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A DEVELOPMENTAL STUDY OF VISUAL ATTENTION AND RESPIRATORY SINUS ARRHYTHMIA IN YOUNG HUMAN INFANTS

By

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ABSTRACT

A DEVELOPMENTAL STUDY OF VISUAL ATTENTION AND
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This study was designed to investigate, in human infants 1- to 6-months of age, the question of whether early developmental and/or individual differences in cardiac responses thought to reflect orienting and sustained attention are due to these types of differences in these psychological processes or are manifestations of these types of differences in the relative parasympathetic-vagal control over the heart. Peripheral noninvasive estimates of parasympathetic-vagal influences on the heart were obtained from spectral and cross spectral analyses of spontaneous (baseline) respiration and heart rate. Behavioral (visual fixation) and physiologic (heart period and respiration) measures of orienting and sustained attention were obtained from discrete presentations of visual stimuli.

Results indicated there were no significant age- or sex-related differences in the relative degree of respiratory sinus arrhythmia as measured by the Porges weighted-coherence function. However, results from two other estimates of vagal functioning--the absolute amount of respiratory sinus arrhythmia and peripheral vagal tone--suggested there were significant increases in vagal influences on the heart from 1- to 3.5-months of age. There also was a significant sex

difference in the age-related increase in the estimate of peripheral vagal tone: Both females and males showed mean increases but only the increase for the males was significant.

Results for the heart period per interval responses replicated previous findings of an age-related shift in the direction of cardiac responses in that from 1- to 3.5-months there was a significant decline in the proportion of infants exhibiting heart rate accelerative responses. No evidence was found to support the hypothesis of increasing magnitude of heart rate decelerative responses over the same age period. Results for the visual fixation, heart period variance, and respiratory amplitude variance measures did not support the hypothesis of an increase in the capacity for sustained attention over the first six postnatal months. Virtually none of the physiologic measures thought to index orienting and sustained attention were correlated with the visual fixation measures. Significant differences between high- and low-C_w groups were not found for the visual fixation nor for the physiologic responses to the stimuli. The only significant correlation between the baseline measures of vagal influences on the heart and the measures of orienting and sustained attention was between mean C_w and mean first fixation times such that greater levels of mean C_w were associated with larger mean first fixation times.

It was suggested that an infant control technique and procedures which would permit the coding of blank stares would allow more adequate tests of these potential physiologic-behavior relations.

To my mother and father.

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CHAPTER I

INTRODUCTION

Over recent years there has been increasing interest in the psychobiological development of the human infant and, in particular, in the psychophysiological study of the processes subsumed under the constructs of orienting and attention as well as in the role of these processes in cognitive development (see Berg & Berg, Note 1; Brackbill & Fitzgerald, 1969; Clifton & Nelson, 1976; Graham & Jackson, 1970; Hirschman & Katkin, 1974; Lipsitt, 1976; Porges, 1974). While noting advances made in adult psychophysiological studies of attention, many infant researchers initially turned to measures of physiological events--most notably those of the autonomic nervous system (ANS)--primarily because of the young infant's limited motoric and verbal capabilities (see Graham & Clifton, 1966; Graham & Jackson, 1970). Of the ANS components measured in these studies, heart rate (HR) changes have emerged as the most widely used physiologic dependent variable.

Although the literature indicates that a majority of infancy studies employing HR have centered upon attention, especially the preparatory orienting reflex, HR parameters have also become increasingly prominent dependent variables in a broad spectrum of psychological and biomedical research. HR dependent variables have been used in studies of infant sensory-perceptual capacities (e.g.,

Bartoshuk, 1964; Glanville, Best, & Levenson, 1977), individual differences (e.g., Bridger & Reiser, 1959; Lipton, Steinschneider, & Richmond, 1966), biobehavioral state (e.g., Hutt, Lenard, & Prechtl, 1969), classical conditioning (see Fitzgerald & Brackbill, 1976), habituation (see Clifton & Nelson, 1976), and social-emotional behaviors (Campos, 1976; Sroufe & Waters, 1977). Recent attentional research with infants considered at risk for dysfunctions in cognitive development (e.g., prematures, see Berkson, Wasserman, & Behrman, 1974; Rose, Schmidt, & Bridger, 1976; Schulman, 1970) as well as the assessment of the status of the central nervous system (CNS) and well-being of the human fetus (e.g., see Nelson, 1976; Paul & Hon, 1974; Porges, Note 2) have relied heavily upon cardiac response measures. Moreover, recent process-oriented HR studies have focused on attentional dysfunctions (disorders) associated with mental retardation (Krupski, 1975, 1976; Porges & Humphrey, 1977), minimal brain dysfunction syndromes such as hyperactivity (Porges & Smith, Note 3; Porges, Walter, Korb, & Sprague, 1975; Sroufe, Sonies, West, & Wright, 1973), and other cases involving brain damage (e.g., Holloway & Parsons, 1971, 1972).

Underlying much of the above cited research is the assumption that peripheral autonomic HR changes can be meaningfully related to psychological processes and their development. In infant research it generally is assumed that the direction of HR change to simple, nonsignal stimuli differentiates orienting reflexes (ORs) from defensive reflexes (DRs): HR decelerative responses are thought to reflect orienting whereas HR accelerative responses reflect DRs

(Graham & Clifton, 1966; Graham & Jackson, 1970). Although there is evidence for this contention, empirical inconsistencies also exist in the literature.

Briefly, one problem has been an "apparent" developmental change in the primary direction of HR responses within these paradigms: From HR acceleration found in newborns to HR deceleration emerging sometime around 6-weeks to 2-months of age (Graham & Jackson, 1970). Although several more recent studies have observed newborn HR decelerative responses (e.g., Adkinson & Berg, 1976; Porges, Arnold, & Forbes, 1973; Sameroff, Cashmore, & Dykes, 1973), it has been argued that this apparently age-related change in the direction of these HR responses stems from the development of central mechanisms of orienting and is not due to maturational changes in the neural control mechanisms of the heart (Berg & Berg, Note 1; Clifton & Nelson, 1976; Graham & Jackson, 1970). Unfortunately, to date there is no study that has tested the latter alternative hypothesis that the direction and, perhaps magnitude, of infant HR responses may be dependent upon maturational and/or individual differences in the neural control of this response system rather than the development of orienting.

The importance of resolving this problem is underscored by the widespread use of cardiac dependent variables as well as the contention that these measures may provide crucial information about infant attentional and other cognitive capacities not easily obtained with other dependent measures (e.g., see Clifton, 1974a). Moreover this issue would seem to have important theoretical implications for the recent increase in research on the problem of early assessment and

intervention strategies for infants at risk for deficient cognitive development (cf. Ross & Leavitt, 1976). Therefore, one aim of the present study was to establish whether there are developmental changes in the neural control of the heart. Specifically, the impetus for the present study derives from hypotheses recently offered by Porges (1976a; Reese & Porges, 1976). Briefly, he proposed that during the course of early infancy there is a developmental increase in the relative parasympathetic (vagal-inhibitory) control of the heart and this trend could possibly account for the age-related trend in the direction of infant HR-responses rather than the development of orienting *per se*. He also suggested that this ontogenetic change in the parasympathetic influence on the heart could be measured, noninvasively, by the relative degree of respiratory sinus arrhythmia (RSA). Despite evidence of an indirect sort which tends to support these hypotheses there has been no study to date that has submitted them to an empirical test.[1] In the present study, therefore, spontaneous levels (baseline) of cardiac and respiratory activities of infants 1-, 3.5-, and 6-months of age were recorded and quantified to obtain estimates of respiratory influences on HR (RSA) as a test

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[1] After completion of the data collection, analyses, and first draft of the present study, I became aware of a longitudinal study of infants which used techniques to quantify RSA very similar to those employed in the present study (see Harper, Walter, Leake, Hoffman, Sieck, Sterman, Hoppenbrouwers, & Hodgman, 1978). The agreement between the findings of the Harper et al study and the present study is quite striking. However, since the present study was designed and run without any knowledge of the research by Harper et al, discussion of their findings was only included in the final chapter of this dissertation.

of the hypothesized developmental increase in the relative parasympathetic control of the heart.

A further aim of this study was to examine something of the potential behavioral significance of the hypothesized developmental change in the neural control of the HR-cardiovascular response system. Therefore, the present study also employed a procedure designed to optimize orienting to stimulus change (stimulus onset and offset) as well as states of sustained attention to visual stimuli. Thus, this study was designed to permit the analysis of both behavioral (visual fixation) and physiologic (cardiac and respiratory) response parameters which have been used as indices of orienting and sustained attention in infant research, as well as whether or not such response measures are related to measures of the neural control of the heart. Since some empirical support has been reported for the claim that the suppression of HR variability and respiration amplitude variability may be important biological concomitants of states of sustained attention in children and adults (e.g., see Porges, 1974, 1976a), and since these hypothesized relations between sustained attention and suppression of HR variability, but not suppression of respiration amplitude variability, have been tested with newborns but not older infants, these response measures also were included in the present study.

CHAPTER II

LITERATURE REVIEW

Contemporary interest in the process of attention is unprecedented (e.g., see Bakan, 1966; Broadbent, 1971; Furby, 1974; Graham & Jackson, 1970; Kahneman, 1973; Kornblum, 1973; Lynn, 1966; Mostofsky, 1970a; Posner, 1975; Pribram & McGuinness, 1975; Sokolov & Vinogradova, 1975). One often cited reason for this renewed interest is that attention, broadly speaking, is regarded by many as a prerequisite for successful performance on a wide variety of perceptual and cognitive tasks (i.e., a critical source of variance in performance). Despite the recent and quite substantial effort devoted to this problem there is little consensus about how attention should be defined (see Meldman, 1970; Mostofsky, 1970b) and it remains an often elusive concept.

The diversity of meanings attributed to "attention" can be traced, in part, to the wide variety of response measures used to infer attentional processes. As Porges and Humphrey (1977) pointed out, "responses associated with attention have ranged from reactions to massive changes in stimulation (such as the startle response) to active instrumental behaviors associated with sustained or focused attention." Posner (1975) adds that "...attention is not a single concept but the name of a complex field of study." Nevertheless, among investigators in this broad research domain there is a consensus that there is need for further work on the nature of the responses

used to infer attentional phenomena before any definitive resolution of the problem will emerge.

Since the present study is concerned with the problem of response definition in HR-psychophysiological measures of attention in human infants, the following literature review examines several psychophysiological approaches used to study attentional processes in the human infant, especially those that involve cardiac orienting and sustained attention. The review was limited primarily to HR-psychophysiological approaches not because other theories or approaches were considered unimportant, but rather, because this research domain is the most relevant to the concerns of the present study.

The review opens, therefore, with an overview of the most prominent psychophysiological theories. Then empirical evidence concerning the general hypothesis that cardiac response parameters can be linked to attention and orienting will be examined. It should become clear that within this overall psychophysiological orientation, there are several lines of research emphasizing different views as to how cardiac responses may be meaningfully related to orienting and attention. Next, the problem of individual differences in attention research as it relates to behavior-physiologic relations will be discussed in light of evidence which bears on hypotheses concerning the underlying physiologic substrates of attentional phenomena. This literature will then be related to the HR research on orienting and attention in infancy with particular emphasis devoted to the issue of an apparent developmental shift in the direction of infant HR responses to simple, nonsignal stimuli.

Finally, evidence will be reviewed on the previously cited hypothesis that maturational and/or individual differences in CNS mediation of ANS control of the cardiac system should be considered in theoretical assertions attempting to link cardiac responses to such psychological processes as those subsumed under orienting and attention.

Psychophysiological Theories of Attention

Sokolov's Theory

As numerous authors have pointed out, the notion that the orienting reflex is an integral aspect of attention is derived from the work of Soviet physiologists, including Sokolov (1963, 1969, 1975, 1977) whose work has provided much of the impetus for OR research and theory (see Brackbill & Fitzgerald, 1969; Cole & Maltzman, 1969; Graham & Clifton, 1966; Graham & Jackson, 1970; Porges & Coles, 1976). Historically, Pavlov (1927) described the OR as an investigatory or "What is it?" reflex that brings the organism closer to the source of stimulation. Sokolov subsequently constrained Pavlov's broad definition. In Sokolov's formulation, the OR refers to a reflexive attention response to stimulus change (see Porges & Coles, 1976).

The OR then, as defined by Sokolov, is the first response of the organism to any type of stimulation. Sokolov also suggested that the "laws" governing the OR are common to all sensory modalities (i.e., they are nonspecific in this respect). The function of the OR is said to be to increase the reception of stimuli. He also proposed that responses reflecting an OR should be the same to both stimulus onset and offset. Responses from somatic, autonomic, and

cortical systems have been identified as components of the OR and include increased receptor sensitivity, orientation of the body or head toward the stimulus, cessation of ongoing activities, EEG activation (desynchronization), and a divergent vasomotor response consisting of cephalic vasodilation with peripheral vasoconstriction.

