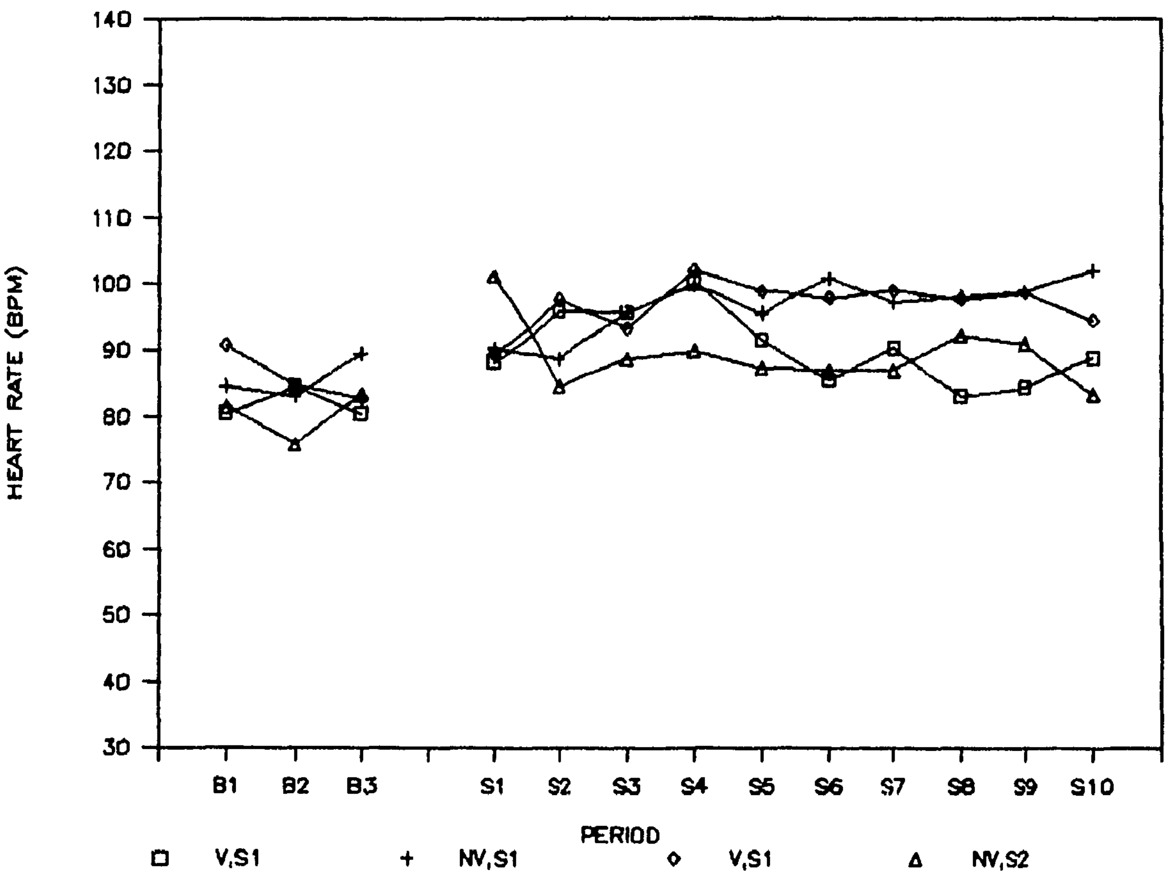
30
HEART RATE (BPM)
B1 B2 B3 S1 S2 S3 S4 S5 S6 S7 S8 S9 S10
PERIOD
V,S1
NV,S1
V,S1
NV,S2

Subject 6



140
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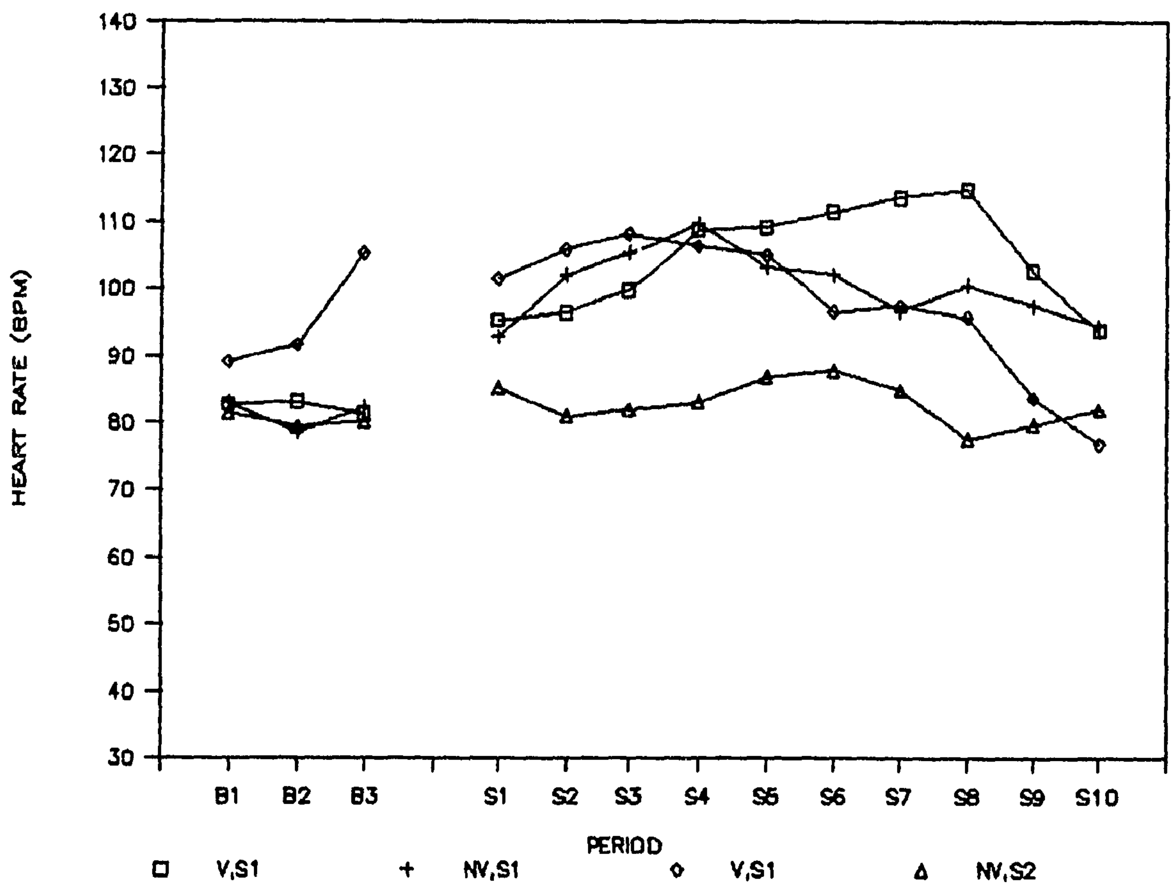
30
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
V,S1
NV,S1
V,S1
NV,S2

Subject 7



140
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30
HEART RATE (BPM)
B1
B2
B3
S1
S2
S3
S4
S5
S6
S7
S8
S9
S10
PERIOD
V,S1
NV,S1
V,S1
NV,S2

Subject 8