If stimulus intensity reaches a critical "aversive" value, Sokolov proposed that a defensive reflex is elicited rather than an OR. Functionally, the DR is said to limit the influence of aversive stimulation on the organism.

Sokolov has proposed that the OR has two additional properties: The first is habituation (extinction) during which the OR shows a decrement as a function of stimulus repetition. Second, the OR is re-evoked by any detectable change in the stimulus, i.e., elicitation of an OR is a function of stimulus change per se. Thus there are two basic stages in the Sokolovian model of OR elicitation. First, repeated presentations of a stimulus leads to the development of a "neuronal model" of the stimulus. This neuronal model is said to include not only simple qualitative, intensive, and temporal characteristics, but may also include more complex or abstract relationships (e.g., the sequence or pattern of stimuli, the probability of occurrence). Second, subsequent stimulus input is then compared to the previously stored characteristics of the stimulus. If there is a "match" between this input and the neuronal model, the OR is suppressed (habituation) whereas a "mismatch" leads to OR evocation.

Although Sokolov did not elaborate on cardiac responses as a component of orienting, his theory has played a prominent role in

psychophysiological approaches employing HR responses, particularily the attempt by Graham to integrate the OR concept and the Laceys' "environmental intake-rejection" hypothesis. Therefore, the views of the Laceys will be discussed prior to turning to those of Graham.

Laceys' Theory

The Laceys' environmental intake-rejection hypothesis postulates a relationship between the direction of HR changes and the individual's sensitivity to environmental stimuli (see J. Lacey, 1959, 1967; Lacey & Lacey, 1974). On the one hand, they argued that HR decreases found in attention demanding tasks (e.g., reaction time) not only reflect a "taking in" attitude but actually facilitate sensitivity to environmental stimuli. On the other hand, they argued that HR increases that have been found during problem solving tasks (e.g., mental arithmetic) not only reflect a "rejection" attitude toward environmental stimuli, but also facilitate cognitive processing. It is important to note that the emphasis of this position is on the functional significance of HR changes for sensorimotor behavior. The model has been described as a cortical feedback model according to which cardiovascular dynamics are held to be both causes and effects of the central nervous system. The Laceys proposed that the mediating mechanism underlying the functional significance of HR change is the negative feedback loop involved in the carotic and aortic baroreceptor reflexes. (Baroreceptors are mechanoreceptors or stretch receptors that are sensitive to fluctuations in arterial blood pressure, see Guyton, 1971.) Thus HR acceleration, they suggest, leads to an

increase in blood pressure in the areas of the stretch receptors of the barosensory loop which presumably leads to cortical inhibition for filtering out irrelevant stimuli or to ensure the raising of thresholds for aversive stimuli. Conversely, HR decreases lead to just the opposite set of events within these systems.

The basic contention that barosensory mediated HR deceleration plays a causal role in attention has met with widespread criticism (for critical reviews, see Elliott, 1972, 1974; Hahn, 1973). Furthermore, there is solid neurophysiological evidence recently available which shows that barosensory reflexes are markedly affected by the respiratory cycle, i.e., changes in arterial blood pressure are not as effective in modulating HR when these changes occur during inspiration as they are when they occur during expiration (see Davidson, Goldner, & McClosky, 1976; Eckberg & Orshan, 1977; Haymet & McClosky, 1975; Jordan & Spyer, 1978; Lopes & Palmer, 1976). However, the Laceys' contention that phasic HR decelerations may reflect attentional states and phasic HR accelerations may be indicative of brief information processing episodes when defined in terms of responses to signal stimuli have found some experimental support (e.g., see Coles & Duncan-Johnson, 1975; Duncan-Johnson & Coles, 1974).

Graham's Theory

In two reviews of newborn, infant, adult human, and infrahuman studies on cardiac responses to simple, nonsignal stimuli, Graham suggested that the models of the Laceys and Sokolov are not inconsistent (see Graham & Clifton, 1966; Graham & Jackson, 1970). Graham

concluded that HR deceleration, which may occasionally be preceded by a short latency accelerative response, may be interpreted as the cardiac component of the OR. HR accelerative responses are viewed by Graham as the cardiac DR. In this model, consistent with the formulation by the Laceys, physiological (HR) responses of the ANS are viewed as having causal influences on central processes related to sensorimotor behaviors. Moreover, these discrete patterns of physiological responses (i.e., direction of HR change) are viewed as reflecting specific psychological processes (i.e., ORs and DRs). As will be discussed in the subsequent section of this chapter on the developmental shift problem, this interpretation by Graham has had a major impact on studies of attention and orienting during the infancy period.

Obrist's Theory

Obrist has offered another interpretation of directional HR responses, the cardiac-somatic hypothesis (see Obrist, 1976; Obrist, Webb, Sutterer, & Howard, 1970; Obrist, Howard, Sutterer, Hennis, & Murrell, 1973; Obrist, Howard, Lawler, Galosy, Meyers, & Gaebelein, 1974). Obrist views HR changes primarily as effects rather than causes as suggested by the Laceys and by Graham. Obrist suggested that the HR deceleration found during the foreperiod of a warned reaction time (RT) task may not reflect negative feedback mechanisms of the cardiovascular system nor orienting *per se*, but rather, a response to the metabolic demands of the body's musculature. Thus, in attentional tasks when HR decelerates, Obrist contends that it is the result of centrally initiated inhibition of both cardiac and

somatic activities. HR acceleration, on the other hand, is viewed by Obrist as reflecting increased metabolic demands of motor activity or in preparation for motoric activity (also see Elliott, 1974).

Obrist has reported results from two experiments which do not lend strong support to the contention by the Laceys that barosensory mediated HR deceleration plays a causal role in attention. In the first study, Obrist, Wood, and Perez-Reyes (1965) found that in human adults when the baroreceptor reflexes have been blocked pharmacologically, decelerative HR responses remained intact. In a subsequent study, it was found that pharmacological blockade of HR decelerative responses in one group of young human adults did not result in significant differences in RT performance when compared to a control group in which HR decelerative responses were not blocked (Obrist, et al., 1970). The conclusion reached by Obrist from these and other studies was that although peripheral ANS HR responses may reflect central states such as attention, their role does not appear to be causal. Furthermore, Porges (1976a; Reese & Porges, 1976) has reviewed several studies which lend support to the contention that neurotransmitter activities of the peripheral ANS (parasympathetic/cholinergic-sympathetic/catecholaminergic) parallel those of the central cholinergic-catecholaminergic systems (e.g., see Anisman, 1975; Manto, 1967; Williams, Hamilton, & Carlton, 1974). Changes in these central neurotransmitter activities via pharmacological and surgical manipulations were accompanied by parallel changes in peripheral ANS neurotransmitter activities. However, changes in peripheral ANS activities (e.g., via peripheral blocking) were not

linked to changes in the central neurotransmitter activites nor linked to disruptions in performance.

These findings suggest support for the notion that peripheral ANS measures such as HR may provide useful information about CNS mediated processes but are not consistent with the formulation that these peripheral ANS system responses are causally linked to these processes and, hence, to behavior.

Porges' Theory

Porges (1976a) presented a two-component model according to which different aspects of HR change are said to reflect different attentional components. From a behavioral viewpoint, he has pointed out that his model is based upon the work of James (1890). Briefly, James distinguished two psychological processes associated with the concept of attention. The first was referred to as passive-involuntary attention and was defined as the immediate or reflexive response to objects that directly affected the sensory systems. The second was referred to as active-voluntary attention and was associated with interest and selection and could be directed toward ideational or representational objects.

In the model offered by Porges, HR response components have been empirically identified which are said to parallel the two categories of attention described by James (e.g., see Cheung & Porges, 1976; Porges, 1972; Porges & Raskin, 1969; Walter & Porges, 1976). The first component is said to reflect reactive attention and consists of two reflexive HR responses: The first is a short latency (within 1 sec of stimulus onset) HR response which is usually decelerative.

The second is a longer latency (within 2 to 6 sec of stimulus onset) directional HR response. Both of these responses are vagally mediated and Porges suggested the first may parallel Sokolov's limited definition of an OR as an intensity sensor. The second response may be either deceleration to novel or mild stimuli or acceleration to meaningful or intense stimuli.

The second major HR response component has been interpreted by Porges as reflecting a state of sustained (or voluntary) attention and consists of suppression of both HR variability (HRV; successive intervals between beats become more regular) and respiratory amplitude variance (RAV; temporary cessation or more frequent shallow breathing). The latency of this component has been found to be around 6 sec after stimulus onset and may continue, according to Porges, for as long as the individual elects to attend. This sustained component of attention may be the result of greater vagally mediated coupling between HR and respiration. In Porges' model, in contrast to the models of the Laceys and Graham, peripheral ANS changes are not viewed as causing attention.

More recently, Porges has elaborated his theory concerning these behavior-physiologic relations in the broader context of what he refers to as a "continuity model" (see Porges & Smith, Note 3). According to this continuity formulation, individual differences in competence and performance are viewed as peripheral manisfestations of individual differences in the organization and integration of the CNS. The principle assumption of this model is that of a continuity among various levels of functioning, i.e., CNS, ANS, behavioral. Thus far, the emphasis of the model has been on inhibitory aspects of behavior

that have been linked to central cholinergic systems and functioning. The arguments advanced by Porges and his group in favor of this theoretical orientation rest upon the following main points: (a) balance in central inhibitory-excitatory activities (cholinergic-catecholaminergic) have parallels in peripheral ANS activity and observable behaviors; (b) physiologically, patterns of variability in HR (RSA) are mediated by the CNS; (c) the final common pathway for patterns of HRV is the parasympathetic vagus (cholinergic/inhibitory) of the ANS; (d) HR responses such as decreases in HR and HRV have been associated with states of sustained attention; (e) these cardiac responses are primarily mediated by the vagus; (f) behavioral inhibition (e.g., suppression of irrelevant ongoing activities) is a prerequisite for optimal performance on tasks which entail sustaining a state of attention.

Thus, according to Porges' theoretical orientation, individual differences in behavioral performance (e.g., reaction time) and physiologic responses such as cardiac responses linked to attentional components may be interpreted as parallel outputs of or reflecting the status of the CNS substrates mediating states of attention as they interact, of course, with situational factors. He has interpreted measures of spontaneous levels of HRV as a crude index of the status of the balance of central inhibitory-excitatory systems and used levels of HRV as the predictor variable for behavioral and cardiac responses in attention demanding tasks. More recently, he derived the weighted-coherence (C_w) function from cross-spectral time series statistics to quantify respiratory influences on HR. The C_w function has been interpreted as a more exact, noninvasive estimate of the status of

central inhibitory-excitatory functioning than levels of HRV. Moreover, Porges has speculated that individual differences in the C_W function may serve as an index of a stable characteristic of the CNS that is predictive of individual differences in performance and cardiac response parameters on attention demanding tasks. Evidence regarding these speculations underlying the continuity model as well as the implications of these data and Porges' model for the present study will be examined in subsequent sections of this review.

Summary

The HR-psychophysiological positions overviewed thus far may be distinguished on the basis of the behavioral significance they attribute to peripheral ANS changes in HR. On the one hand, the Laceys and Graham suggest that peripheral ANS changes in HR modulate such central events as attention. However, the previously cited findings with pharmacological blocking agents by Obrist and his group and the more recent neurophysiological findings on respiratory influences on baro-sensory feedback on HR would seem to make this position untenable. On the other hand, both Obrist and Porges view these peripheral changes in HR as effects of or reflective of centrally mediated inhibitory processes. The Obrist-Porges position appears to be more consistent with the extant empirical evidence. However, despite differences in the interpretations of the behavioral significance of HR changes, all of these positions agree with the contention that cardiac responses can be meaningfully related to orienting and attention. In the next section I will discuss additional evidence bearing on this claim.

HR-Psychophysiological Studies of Attention

Reaction Time, Visual Search, and Cardiac Responses

Although evidence supporting the general hypothesis that cardiac response parameters can be meaningfully related to attention has been obtained from experiments employing visual search and vigilance tasks (e.g., see Coles, 1972, 1974), the simple, warned reaction time (RT) task with adult human males as subjects has been, by far, the most commonly used procedure. The general assumption underlying the use of the RT procedure is that the degree to which subjects are attending during the preparatory interval (PI) may be inferred from their RT scores. (For a discussion of RT measures *per se*, the reader is referred to Pachella, 1974.) Regarding HR responses, the well-documented findings are that during the PI, HR typically increases with the onset of the warning signal and then decreases and usually reaches its nadir approximately at the time of the go signal (when the subject is to respond). In this experimental context, the prediction offered by all of the previously cited models is that if RT and HR responses are related to some component of attention, then a significant relationship should be found among these measures. Indeed, most of the HR-RT studies to date have reported moderate but significant correlations such that greater magnitude HR decelerations were associated with faster RT scores (e.g., Cocquery & Lacey, Note 4; Lacey, 1967; Lacey & Lacey, 1974; Obrist, Webb, & Sutterer, 1969; Obrist, et al., 1970; Webb & Obrist, 1970). However, there have been a few studies which have not found significant RT-HR deceleration magnitude correlations (e.g.,

Jennings, Averill, Opton, & Lazarus, 1971). Porges (1972) has suggested that some of these discrepancies may be due to the use of a fixed PI. He suggested the fixed PI procedure may confound temporal conditioning of anticipatory responses to the go signal with processes underlying sustained attention. In support of this notion, he found that in a fixed PI RT task, the HR deceleration response increased in magnitude and its nadir moved closer to the time of the imperative signal across trials. RT performance was found to improve over the first five trials and remain stable thereafter. Moreover, none of the HR response measures (decreases in mean HR and HRV) correlated with RT performance in this fixed PI task. However, with another group of subjects run under a variable PI condition, Porges found a significant correlation between the magnitude of HRV decreases and RT scores such that greater reductions in HRV were associated with faster RT scores. In interpreting these data, the point may by Porges was not that fixed PI-RT procedures do not involve sustained attention, but rather, within this type of task temporal conditioning develops, and to the extent that this occurs, the attention demanding characteristics of the task may be reduced.

Several studies have also examined HR-performance relationships in RT and visual search tasks in subjects for whom attentional deficits have long been hypothesized to play a prominent role in cognitive deficits. In a study comparing nonretarded and retarded young adults, Krupski (1975, 1976) found that retardates showed significantly slower and more variable RT scores that did nonretarded controls. This finding was clearly in line with many previous RT studies with mentally

retarded individuals (for a review, see Baumeister & Kellas, 1968). However, retardates also showed lower magnitude HR decelerations during the foreperiod than did the nonretarded controls. Krupski's conclusion that these results reflected an inability of the retardates to sustain a state of attention throughout the PI was consistent with previous interpretations of RT studies (e.g., see Denny, 1964). In a study comparing retarded adolescents and nonretarded grade-school age children (mental age match), Porges and Humphrey (1977) examined performance and HR and respiratory response parameters within a visual search paradigm. In addition to the poorer performance by the retardates on this task, they also showed increases in HRV and respiration amplitude variability on task; the nonretarded controls showed superior performance and suppression of both HRV and RAV.

Sroufe et al. (1973) used a fixed foreperiod RT paradigm in a study comparing grade-school age boys referred for learning disabilities (minimal brain dysfunction syndrome) and nonreferred age-mates. Consistent with previous RT studies of learning disabled and hyperactive children (e.g., see Cohen & Douglas, 1972), the referred children showed slower and significantly more variable RT scores than the controls. In addition, they showed lower magnitude HR decelerative responses during the PI than did the controls. More recently, Porges et al. (1975) employed a within-subjects design with a variable PI-RT task (10, 15, 20 sec PIs) to examine his two-component model of attention and the widely hypothesized attentional deficit associated with hyperactivity. In the placebo condition, hyperactive children showed both behavioral and physiologic response patterns consistent

with the hypothesized deficit in sustaining a state of attention (i.e., there were increases in mean HR and HRV during the last 5-sec interval or tonic period of the PI). When the learning disabled and hyperactive children in the Sroufe and Porges studies were treated with methylphenidate (Ritalin), their RT performance improved; Sroufe found greater magnitude foreperiod HR decelerations whereas Porges found (in contrast to the placebo condition) reductions in mean HR and HRV during the tonic period.

These studies support the contention that attentional deficits are associated with mental retardation and hyperactivity and that these deficits may be observed at both the behavioral and physiologic levels. It is also noteworthy that evidence was found in the study by Porges et al. that these attention deficits were related to a sustained component of attention and not to a reflexive OR component when the latter was indexed by HR accelerative responses to the warning stimulus: Porges et al. did not find differences in this HR response component in his sample of hyperactive children. Holloway and Parsons (1971, 1972) have reported very similar findings with brain damaged adult subjects. Other studies with adults have found that these HR responses (the initial HR acceleration and the subsequent decreases in HR and HRV) were not only statistically independent, but also differentially sensitive to manipulations of stimulus significance, discriminability, and response requirements (e.g., see Coles & Duncan-Johnson, 1975; Walter & Porges, 1976). These results have been interpreted as supporting the contention that differential cardiac response components may be correlates of different attentional components.

Summary

The above discussion was not intended to be a complete review of all of the HR-psychophysiological research on attention, but rather, to show that there seems to be sufficient empirical support to justify the general hypothesis that cardiac response parameters can be meaningfully related to attentional processes. Although the theoretical assertions offered to interpret these data are not very elaborate, and there is an obvious need for further research on all aspects of these physiologic-behavior relations, it seems equally clear that advances have been made. To date, evidence from studies with children and adults indicates that reasonably reliable time-locked decreases in HR and HRV occur in situations requiring moderate to more prolonged states of attention. Evidence has also been reported showing that these peripheral ANS response measures are often correlated with behavioral-performance measures such as reaction time. Moreover, studies of individuals from populations in whom attention deficits are thought to play a prominent role in their cognitive deficiencies, showed parallel deficiencies in behavioral and cardiac measures thought to reflect central processes mediating states of sustained attention. An equally interesting finding from the studies with hyperactive children was that pharmacological treatment not only resulted in improved behavioral performance, but also a parallel shift in cardiac response measures of sustained attention.

Individual Differences and Attention

In general, the question of individual differences in psychophysiological studies of attention has been the focus of relatively

little research. However, several studies that have addressed this problem have sought to relate individual differences in some index of attention to indices of individual differences in performance on subsequent tasks such as those involved with conditioning, habituation, or discrimination learning. For example, Ingram and Fitzgerald (1974) examined and found a significant relation between individual differences in OR magnitude (as measured by skin potential responses) and condition-ability in a conditional descrimination and discrimination reversal paradigm with 3-month old infants. Their findings showed that high-magnitude orienters learned the discriminations whereas the low-magnitude orienters did not. A study by Cousins (1976) may serve as another example of this type of research. In this study with 9- to 11-year old males, Cousins found a relationship between the magnitude of a cardiac OR (HR deceleration) and subsequent learning rate (trials to criterion) in a two-choice discrimination learning task: "Fast learners" showed a significantly larger magnitude OR than did the "slow learners."

Of particular interest, however, is the suggestion offered by Ingram and Fitzgerald (1974) as a possible interpretation of their findings. They pointed out that the individual differences observed in their study were consistent with the Pavlovian concept of "nervous-system dynamism" which refers, theoretically, to the balance of cortical inhibitory-excitatory processes. Moreover, as Fitzgerald and Brackbill (1976) noted in reference to these findings, "...habituation and discrimination both involve inhibition; what we may be seeing are simply different manisfestations of a general inhibitory ability." Fitzgerald and Brackbill also pointed out that the question remains as

to whether these individual differences reflected differences in maturational rate of inhibitory-excitatory balance or individual differences in this balance which would be predictive of subsequent stable individual differences in these processes later on in development. Note the similarity between this theoretical stance and the previously cited one by Porges concerning the continuity among various levels of functioning. Recall that Porges also speculated that individual differences in the balance of the theoretical inhibitory-excitatory continuum of the CNS are paralleled by individual differences in behavior and ANS activity. However, in his approach to this question, Porges has employed a slightly different experimental strategy. He has attempted to identify individual differences in spontaneous levels of what is essentially an inhibitory physiologic control system, and then relate this to subsequent performance and physiologic responses on tasks which have been linked to a general inhibitory capacity rather than attempting to predict individual differences from one measure of responsivity to another. As mentioned previously, Porges initially employed measures of spontaneous levels of HRV as a noninvasive estimate of the parasympathetic-vagal control of the heart and, by inference, the status of the CNS cholinergic-inhibitory systems. Estimates of HRV were then used as a predictor variable for performance as well as cardiac response measures in attention demanding tasks which entail inhibition of ongoing activities for optimal performance.

In order to evaluate these hypotheses in the present study, the physiology of cardiac function will be considered since the choice of a viable noninvasive measure of the parasympathetic influences on the

heart should be consistent with the physiologic basis of cardiac pacemaking. Consequently, it seems appropriate to briefly examine cardiac pacemaking processes prior to turning to the literature on measures which have been used to estimate inhibitory influences on the heart and the relations between these estimates and attention processes.

Physiology of Cardiac Pacemaking

The heart is innervated by the two antagonistic branches of the ANS, the parasympathetic vagus (cholinergic) and the sympathetic (catecholaminergic) nerves. However, it has long been known that even if the heart is excised it will continue to beat indicating that to a certain extent pacemaking is endogenous and, therefore, not solely dependent upon exogenous neural input for the initiation of contractions. (For more detailed discussions of the physiologic basis of cardiovascular function, the reader is referred to Berne & Levy, 1972; Brooks & Lu, 1972; Calaresu, Faiers, & Mogenson, 1975; Goldman, 1973; Gunn, Wolf, Block, & Person, 1972; Guyton, 1971; Schneiderman, Dauth, & VanDercar, 1972; Schneiderman, Francis, Sampson, & Schwaber, 1974).

Endogenous pacemaking. There are two primary structures located in the wall of the right atrium responsible for the initiation of contractions, the sinoatrial (SA) node and the atrioventricular (AV) node. Normally, cardiac impulses for contraction originate within the SA node. Impulses then spread through the atria to the AV node. The EKG P-wave represents the bioelectric activity of atrial contractions (see Figure 1). From the AV node, impulses spread down the AV bundle (bundle of His) to the right- and left-bundle branches and then along

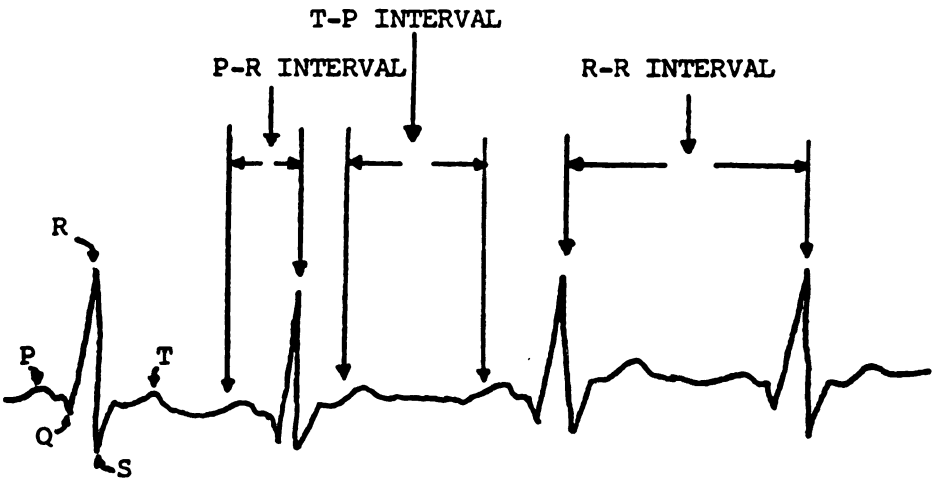

Figure 1. Diagram of a raw EKG tracing illustrating various components of the cardiac cycle.

the Purkinjie fibers to the myocardial cells for ventricular contractions. The QRS-complex of the EKG represents the bioelectric stimulus for ventricular contractions. Although the QRS-complex is ususally taken to represent the ventricular contraction, the contraction actually last longer than the duration of the QRS-complex. The T-wave of EKG tracings represents the repolarization of the ventricles.

In sum, the cardiac cycle may be described as atrial systole, ventricular systole, and the "resting" stage between beats. The SA node is the normal endogenous pacemaker. There are other potential pacemakers within the heart, collectively referred to as ectopic pacemakers, which only function in pathological or emergency situations, i.e., the ectopic atrial, AV junctional, and indioventricular pacemakers (e.g., see Goldman, 1973).

It should be noted that the mechanisms responsible for spontaneous cardiac pacemaking are still incompletely understood. Recently, however, Pollack (1977) proposed a model of the mechanisms underlying spontaneous pacemaking. As was noted, sectioning of both parasympathetic and sympathetic fibers innervating the heart does not halt beating. It has also long been known that the sympathetic fibers innervating the heart are excitatory (i.e., positive ionotropic or increased contractile force and chronotropic or rate increasing effects) via storing and secreting catecholamines, their neurotransmitter substance. However, while sectioning the sympathetic fibers does not halt the heart, complete depletion of cardiac catecholamines does. Pollack reviewed evidence that suggests the presence of extraneural catecholamine storage in the SA node. Moreover, he showed that endogenously synthesized catecholamines

are indespensible for the spontaneous activity of the SA node. Pollack thus proposed an interaction between the extraneural (endogenous) and neural (exogenous) catecholamines in normal cardiac pacemaking that takes into account the regulatory role of sympathetically discharged catecholamines. When endogenous catecholamines have been depleted, and pacemaking tends to cease, exogenous sympathetic catecholamines serve to supplement endogenous stores and thus augment pacemaking. Furthermore, Pollack's model includes a three-way interactive function among exogenous parasympathetic and sympathetic inputs, and the endogenous catecholamines that takes into account and may explain the local (cardiac) regulatary role of the parasympathetic-cholinergic system (inhibitory) in pacemaking. Acetylcholine (parasympathetic neurotransmitter) inhibits SA node depolarization via a set of processes that diminishes the rate of $Ca^{2}+$ entry into pacemaking cells. It is the entry of $Ca^{2}+$ into pacemaking cells that initiates depolarization (the stimulus for $Ca^{2}+$ entry is the catecholamines).

Exogenous pacemaking. As noted above, the heart is innervated by the two antagonistic branches of the ANS, the vagus and the sympathetic nerves. The heart's action is inhibited by the vagus but augmented by the sympathetics. Each type of input exerts a somewhat continuous influence on the heart. Efferent vagal nerves originate from the dorsal motor nucleus of the vagus or the nucleus ambiguus in the brainstem medulla and are often referred to as the medullary cardioinhibitory neurons. It appears that the left vagus innervates the AV node whereas the right vagal fibers innervate the SA node (e.g., Gunn, et al., 1972). Some evidence also exists to suggest that the vagus may innervate the

ventricular walls so that it may inhibit myocardial contractile force (negative inotropic effect). Stimulation of the vagus results in bradycardia (negative chronotropic effect) whereas sectioning the vagus produces tachycardia (positive chronotropic effect).

The preganglionic efferent sympathetic fibers originate from the intermediolateral cell column of the upper five thoracic levels of the spinal cord. The preganglionic fibers from the right sympathetic chain appear to innervate the SA node and those from the left chain appear to innervate the walls of both ventricles. Stimulation of the right sympathetic chain postganglionics results in a positive chronotropic effect (via beta adrenergic receptors). Stimulation of the left sympathetic chain postgangionics results in a positive inotropic effect (via beta adrenergic receptors).

The biomedical and physiological literature also indicates that HRV or beat-to-beat fluctuations stems from efferent vagal input to the SA node (e.g., see Calaresu, et al., 1975; Chess, et al., 1975; Guyton, 1971; Katona & Jih, 1975) and is observed as the fluctuations in time of the T-P or R-R intervals of successive cardiac cycles (see Figure 1). Another source of HRV is the P-R interval. However, the literature indicates that the variance of this interval is minimal relative to the variance of the T-P or R-R intervals, except in several cardiovascular diseases. The relative lack of variability of the P-R is viewed in the cardiological and physiological literatures as indicating that pacemaking mechanisms <u>within</u> the heart (endogenous) are functioning normally (e.g., see Goldman, 1973; Guyton, 1971). Thus efferent vagal activity appears to be the primary source or final common pathway of

"normal" HRV and is indicated by beat-to-beat fluctuations with fairly constant P-R intervals.

Individual Differences in HRV and Attention

Despite disagreements among psychophysiologists over the interpretation of the behavioral significance they attribute to cardiac responses, they agree that the final common pathway for the cardiac responses linked to orienting and attention is the efferent vagus rather than sympathetic influences (see Graham & Jackson, 1970; Lacey, 1967; Obrist, 1976; Porges, 1976a). Since HRV may and has been used as a rough estimate of vagal efferent influences on the heart (e.g., Chess, et al., 1975; Katona & Jih, 1975), it seems reasonable to ask: Are individual differences in HRV related to HR responses and performance measures in studies of attentional phenomena? As noted before, only a few studies in the literature have addressed this question. However, Porges and his group have put this question to empirical test and it is to this research that the review now turns.

In the previously cited HR-RT study with male college students, Porges (1972) found in the variable PI group, a significant between-subjects correlation ($r = -.774$) between the mean pretrial level of HRV and mean RT performance such that higher levels of HRV were associated with faster RT scores. The magnitude of the suppression of HRV during the PI was also significantly correlated with RT performance in this group ($r = .717$) with greater reductions in HRV associated with faster RT scores. In a subsequent study with male college students as subjects, Porges (1973) asked whether spontaneous

levels of HRV recorded during a resting period (baseline) prior to the RT sessions would be predictive of subsequent RT performance. Baseline levels of mean HR and HRV were intercorrelated (between-subjects) with mean RT scores within each of the two groups (fixed PI- and variable PI-RT groups). Consistent with the findings of his previous study, the only significant correlation to emerge out of this analysis was between resting level HRV and mean RT ($r = -.70$) within the variable PI group. Moreover, when the subjects within each PI group were subdivided into high-, mid-, and low-HRV groups, the high-HRV group showed significantly faster RT scores that either the mid- or low-HRV groups, the latter two groups did not differ significantly in RT performance. There were no significant differences in RT among the three HRV subgroups within the fixed PI condition. Unfortunately, Porges apparently did not measure cardiac responses during the RT tasks in this study.

In the RT-HR study with hyperactive children, recall that Porges et al. (1975) reported (a) increased levels of mean HR and HRV (pre-trial) on methyphenidate as compared to the placebo condition, and (b) decreases in mean HR and HRV from the pre- to tonic-periods (on task) in the methyphenidate condition but increases in these measures in the placebo condition. However, when these children were subdivided into slow and fast groups based on their RT scores during the placebo condition, post-hoc analysis indicated that only 2 of 8 children in the fast group showed improved RT performance on methylphenidate whereas 7 of 8 children in the slow group showed improved RT performance. Interestingly enough, only the slow group showed significantly increased reductions of HRV on task (pre- versus tonic-period) on methylphenidate.

In the Porges and Humphrey (1977) study the retarded subjects were found to have markedly lower levels of baseline HRV as compared to the nonretarded controls. In addition to their poorer performance on the visual search task (mean detection latency equaled 35.3 sec vs. 19.4 sec for the nonretarded subjects), they showed on-task increases in HRV and respiratory amplitude variability in striking contrast to the on-task reductions in these measures exhibited by the nonretarded control subjects.

Of interest for the present study are two studies which examined the relations between levels of HRV and cardiac responsiveness to moderately intense stimuli of newborn human infants who were in quiet-awake states. In the first study, Porges et al. (1973) found both high- and low-HRV groups (based on mean pretrial levels of HRV) showed significant HR accelerations in response to the onset of a 30 sec moderately intense auditory stimulus. However, the magnitude of this response was significantly greater for the high-HRV newborns. Analysis of tonic HRV responses (differences between the mean HRV for the last 5 sec before stimulus onset and the last 5 sec before offset) revealed significant decreases in HRV by the high-HRV group and significant increases for the low-HRV newborns. Finally, only the High-HRV newborns showed a significant cardiac response to stimulus offset (i.e., HR deceleration). In another newborn study, Porges et al. (1974) compared HR responses of high- and low-HRV groups (based on pretrial levels) to a 30 sec change in room illumination. It was found that in response to stimulus onset, the high-HRV newborns showed a significant biphasic response consisting of an initial deceleration followed by an

acceleration; the low-HRV newborns responded with a longer latency deceleration. Analysis of offset responses indicated that only the high-HRV newborns showed a significant HR response which changed across trials from an initial acceleration to deceleration.

The studies just cited provide at least tentative support for the notion that individual differences in the organismic variable HRV, in addition to situational variables such as the type of task and stimulus qualities, are related to or are predictive of behavioral-performance and cardiac response parameters postulated as measures of attention. Higher levels of HRV, interpreted as rough or crude estimates of efferent vagal influences on the heart and, by inference, the status of central cholinergic functions, were associated with superior levels of performance (e.g., RT) as well as magnitude changes in cardiac response measures (HR and HRV) indicative, theoretically, of states of greater attentiveness. However, while there is data linking spontaneous (baseline) and pretrial levels of HRV to measures of attention, negative findings also have been reported (see Coles, Porges, & Duncan-Johnson, 1975). These authors suggested that the lack of a significant correlation between HRV and RT scores in their study might have been due to the restricted range of RT scores. Porges and Smith (Note 3), however, recently have suggested another possible explanation that relates to the nature of the HRV measures used as estimates of vagal control.

Higher Neural Influences on the Efferent Vagus and Patterns of HRV

As pointed out by Porges and Smith, HRV measured as either the standard deviation or variance of beat-to-beat fluctuations is a

"crude" estimate of parasympathetic-vagal influences on cardiac activity (see also Porges, 1976a). There are several reasons for their use of the qualifier "crude" (Porges, Note 6). Although the efferent vagus to the heart is the final common pathway for HRV, a substantial neurophysiological and neuroanatomical literature on the central neural regulation of the cardiovascular system has identified a number of projections to the medullary cells of origin (nucleus ambiguus or dorsal motor nucleus) of these cardioinhibitory neurons (e.g., Calaraesu, et al., 1975; Schneiderman, et al., 1974). This literature also indicates that while neural regulatory mechanisms have been identified at all levels of the neuroaxis, it also has long been known that brainstem medullary centers are prominent sites for the neural integration of both supramedullary and peripheral influences on vagal projections to the heart.

Baroreceptor, chemoreceptor, and respiratory influences on the cardioinhibitory neurons are three main integrative functions which occur at this level. The afferent fibers of these mechanisms project to the medullary nucleus tractus solitarius: baroreceptor and chemoreceptor afferents via the glossopharyngeal and vagus nerves, pulmonary stretch afferents via the vagus. In respiratory sinus arrhythmia increases and decreases in HR have been shown to be systematically related to respiratory changes such that during inspiration HR increases and during expiration HR decreases. These rhythmic changes in HR result from "gating" or inhibitory influences on the vagal efferents to the heart during inspiration. Moreover, recent studies with a variety of species (humans, cats, dogs, rabbits) have shown that HR decreases

resulting from increases in or stimulation of baroreceptor or chemoreceptor afferents are markedly affected by the respiratory cycle (Davidson, et al., 1976; Eckberg & Orshan, 1977; Haymet & McClosky, 1975; Jordan & Spyer, 1978; Lopes & Palmer, 1976). In these studies, increases in baroreceptor and/or chemoreceptor afferent activities resulted in HR decreases only during expiration and did not have any discernable influence on HR during inspiration. The conclusion was drawn, therefore, that medullary inspiratory neurons serve an integrative function and thus gate other neurally transduced influences on cardioinhibitory-vagal activity. Evidence also points to a primary role of respiratory sinus arrhythmia in modulating vagal efferents or the patterns of cardiac pacemaking. Lopes and Palmer (1976) found that afferent impulses from pulmonary stretch receptors often override the "centrally generated respiratory rhythm" in the modulation of vagal efferent activity. (For a recent review of the neurophysiology of respiration, the reader is referred to Wyman, 1977). These neurophysiological findings suggest that HRV not only reflects efferent vagal influences on the heart, but also the "sum" of several neural inputs to this final common pathway and hence, a major reason underlying the use of the qualifier crude.

From a statistical viewpoint, Porges (Note 6) has also pointed out that levels of HRV as estimated by the standard deviation or variance statistics would be affected by the presence or absence of slow changes or trends in HR. These slowly changing levels of HR may or may not be related to the neural control of the heart (e.g., circulating hormones, see Schneiderman, et al., 1974).

These and other considerations led Porges to suggest that patterns rather than levels of HRV may provide more exact estimates of vagal influences on the heart as well as a peripheral, noninvasive indicator of the status of the central inhibitory-excitatory balance continuum (Porges, Note 2, 6, 1976a; Porges & Smith, Note 3; Porges, et al., Note 5). Consequently, he turned to the application of time series statistical techniques--spectral and cross spectral analyses--to quantify respiratory mediated influences on the patterns of HRV.

Spectral Analyses Estimates of the Neural Control of the Heart

Essentially, spectral analysis is a method by which the frequencies (defined as the proportion of a sinusoidal function occurring in 1 sec) of an ongoing rhythmic process (in this case respiration or HR) are identified and the proportion of the total variance of the series associated with each frequency component is partitioned. Cross spectral analysis is then performed to obtain an estimate of the proportion of variance accounted for or the influence of each frequency component of one spectrum on the corresponding frequency component of the other spectrum. The coherence function from cross spectral analyses provides the estimate of the proportion of variance of one frequency component accounted for by the variance of the corresponding frequency component and, as Porges (1976a) pointed out, is analogous to the omega-squared statistic described by Hays (1963). From these techniques Porges derived the weighted-coherence (C_W) function as an estimate of the relative parasympathetic-vagal control of the heart. The C_W function

is defined as the percentage of cardiac activity that is coherent with respiration within the normal frequency bandwidth of respiration (see Appendix A for a more complete discription of the formulas used in these techniques).

Porges (1976a) originally examined C_W in two studies of hyperactive children. Recall that when the hyperactive children in the Porges et al. (1975) study were off-drug, they showed behavioral and HR responses consistent with an hypothesized attention deficit. When these children were treated with methylphenidate, however, their performance improved and their HR responses changed from increases (off-drug) to decreases in mean HR and HRV during the tonic period of the PI. Consequently, Porges (1976a) asked whether hyperactive children would show a relative lack of vagal influences on the heart (C_W) as compared to nonhyperactive controls. In the first study baseline HR and respiration were simultaneously recorded for hyperactive children and age-matched controls. The control children were not treated with any medications whereas the hyperactive children where recorded during five conditions: placebo, off-drug (after all other treatment conditions), high- and low-dose of dextro-amphetamine, and a low dose of methylphenidate. The results revealed that the hyperactive children showed significantly lower C_W (in all treatment conditions) than the controls and that for the hyperactive children, C_W was significantly greater in the methylphenidate condition than in any of the other conditions. None of the other conditions differed significantly in C_W.

The second study by Porges (1976a) included only hyperactive children not previously treated pharmacologically for hyperactivity.

In this study the questions were asked as to whether hyperactive children would show lower levels of C_w prior to any pharmacological treatments as well as whether different dosages of methylphenidate would differentially affect C_w. The treatment conditions under which C_w was measured in this study were the same as in the previous study but also included conditions of a higher dose of methylphenidate, and before and after drug treatment conditions. C_w in the low-dose of methylphenidate condition was significantly greater than in any of the other conditions. Again it was found that none of the other conditions differed significantly in C_w.

In a third study, Porges et al. (Note 5) measured C_w for hyperactive children under conditions of high- and low-dose of methylphenidate, placebo, and off-drug. Again it was found that C_w in the low-dose of methylphenidate condition was significantly greater than in any of the other conditions which, again, did not differ significantly. The further question was asked in this study as to whether the increase in C_w in the low dose of methylphenidate condition was due to an overall suppression of cardiac activity not due to respiration rather than due to an increase in the absolute amount of cardiac activity coupled with respiration (see Appendix A for the derivation of these parameters). The analyses of these parameters indicated that the increase in C_w was due to a selective influence of the low dose on the absolute amount of cardiac activity coupled with respiration.

The findings of these preliminary studies with hyperactive children were interpreted by Porges and his associates along the lines of the above described continuity model. They also argued that spectral

analysis provided a viable, noninvasive technique by which the neural control of the heart can be more precisely studied. If this assumption is correct, then it would seem to follow that these findings suggest that a deficiency in the inhibitory control of the heart may be involved in the hyperactivity syndrome. Consistent with their continuity formulation, it also was suggested that this deficiency may point to a dysfunction in the theoretical inhibitory-excitatory continuum of the CNS as a possible physiological substrate of this syndrome. The findings on the differential effects of the various pharmacological conditions on C_w also appear to indicate that this dysfunction is susceptible to remediation by quite specific dosages of methylphenidate. Furthermore, when these data are taken together with those from the previously cited studies of hyperactive children (Porges, et al., 1975; Sroufe, et al., 1973), they lend at least indirect support to the proposal that the relative degree of respiratory sinus arrhythmia (C_w) may be predictive of both behavioral and cardiac responses thought to reflect the deficit in sustaining states of attention widely linked to hyperactivity. The apparent parallel changes in C_w and these indices of sustained attention as a function of pharmacological intervention are, indeed, quite striking.

Although the significant group results tend to support these interpretations, it is also interesting to note the wide individual differences in C_w observed among the hyperactive children in these studies. As pointed out, most of the children showed changes in C_w as a function of the treatment conditions as reported for the group results. However, several children with the lowest levels of C_w in

the off-drug condition not exhibited increased C_w on the lowest dose of methylphenidate, but also showed still further increases in C_w on the higher dose. There also appeared to be a third subgroup, which prior to the drug conditions, showed levels of C_w within the range of those of the control children in the first study (Porges, 1976a). Moreover, they did not exhibit changes in C_w across the treatment conditions.

Considering this latter data together with the individual differences reported in the Porges et al. (1975) study, it is not difficult to see why Porges and his associates hypothesized that the relative degree of respiratory sinus arrhythmia may serve as a stable, individual difference characteristic that reflects the overall status of the CNS and should be predictive of performance and cardiac responses associated with the construct of sustained attention. Unfortunately since Porges and his colleagues have as yet to report more direct tests of these hypotheses within the same experiment, the delineation of these relations and more adequate tests of the merits of these hypotheses must obviously await further studies.

Summary

As noted there is a paucity of solid empirical findings that bear on the general problem of individual differences in behavior-physiologic relations from HR studies of attentional phenomena. However, the studies cited above appear to have several interesting implications and suggest several approaches worthy of being pursued in future research. Two slightly different research strategies have been applied to these issues in psychophysiological studies. One approach has sought to examine and

found relations between individual differences on one measure of responsivity (e.g., magnitude of orienting) and measures of responsivity on subsequent cognitive tasks (Cousins, 1976; Ingram & Fitzgerald, 1974). These issues have also been explored in terms of an attempt to relate estimates of the neural control of the heart, thought to reflect the overall status of the integration and organization of the CNS, to subsequent measures of performance and physiologic responses linked to attentional phenomena (Porges, 1976a; Porges, et al., 1975; Porges & Smith, Note 3). Nevertheless, both approaches appear to have assumed that individual differences in processes subsumed under the theoretical constructs of attention are key factors involved in individual differences in cognitive behaviors. The data just cited tends to support this broad view of the role of attention in the organization of behavior. Clearly, what is needed now is a series of comprehensive studies aimed at clarifying the precision and scope of these relations.

It also seems that the Porges et al. notion of continuity among various levels of functioning provides a theoretical framework, though as yet not very elaborate, according to which several eminently testable hypotheses could be pursued. For example, whether or not the relative degree of respiratory sinus arrhythmia is a stable individual characteristic, since it is primarily CNS controlled, seems a reasonable question that ought to be pursued. It also seems that research on individual differences in respiratory sinus arrhythmia as they relate to or do not relate to individual differences in behavioral and physiologic measures of attention (orienting, sustained attention) would be another line of work that ought to be empirically advanced. Moreover to the extent that

the Porges continuity formulation is correct, and since it appears consistent with the points recently made by Fitzgerald and colleagues concerning the various manisfestations of a general inhibitory ability, it would seem that future studies on these methodological and theoretical points could prove valuable and provide more solid empirical information upon which a more comprehensive account of such physiologic-behavior relations could emerge. Furthermore, these points appear to have implications for HR-psychophysiological studies of attention during early human infancy; it is to this literature that this review now turns.

Infancy HR-Psychophysiological Studies of Attention

Unlike the typical paradigms cited above which have entailed more prolonged periods of time during which the subjects were to "pay attention," infancy HR studies have focused almost exclusively upon orienting reflexes. This experimental strategy, however, like that underlying the previously overviewed work with children and adults, has been based, in part, upon the growing body of evidence linking HR response measures to attention. As in the HR research with children and adults, the ubiquitous issues of response meaning, definition of viable dependent variables, and individual differences are major problems currently confronting infancy researchers. Infancy researchers also are challenged by an organism with a limited response repertoire relative to that of the young child or adult. On the other hand, it is equally obvious that the response repertoire of the human newborn undergoes quite rapid and apparently profound changes during the early course of development. Although it has long been recognized that the structural and functional

organization of infant behavior undergoes rapid and substantial changes, it has been quite another matter to develop reliable and valid measures that are sensitive to the subtleties as well as the complexities of these changes. Thus, consistent with the previously noted broad view of the role of attention in the organization of behavior, and since cardiac responses do not require elaborate motor or verbal capabilities, it is not difficult to understand the attraction that cardiac dependent variables have for many infancy researchers.

The Developmental Shift Problem

At the time of the Graham and Jackson (1970) review, HR studies of human newborns reported, almost without exception, monophasic HR accelerative responses to simple nonsignal stimuli. However, a few newborn studies also had been reported that did not find any significant HR response, either acceleratory or deceleratory (e.g., see Graham, Berg, Berg, Jackson, Hatton, & Kantowitz, 1970). These findings were in sharp contrast to those with older infants and adult subjects which showed that around 6-weeks to 2-months of age, but more reliably around 3- to 4-months of age, HR responses in these stimulus situations were decelerative (see Graham & Clifton, 1966; Graham & Jackson, 1970). This developmental trend had been observed in studies using cross-sectional (e.g., Graham, et al., 1970) and short-term longitudinal designs (e.g., Lipton, et al., 1966). Graham and her associates interpreted this apparently age-related change in the direction of this physiologic response as indicating a shift in the fundamental nature of the young infant's psychological response to stimuli, i.e., a shift from primarily defensive responding to orienting.

At the time of the Graham and Jackson review there also was evidence to suggest that the magnitude of HR deceleratory responses increased during the period of 6- to 16-weeks of age. These findings were interpreted as additional support for the contention that the capacity for orienting increases with development (e.g., see Graham & Jackson, 1970; Graham, et al., 1970; Rewey, 1973). However, the relation of increasing magnitude of HR decelerations with age has not been invariably found within this age range in subsequent studies (e.g., see Berg, 1974).

Subsequent to the reviews by Graham several investigators sought to answer her question that methodological rather than developmental factors may have been responsible for the apparent developmental trend. In these newborn studies such stimulus parameters as 'rise time' and 'intensity' as well as the organismic variable 'biobehavioral state' were carefully controlled since each of these factors had been previously associated with HR-accelerative-DRs in older subjects. (For reviews of these more recent infancy studies see Berg & Berg, Note 1; Clifton, 1974a). Interestingly, several studies in which slow rise time, moderately intense stimuli were presented to newborns in a quiet-awake state reported significant HR decelerations in response to: (a) onset of auditory, visual, vestibular stimuli (Adkinson & Berg, 1976; Forbes & Porges, 1973; Kearsley, 1973; Pomerleau-Malcuit & Clifton, 1973; Sameroff, Cashmore, & Dykes, 1973); (b) offset of a stimulus (Adkinson & Berg, 1976; Porges, et al., 1973; Porges, et al., 1974); and (c) in the absence of a signaled stimulus (Clifton, 1974b; Forbes & Porges, 1973). Thus it seemed that newborns were indeed capable of HR

decelerative responses and, according to the interpretation of directional HR responses by Graham and her colleagues, capable of orienting although apparently under limited conditions.

It is important to note, however, several studies in which these stimulus and state variables were manipulated for "optimal" orienting conditions did not find HR decelerative responses by newborns (e.g., see Graham, et al., 1970; Jackson, Kantowitz, & Graham, 1971; Pomerleau-Malcuit & Clifton, 1973). These conflicting findings raise several interesting and pertinent points: The first and perhaps most obvious concerns the basic question of the value of directional HR responses as an index of orienting. The important implication of these studies from the point of view of Graham and her colleagues is that the difficulty of elicting newborn HR decelerations suggests that the capacity for attending is not firmly established during the newborn period. The principle assumption of this view is that a specific HR response topography (deceleration) in these stimulus situations reflects orienting regardless of age. However, responses linked to attention other than directional HR changes such as head turning, sucking, and visual fixation have been repeatedly reported in newborn studies (e.g., see Fantz, Fagan, & Miranda, 1975; Fitzgerald, Strommen, & McKinney, 1977; Friedman, 1975; Sameroff, 1971, 1972). Although the results from these latter studies have not always been clearcut, overall they seem to suggest (at least indirectly) the absence of HR decelerative responses by the newborn may not unequivocally indicate a lack of some attentional capacity. Regrettably, to date there appears to have been no study with newborns that has focused on the relations between behavioral and

cardiac measures of orienting and attention. However, with 6-month-old infants, Lewis, Kagan, Campbell, & Kalafat (1966) found that total fixation times were significantly related to the magnitude of HR deceleration responses with longer fixation times associated with greater magnitude HR decelerations (also see Lewis & Spaulding, 1967). These findings suggest a need for more research with younger infants aimed at clarifying the relations between these measures and their developmental course.

Second, the negative findings cited above are of interest in that they suggest that although stimulus and state factors are important, it is still not clear what the necessary conditions are for newborn HR decelerative responses (Porges, 1974). A related point is that the more recent findings of newborn HR decelerations seem to challenge the previously cited developmental trend. Nevertheless, newborn HR decelerative responses have only been found under limited and strictly controlled conditions. In contrast, studies with older infants have observed HR decelerations under a much broader range of conditions, as well as to the stimuli newborns showed HR accelerative responses (e.g., see Clifton & Myers, 1969). In discussions of more recent findings it has been argued that whereas there does not appear to be an absolute developmental shift from HR acceleration to HR deceleration, these data may be interpreted as indicating the probability of eliciting HR decelerative responses increases over the opening months of extrauterine life (see Berg & Berg, Note 1; Clifton & Nelson, 1976; Clifton, 1974a). An increase in the liklihood of eliciting HR decelerations reflects the development of central mechanisms mediating orienting and, as such,

suggest a fundamental increase in information processing capacity during early infancy.

Thus, a basic issue confronting developmental psychophysiologists is whether directional HR responses provide a reasonable heuristic tool for the study of orienting during early infancy. Again, the main assumption underlying the interpretation of directional HR responses by Graham and her colleagues is that the cardiovascular system is sufficiently mature so that HR decelerative responses can be elicited, and whether an accelerative or decelerative response is observed, depends upon the infant's psychological response to the stimulation. In contrast to this theoretical stance, Hirchman and Katkin (1974) discussed several alternative hypotheses in a recent review. These hypotheses centered around the question of the functional maturity of the efferent vagal innervation of the heart. Recall that despite disagreements among psychophysiologists over the interpretation of the behavioral significance of cardiac responses in attention research, they agree that the final common pathway for these responses is vagal rather than sympathetic.

Therefore it is not surprising that the following hypotheses centered around the question of the development of vagal tone. (The concept of vagal tone refers to the extent to which neural impulses of efferent vagal fibers to the heart influence the rate and rhythm of the heart.)

Hirschman and Katkin addressed Obrist et al's (1965) arguement that the "developmental shift may be accounted for by the development of vagal tone in the first two months." However careful inspection of the Obrist et al. paper as well as of two subsequent reports of

developmental research (with grade school age children) failed to provide any statement by Obrist about the development of vagal tone (see Lawler, Obrist, & Lawler, 1976; Obrist, et al., 1973). Hirschman and Katkin then cited a hypthesis by Lipton et al. (1966) in which it was stated that vagal tone is present at birth and HR accelerative responses shown by the newborn are the result of inhibition of the vagus. Lipton et al. also proposed that inhibition of the vagus is not elicited later on in development.

In their monograph on HR-classical conditioning with newborns, Crowell, Blurton, Kobayshi, McFarlan, and Yang (1976) cited several studies they interpreted as suggesting cardiac reflexes function maturely by term birth (see Brady & Tooley, 1966; Dawes, 1968; Phillips, Agate, Silverman, & Steiner, 1964). However, while Phillips et al. acknowledge that the ANS is developed and functional at term, they also noted "...the complexity of its interactions quantitatively and qualitatively differs from those seen in the adult." Similarily, Porges (1976a, 1976b; Reese & Porges, 1976) proposed there is a shift in the relative balance of ANS influences on cardiac activities from predominately sympathetic (excitatory) to parasympathetic-vagal (inhibitory) influences during the early course of human development. Porges also suggested that the directionality of infant HR responses may be dependent upon this differential maturation of the parasympathetic and sympathetic control over the heart. Accordingly, the predominance of newborn HR accelerative responses would reflect the relative immaturity of vagal control over the heart and the shift to the more readily observed HR decelerative responses with age may reflect the development

of the relative vagal control over the heart rather than the development of the central mechanisms mediating orienting.

Unfortunately the hypothesis suggesting that the directionality of infant HR responses may be a function of developmental trends in the relative vagal control over the heart has not as yet been subjected to empirical test. Information regarding the development of the relative vagal control of the heart would have implications for the three points outlined above. Regarding the utility and interpretation of infant HR responses, if directionality is dependent upon the functional maturity of the vagus, then it may be that both accelerative and decelerative responses reflect orienting early in infancy. If this is the case, the direction of HR responses would not be a reliable index of orienting during the newborn period, and the CNS mechanisms mediating orienting may be independent of the status of the neural control of the heart during this period of development.

Second, the vagal control hypothesis points to the potential importance of an organismic variable in addition to stimulus and state factors for newborn HR decelerative responses, i.e., a certain level of vagal development may be a necessary condition for newborn HR decelerative responses. Although certain stimulus and state conditions may be important, the evidence to date suggests they are not sufficient. Thus, in contrast to Graham's view, whether or not HR decelerative responses are observed in the newborn may depend on interactions among the functional maturity of the vagus, stimulus, and state conditions.

Third, the apparently age-related trend in directionality may be dependent upon the development of the cardiovascular system and its

development may not coincide with the development of orienting. In addition to developmental differences, it is also possible that individual differences in the vagal control of the heart could partially account for the conflicting findings reported in the literature. For example, newborn HR response data usually has been reported in the form of curves averaged across subjects, and often across trials. Averaging across subjects may mask individual response patterns reflecting HR decelerative responses. A recent study by Brown, Leavitt, and Graham (1977) illustrates this point. They compared HR responses of 6- and 9-week-old infants to complex auditory stimuli under conditions "appropriate" for orienting (i.e., the infants were in an awake-alert state and the stimuli were of moderate intensity and had a slow rise time). The group results indicated the 6-week-old group did not show a significant cardiac response to the stimuli (neither acceleration or deceleration), whereas the older group showed a significant decelerative response. To test whether the lack of a significant cardiac response in the younger group may have been a consequence of averaging, Brown et al. tabulated directional HR responses according to the criterion of at least a peak change of 9 beats/min for at least 5 consecutive sec during the postonset period of a stimulus presentation. According to this criterion, they reported 7 of 24 infants of the 6-week group showed decelerations and 7 of 24 infants showed accelerations. In the 9-week group, 13 of 24 infants showed decelerations and 7 showed accelerative responses.

Although these data may be interpreted along the lines of the Graham model (i.e., the individual and developmental differences in

directional HR responses reflect individual and developmental differences in orienting), they are also in agreement with the alternative formulation that individual and/or developmental differences in the vagal control of the heart, perhaps independent of orienting, influences the directionality of the HR response. Accordingly, the HR acceleratory responses of the individual infants from both age groups might have resulted from a relative immaturity of vagal control of the heart; the HR deceleratory responses of the other infants may have reflected more mature vagal control. It is also tempting to speculate that since both the acceleratory and deceleratory responses were apparently time-locked to stimulus onset, the HR responsivity shown by these infants may have reflected orienting, independent of response directionality. Furthermore, the vagal control hypothesis would also seem to imply that increases in the number (or proportion) of infants exhibiting decelerative responses may be a manifestation of a more general ontogenetic trend of increasing vagal control with age. There is another possibility according to the Porges' continuity model, namely, individual and/or developmental differences in the relative vagal control of the heart may covary along with these types of differences in central mechanisms mediating orienting and attention. Of course until more precise information becomes available concerning the development of cardiac control mechanisms during infancy, all of these hypotheses are equally likely.

Summary

One unresolved question currently confronting developmental psychophysiologists is whether individual and/or developmental differences in infant HR response topographies are manifestations of psychologic

processes related to orienting and attention, or are due to differences in the cardiac response system itself (i.e., the neural control of the heart). The importance of the theoretical and methodological implications of this question is underscored by the widespread use of cardiac responses in contemporary infancy research. In view of the lack of successful prediction to date of the directionality of infant HR responses based on stimulus and state factors, it seems clear that without systematic study of at least the following two questions, answers to this controversy will not be found. First, during early infancy are there individual and/or developmental differences in the relative vagal control of the heart? Second, if so, are these differences related to infant HR response topographies?

It appears that only two studies in the infancy literature have been concerned with these questions and both studies were restricted to the newborn period (see Porges, et al., 1973; Porges, et al., 1974). Recall that in both of these studies relations were found between levels of HRV and subsequent HR response topographies. For the most part these findings were in the expected direction according to the vagal control hypothesis as advanced by Porges and his colleagues. However, not all of the findings were clearcut and based on the previous discussion concerning the merits of <u>patterns</u> versus <u>levels</u> of HRV as estimates of the neural control of the heart, these findings only can be taken as suggestive at best. Keeping in mind the previously cited data by Porges and his associates concerning the rational and application of spectral analysis techniques, it seems that methods are at hand which could be easily applied to these questions.

Although Porges has asserted there is a developmental increase in the relative vagal control over the heart, he has as yet to provide a review of the pertinent evidence underlying this claim. Since the overall aim of the present study is concerned with this question, the literature bearing on this issue will now be discussed.

Infant Cardiovascular Development

It should be stated at the outset of this section that a fair number of interacting variables influence cardiac functioning--particularily in terms of cardiac rates--including, but not limited to activity, posture, respiration, oxygenation, metabolic rate, and blood pressure. It also should be noted that there are few empirical data bearing directly on the question of the development of cardiac control mechanisms in the human. The complexity of this system combined with strong ethical reasons obviously makes definitive research in this area difficult at best. It is largely for these reasons that at this time generalizations regarding cardiovascular development must be somewhat vague and tentative. There is evidence of an indirect sort, however, upon which inferences have been drawn regarding the early development of the cardiovascular system.

If there is, as Porges hypothesized, an immaturity of the vagal control of the heart relative to sympathetic influences, then one question which comes to mind is: Does vagal innervation of the heart occur prenatally? Because of its clinical implications, much of the currently available evidence concerning cardiovascular functioning during the perinatal period is found in the biomedical literature. As several recent reviews of this literature have concluded, there is

ample evidence showing that the vagus has innervated the heart and is functional prior to birth, although estimates vary as to when during prenatal development this occurs (see Eichorn, 1970; Lemire, Loeser, Leech, & Alvord, 1975; Nelson, 1976; Papano, 1977). For example, Eichorn, Nelson, and Pappano have all reviewed evidence indicating that vagally mediated baroreceptor and chemoreceptor reflexes are functioning by birth although these homeostatic reflexes are "immature" when compared to those of the adult. One example of the type of evidence upon which inferences concerning vagal functioning have been made comes from the obstetrical literature. Obstetricians (via ultrasound, or electrodes placed on the maternal abdomen or fetal scalp) often monitor fetal cardiac activities during the course of labor and delivery as an assessment technique for evaluating the status of the fetal CNS and overall well-being (see Nelson, 1976; Paul & Hon, 1974; Porges, Note 2). Of interest here are the phasic changes in fetal HR as they have been related to changes in intrauterine pressure. Two classes of fetal HR decelerations have been identified (Nelson, 1976). In the first ("Type I dips"), fetal HR begins to decelerate with the onset of a uterine contraction and reaches its nadir within 5 to 15 sec and then returns to baseline. In the second ("Type II dips"), the fetal HR deceleration lags behind the uterine contraction by at least 18 sec and is more pronounced than the Type I dip. The Type I dip is thought to be vagally mediated since it is blocked with atropine (a cholinergic blocker) and is not viewed as a pathological sign. Type II dips are thought to reflect a state of fetal hypoxic distress. Since Type II dips cannot always be blocked with atropine, they are not

viewed as primarily vagally mediated. It is also interesting to note that prolonged episodes of hypoxia have been associated with (in addition to recurrent Type II dips) CNS depression, an overall rise in baseline cardiac rates, and loss of beat to beat HRV (Nelson, 1976; Petrie, 1978). (Postnatal clinical signs associated with these indices of severe hypoxic distress include, for example, meconium staining and fetal metabolic acidosis with a nonacidotic mother.)

Recent evidence in the obstetrical literature from studies on the effects of various drugs and anesthetics on fetal HR and HRV also suggests the vagus has innervated the heart and is functional by birth (for reviews see Bowes, Brackbill, Conway, & Steinschneider, 1970; Petrie, 1978). As in the adult, administration to the human fetus or newborn of atropine (and scopolamine) results in increased HR and reductions in beat to beat HRV. Moreover, nearly all of the intrapartum pharmacologic agents in current use readily and rapidly cross the placenta and enter fetal circulation. Many of the drugs become deposited in the fetal cerebral cortex, floor of the fourth ventricle (near the brainstem cardiorespiratory centers), and the liver because the ability of the fetal liver to metabolize these agents has yet to develop. It is of interest that a large number of drugs--including local and general anesthetics, narcotic analgesics, barbituates, tranquilizers, and alcohol--act upon the fetal nervous system resulting in CNS depression and, consequently, elevations in HR and reductions in HRV. (In addition to the effects of intrapartum medications on the fetal nervous system, several pharmacologic agents have also been found to bring about vagally mediated changes in fetal HR and HRV via

alterations in the frequency and amplitude of uterine contractions and maternal blood pressure.)

The reviews by Eichorn, Nelson, and Pappano also concluded that sympathetic innervation of the heart occurs sometime during the first few postnatal months. Therefore, in contrast Porges' hypothesis one might expect greater vagal influences, relative to sympathetic, on the newborn and neonatal heart. However, the human fetal and newborn heart is not only responsive to, but "supersensitive" to circulating catecholamines which suggests that despite an immature neuroeffector system, alpha- and beta-adrenergic receptors are intact and functioning prior to term (Eichorn, 1970; Nelson, 1976; Pappano, 1977). Studies with chronic animal preparations also have described developmental changes from early in the fetal period through adulthood in ANS control exerted on basal cardiac rates. For example in the lamb, Assali, Brinkman, Woods, Dandavino, and Nuwayhid (1977) examined and found developmental changes in both the absolute and relative amounts of control exerted by vagal and sympathetic branches of the ANS, with sympathetic influences (catecholaminergic) appearing earlier than vagal influences and predominating in the fetus. After term birth, vagal control emerged and became predominate with the greatest increases (i.e., influences on basal cardiac rates) observed during the first two postnatal weeks. In the human, Pollack (1977) has cited evidence that as early as the fourth prenatal month there is endogenous storage of catecholamines in the fetal heart (also see Pappano, 1977). Thus according to his previously cited model of cardiac pacemaking, there does appear to be an empirical basis for asserting, as he does, a catecholaminergic role

in newborn pacemaking. The literature suggests, then, both parasympathetic-vagal and sympathetic cardiac control mechanisms are present by birth although questions regarding the extent of their relative contributions in the human remains open.

If there is a postnatal increase in the relative vagal control of the heart, then at least two aspects of this control may be considered. First, regarding vagal tone as it refers to the functioning of the efferent vagus to the heart, there may be a maturation of the vagus itself in terms of more complete myelination and/or an increase in the diameter of these nerves. Either or both of these would lead to increased conduction velocities and, hence, more efficient vagal regulation. Unfortunately it appears that the precise developmental course of the morphological characteristics of the vagus in the human has not been described. Second, there may be a maturational increase in the higher CNS influences on the efferent vagus that could be observed in terms of an age-related trend of increasing respiratory sinus arrhythmia. This could be the result of maturation of medullary mechanisms or supramedullary influences on these mechanisms. Respiratory sinus arrhythmia (RSA), as measured by the difference between HR during inspiration and expiration, has been found to change with age with increases up until adolescence and leveling off thereafter (Reeve & DeBoer, 1960). Unfortunately the youngest age group represented in this study was 1 year. It has been that RSA is "rare" during infancy, though the data upon which this conclusion was drawn were not presented (see Kero, 1974). It thus appears that two important types of data that could clarify the development of vagal function are not currently

available.

However, one line of evidence that appears consistent with the hypothesized age-related increase in the relative vagal control is the overall course of cardiac rates. Since the vagus has an inhibitory effect on HR, one might expect at least a monotonic decline in HR with age. While it has long been known there is an overall decline in cardiac rates from birth to adulthood (e.g., see Eichorn, 1970), the precise developmental course of HR has yet to be thoroughly documented. Most of the recent infancy studies which have collected normative HR data have been restricted to the newborn period (e.g., see Kero, 1974). This literature suggests that immediately after birth (within minutes) there is a marked decline in HR which slowly begins to return to prenatal levels over the first few days (see Kero, 1974; Nelson, 1976). The reason for this postpartum decline is not entirely clear but there is some evidence suggesting it may be barosensory mediated (and hence, vagally mediated) since (a) there is a concomittant rise in systemic blood pressure, (b) the HR decline can be blocked with atropine, and (c) pre-term newborns also show the same pattern although their rates are higher than those of full-term infants. The evidence indicates that the HR increase following this immediate postpartum decline appears to continue gradually for the first month in full-term infants and somewhat longer in pre-term infants. Although this trend of increasing basal cardiac rates would not appear to be consistent with the hypothesized age-related trend of increasing vagal control, the literature indicates there are also hemodynamic, respiratory, metabolic, and electrolytic transistions which may also influence basal cardiac rates

during the cardiopulmonary conversion from fetal to postnatal life. Moreover, while recent research has found short-term effects of obstetrical medications on fetal and newborn cardiac rates and HRV, possible effects beyond the immediate newborn period have not been studied nor adequately controlled in the available normative HR research on the newborn period.

Beyond the newborn period the normative HR data available in the pediatric physiology and cardiology literatures presents a somewhat ambiguous picture of the postnatal course of HR (e.g., see Cassels & Ziegler, 1966; Eichorn, 1970; Krovetz, Gessner, & Schieber, 1969; Namin & Miller, 1966; Nelson, 1976). Of the published studies in this area, most have only reported range data and have not adequately reported the methods used to obtain the data (e.g., sample size, whether the data are cross sectional or longitudinal, whether bio-behavioral state was controlled, recording conditons, scoring techniques, and so forth), or have included such broad age ranges within groups so as to preclude meaningful comparisons either between or within studies. There is also a paucity of solid normative HR data available in the developmental psychophysiology literature. When these data have been reported in the latter literature, it has often consisted of means and some estimate of variability (between subjects) derived from the last second or complete beat prior to stimulation (e.g., see Graham, Clifton, & Hatton, 1968; Lipton, et al., 1966; Rewey, 1973).

There have been several recent short-term longitudinal studies on the developmental course of cardiac rates during early infancy that

appear to support the hypothesis of postnatal shifts in the relative vagal control of the heart. Lewis, Wilson, Ban, and Baumel (1970) reported normative HR data derived from EKGs recorded during 15-min rest periods for infants (n = 13) when they were 4-, 12-, 24-, 36-, and 56-weeks of age. Analyses of the data revealed a significant linear decline in HR from 4 through 56 weeks of age. The means (in beats per minute or bpm) for the respective ages were approximately: 152, 149, 146, 140, and 136 (see their Figure 3, p. 805; standard deviations were not published). A more recent series of short-term longitudinal studies reported by Harper and colleagues also lends support to the hypothesized postnatal shift in vagal influences. In these studies EKGs and respiration were recorded for an entire overnight session (12 hr) with biobehavioral state (i.e., quiet sleep, active sleep, waking, and a transitional state) coded for each minute of each session when the infants were 1 week, 1-, 2-, 3-, 4-, and 6-months old. Normative HR data (n = 8) were reported in the first published study (Harper, Hoppenbrouwers, Sterman, McGinty, and Hodgman, 1976) with the analyses revealing that HR was a function of age and state although the interaction between these factors was not significant. Mean HR (mean of the median HR for each minute sample) increased significantly from 1 week to 1 month, no significant change from 1 to 2 months, a significant decrease from 2 to 3 months, and no significant change thereafter (means and standard deviations for the respective ages in the awake state were: 163, 13.7; 167.4, 12.0; 167.1, 8.8; 152, 6.4; 152.1, 8.0; 149, 15.8 bpm). Regarding state, HR was also found to be the highest (at all ages) during the awake state, followed by, in descending order, transitional, active sleep, and quiet sleep states. It is also interesting

to note that while the developmental trend was the same for all states, (i.e., a significant cubic trend), Harper et al. also found that the differences in HR between states were markedly larger than differences as a function of age (e.g., at 3 months, mean HR for the quiet sleep state was 118.8 bpm whereas for the waking state 152 bpm).

In a subsequent study Harper, Leake, Hoppenbrouwers, Sterman, McGinty, and Hodgman (1978) compared the HR trends of the infants from the previous study (2 additional "normal" infants were included in this sample) with 10 infants at risk for sudden infant death syndrome (SIDS). The EKGs were collected as in the previous study and the analyses revealed developmental differences between the control and risk infants, particularily in the awake state. The interpretation offered by these authors was that the data indicated either a delayed maturation or impaired functioning of vagal control over the heart for the infants at risk for SIDS. The main differences between these groups were: (a) mean HR was significantly higher in the risk group at 3 months of age (162.6 vs 150.5 bpm) and (b) the risk group showed a significant decline in mean HR from 3 to 4 months of age whereas the control infants showed a significant decline from 2 to 3 months of age. Thus the overall developmental trend of the risk infants appears delayed but still quite similar to that of the normal infants.

Katona and Egbert (1978), in another short-term longitudinal study, compared the postnatal course of HR of normal fullterm and preterm infants during quiet sleep states. They found that the HR of the preterm infants was higher than that of the fullterm infants throughout the first six months with the greatest differences between the groups

occurring during the period of 10- to 14-weeks of age. HR means and standard deviations at 1-, 2-, 3-, 4-, and 6-months of age for the term infants were: 135, 3; 132, 3; 123, 3; 120, 3; and 112, 3 bpm. For the preterm infants at the same ages these figures were: 144, 5; 148, 7; 141, 5; 134, 5; and 118, 2 bpm. The agreement between these results for the fullterm infants and for the control infants in the Harper et al. (1976) study at the same ages for the quiet sleep state is quite striking (i.e., 138.6, 3.4; 129.7, 5.5; 118.3, 5.3; 118.4, 9; and 113.8, 6.2 bpm).

Summary

Despite small sample sizes, the postnatal course of cardiac rates depicted for normal fullterm infants in these studies are fairly consistent. As Harper et al. (1976) suggested, the discrepancy between the cubic trend they found and the linear trend reported by Lewis et al. (1970) may have been due to the inclusion of both waking and sleep states during the "resting" periods of the latter study. Nevertheless, all of these research groups suggested the overall decline in HR with age is due to the development of the vagal control of the heart. This interpretation is consistent with the finding by Reeve and DeBoer (1960) of an inverse relation between cardiac rates and RSA with increasing age. The above interpretation is also consistent with the data reviewed by Eichorn (1970) in which the greatest changes within the cardiac cycle with age were found in the T-P interval and not in the P-R interval. Of course the question of the development of RSA during the opening postnatal months of life has not been answered. Nevertheless, developmental changes in RSA during this age are suggested, indirectly of

course, by the Reeve and DeBoer (1960) study and by Kero (1974) as well as by the more recent finding that respiratory rates decline in a slightly different fashion than HR during the first six months (see Hoppenbrouwers, Harper, Hodgman, Sterman, and McGinty, 1978). In a report of normative respiratory rates for the infants in the Harper et al. (1976) study, Hoppenbrouwers et al. found that like HR, respiratory rates were significantly related to age and state. However, unlike HR, the interaction between age and state was significant with respiratory rates showing the same postnatal trend as HR during the waking state but a significant linear decline from 1- to 3-months and no significant change thereafter in the sleeping states. It was suggested that these different developmental trends could have been due to either the development of CNS control mechanisms or different maturation rates for vagal and sympathetic influences on the heart.

General Summary

The general purpose of the present study was to explore the problem of developmental differences in cardiac functioning during the first six months of life. There are two major aspects of this problem that are of interest. The first has to do with the question of the development of the neural control of the heart *per se*. The second has to do with the general question of cardiac responsivity as a biological concomittent of orienting and sustained attention. As should be evident from this chapter the literatures bearing on these problems represent two relatively independent research domains.

Regarding the first aspect of the above problem, the studies just reviewed strongly suggest developmental changes in cardiac functioning

during the early postnatal period in the human. On the other hand, while the hypothesis has been advanced by several research groups, it seems clear that it is not yet known whether or to what extent these changes are a function of the development of the relative vagal control over the heart or other variables during the transition from fetal to extrauterine life. Despite the paucity of data, the data that do exist suggest that the question of individual and developmental differences in the neural control of the heart during the opening months of postnatal life are reasonable questions worthy of being pursued. Moreover, in view of the conflicting literature on infant cardiac responsivity, the need for additional developmental studies on these questions is apparent. Thus, keeping in mind the previously cited data and rationale underlying the derivation of the C_w function by Porges and colleagues, these techniques seem particularily suited for exploring this problem. Consequently the present study was designed to obtain cross-sectional estimates of the developmental sequence of C_w for infants at 1-, 3.5-, and 6-months of age. Since there is comparatively little research on the development of this system, precise predictions are difficult and the present study must be essentially descriptive in nature. Therefore, the first part of the present study was designed to explore the following specific questions: (1) what is the developmental course of the relative degree of RSA (C_w) during the first six months; (2) what, if any, are the relationships of C_w to the developmental sequences of HR, HRV, and respiratory variables?

The purpose of the second part of the present study was to examine something of the potential behavioral significance of the ontogeny of

the relative parasympathetic-vagal control of the heart. As the infancy studies reviewed in this chapter make all too painfully clear, it is difficult to reliably predict the outcome (i.e., directionality and/or magnitude of cardiac responses) of presenting discrete stimuli to human infants on the basis of age, stimulus, and biobehavioral state variables. Moreover, given the conflicting findings in the infancy literature and the present state of knowledge of early cardiac functioning, it simply does not appear valid to assert that since HR decelerative responses have been observed in some human newborns, this response system is sufficiently mature so that whether or not a decelerative response is observed depends on the infant's psychological response to the stimulation (cf. Berg & Berg, Note 1). On the other hand, although it also seems premature to dismiss this hypothesis completely, the data that do exist certainly suggest it may be questioned. Therefore, the primary concern here is whether individual and/or developmental differences in infant cardiac responsivity are due to these types of differences in orienting and sustained attention or are manifestations of these types of differences in the cardiac response system itself. Thus, the second part of this study was designed to optimize orienting to stimulus change (onset and offset) as well as states of sustained attention to visual stimuli by the infants in the above age groups. The study was designed to evaluate both behavioral (visual fixation) and physiologic (cardiac and respiratory) response parameters that have been proposed as indices of orienting and sustained attention as well as whether these response measures relate to the measures of the relative vagal control of the heart.

Earlier in this chapter the distinction between orienting and sustained attention made by Porges was reviewed. The principle difference between these types of attention was that the OR is thought to be a stimulus determined reflex ("involuntary") whereas sustained attention refers to a voluntary or more internally-driven cognitively mediated state of preparedness to receive or process information. As such, it is assumed that sustained attention is not information processing itself, but rather, a change of state of the CNS underlying information processing. Thus, as opposed to the OR, states of sustained attention are not necessarily limited to the presence of discrete stimuli but may occur in the absence of such stimulation (e.g., in vigilance tasks or RT tasks). Other researchers have also discussed the distinction between these types of attention (e.g., see Fitzgerald, et al., 1977; Kagan & Lewis, 1965; Mackworth, 1976; van Hover, 1974). Unfortunately, with the exception of the two previously cited studies with newborns by Porges and his colleagues, research on sustained attention in young human infants has not been pursued. Thus it is not yet known whether these types of attention follow the same or different developmental trends. From a larger developmental perspective, although much of the infancy research has concentrated on the OR as well as using paradigms using the OR to provide information about the development of the young infant's sensory, perceptual, and learning capacities, it would seem that any adequate developmental theory of infant cognitive development may eventually have to also encompass the development of sustained attention. Obviously this brings to the fore the question of reliable and valid dependent measures of the construct of sustained attention.

In light of these considerations, presenting visual stimuli which have been found to elicit visual fixation responses (see Fantz, et al., 1975; Cohen & Gelber, 1975; Horowitz, 1975) for a duration similar to that of the PI of warned reaction time studies should allow a test of the hypothesis by Porges concerning the HR, HRV, and RAV measures of sustained attention. Visual fixation responses were also included and should provide information on the general question of the utility of the above described physiologic measures of orienting and sustained attention, i.e., it seems reasonable to assume that visual fixation responses to visual stimuli may be considered criterion variables for evaluating whether changes in peripheral physiologic activities are related to the extent to which infants pay attention to the stimuli. Therefore, the second part of the present study was designed to explore the following questions: (1) what are the developmental sequences of the visual fixation, cardiac, and respiratory measures thought to reflect orienting and sustained attention; (2) what, if any, are the relationships among these measures; (3) what, if any, are the relationships between the measures of the neural control of the heart and the behavioral and physiologic measures of orienting and sustained attention?

Hypotheses

Based upon the preceding literature review, the following hypotheses were developed.

Respiratory Sinus Arrhythmia

1. If there is a shift with age in the predominance of the relative vagal control of the heart, then

a. there should be at least a significant monotonic increase in C_w from 1- to 6-months of age,

b. heart period (HP or the reciprocal of HR) and HRV should show a significant moderate to high positive correlation with C_w, and

c. respiratory frequency should show a significant inverse relationship to C_w.

Orienting and Sustained Attention

2. If there is a developmental increase in the infant's abilities involved in orienting and sustained attention over the first six postnatal months, then the infants should show, with increasing age,

a. significant increases in the lengths of visual fixations and total fixation time,

b. significant increases in the magnitude of HR decelerations to stimulus onset and offset,

c. significant increases in the suppression of HRV and RAV during the stimulus presentations, and

d. significant moderate to high intercorrelations among the above measures of orienting and sustained attention.

C_w and Attention

3. If C_w is related to the processes of orienting and sustained attention, then the above measures of these processes should vary significantly as a function of the levels of C_w with greater levels of C_w associated with superior levels of the above dependent measures.

CHAPTER III

METHOD

Subjects

The final subject pool consisted of 36 infants with 12 infants (six males and six females) in each of the 1-, 3.5-, and 6-month old age groups. Means and standard deviations of chronological age (in days) were as follows: $\underline{M}$ = 34, $\underline{SD}$ = 2.5; $\underline{M}$ = 108.4, $\underline{SD}$ = 3.3; $\underline{M}$ = 186.5, $\underline{SD}$ = 7.1. Only fullterm infants with complete sessions, not on any medications, and no reported (parental reports, see Appendix B) histories of perinatal complications (fetal, labor, delivery), eye disorders, neurological disorders, or chronic illnesses were included in the final sample. An additional 22 infants were tested but not included in the final sample: Twelve of these infants were excluded because of incomplete sessions due to inappropriate states (e.g., crying, sleeping), five because of perinatal complications, and five due to equipment failure or experimental error. As part of another study, some of the 1-month old infants were tested again in the laboratory and their parents received financial remuneration for each visit to the laboratory.

Names of families of potential subjects were obtained from area newspaper announcements and birth records. A letter describing the study and a permission-to-contact-them-card were sent to these families (see Appendix B). All families returning a signed permission card were contacted by phone to answer questions about the study and to arrange

an appointment for their participation. Appointments for testing were made for a time when parents expected their infant to be most alert and cooperative.

Apparatus

All stimulus exposures, visual fixation responses, and physiologic activities were recorded on a Grass Model 7 polygraph and an eight-channel Vetter FM tape recorder. The polygraph, tape recorder, and electronic timing equipment were located in a room adjacent to the room containing the sound attenuated experimental booth. Infants were in the booth for the baseline recordings of physiological measures and the visual stimulus presentations. Ambient noise level in the booth was approximately 55 db re .2000 dynes/cm^2 and the room temperature was 72°F. A 25-watt overhead light--which remained on throughout each session--provided a low level of room illumination within the booth.

Visual stimuli were presented with a Kodak Carousal 700 projector. Each stimulus was rear-projected onto a 48.5 by 41.5 cm screen. The screen was set into one wall of the booth and was located approximately 1 m directly in front of the infant. Presentation and duration of stimulus exposures were controlled with a series of electronic timers.

The stimuli were five colored slides and one black and white slide. Stimuli were selected on the basis of previous pilot work and from a review of the infant visual fixation literature (e.g., see Cohen & Gelber, 1975; Fantz, et al., 1975; Friedman, 1975; Lewis, 1974; Horowitz, 1975; Sigman & Parmelee, 1974; Tennes et al., 1972). The colored slides consisted of a frontal view of a baby's face, a frontal view of an adult female face, a red and white checkerboard (6 x 6), a sunset scene, and

a landscape photograph of mountains and trees. The black and white slide pictured four white triangles on a black background. The projected size of the stimuli on the rear-projection screen was 38 by 38 cm. Although fairly large, this size was employed to avoid any possible acuity differences across age groups. Stimulus slides were arranged within the caraousal with a black slide before and after each stimulus slide. A small hole in each of the black slides permitted the projection of a small white dot (2 cm) onto the center of the screen which was used to help keep the infant's head oriented, more or less, toward the screen between trials.

Dependent Variables

Heart Period

For recording the cardiac data three Grass Ag cup electrodes (contact area .12 cm^2) were placed 1-inch above each nipple and the naval after each of these recording sites were first cleaned with 70% ethyl alchohol prep pads. Electrodes were filled with Beckman Offner electrod paste and attached by double adhesive collors and surgical tape. Leads from the electrodes were connected via a double-shielded cable to one channel of the polygraph and from this channel to one channel of the tape recorder.

Respiration

Respiration was recorded by placing a small Phipps-Bird pneumograph (rubber bellows) around the infant's thorax at the level of the diaphragm. Inspiration and expiration produced pressure changes within the bellows. These pressure changes were transduced into a voltage by

a Grass Model PT5A volumetric transducer which was connected to one channel of the polygraph and from the polygraph to the tape recorder.

Visual Fixation

A traditional corneal reflection technique was used to monitor visual fixation responses (Fantz, et al., 1975). From outside the booth an observer watched one of the subject's eyes through a small (.64 cm in diameter) peephole and presses a silent button whenever and for as long as a stimulus was fixated. Peepholes were located off to each side or underneath the rear-projection screen. Whenever the observer pressed the fixation button, a 1.5 volt DC signal was supplied to one channel of the polygraph and then to one channel of the tape recorder for as long as the button was pressed. Light from a stimulus superimposed over an infant's pupil was the criterion used for determining fixation responses.

Interobserver reliability. Three individuals, previously exper experienced with corneal reflection techniques, served as observers for monitoring fixations. Only one observer per infant was used except when interobserver reliability checks were made. Prior to this study observers were retrained until they obtained interobserver agreement scores of at least 90. These scores were determined by subdividing the total time of the stimulus exposures (six 12 sec exposures per subject) into 500 msec intervals. The total number of intervals of agreement was divided by the total number of intervals per subject (144) and then multiplied by 100. Paired observations of eight infants (from the final subject pool) during the course of the study provided the data for interobserver reliability checks. Mean interobserver

agreement was 89.2 and ranged from 75 to 95.8.

Procedure

Infants were accompanied to the laboratory by at least one parent and one parent remained with the infant at all times. Upon arrival they were shown the laboratory rooms and the general purpose and procedures of the study were again explained. After the parent(s) had read and signed the permission form, completed background and follow-up information forms (see Appendix B), the infant was prepared for the recording of the physiologic variables. Following this preparation the infant was taken by a parent to the experimental booth and placed in a portable infant seat (a commercial infant seat) which rested upon a short table which was positioned directly in front and at the bottem of the rear-projection screen. The seat was in a semireclining position so that the infant was half lying and half sitting down. The infant was then left facing the parent, who sat in a chair at the rear of the booth, for a five minute adaptation period. During this period polygraph and tape recorder checks were run to ensure that physiologic activities were being properly recorded. The parent was able to communicate with the experimenter in the polygraph room at all times via a Fanon Intercom device.

From this point on the general procedure followed that shown in Table 1. As indicated in Table 1, EKGs and respiration were continuously recorded before the first trial and after the last trial until 60 sec pre- and post-baseline periods with the infant in a quiet-awake state were obtained. Following the pre-baseline period the infant was repositioned so that he/she faced the screen for the visual stimulus

Table 1

Experimental Design Used to Study the Development of Respiratory Sinus Arrhythmia and Visual Attention in Young Human Infants

Group		Pre-Baseline	Visual Stimulus Trials	Post-Baseline
Age	Sex			
1 month (n = 12)	Males (n = 6)	60 sec	6 Stimuli	60 sec
	Females (n = 6)	60 sec	6 Stimuli	60 sec
3.5 months (n = 12)	Males (n = 6)	60 sec	6 Stimuli	60 sec
	Females (n = 6)	60 sec	6 Stimuli	60 sec
6 months (n = 12)	Males (n = 6)	60 sec	6 Stimuli	60 sec
	Females (n = 6)	60 sec	6 Stimuli	60 sec

presentations. The parent remained in the booth behind and out of the infant's visual field. During this phase of the study the visual stimuli were presented in six discrete trials and only when the infant was in a quiet-awake state. When a change in state occurred, testing was halted and the parent was asked to try to quiet the crying or fussing infant, or to awaken a sleeping infant. Three infants could not be maintained in a quiet-awake state while they remained in the infant seat and consequently sat in their mother's lap for the study. On those occasions when an infant failed to return to the appropriate state, the session was terminated and the parents thanked and assured their infant's reaction was not unusual.

The duration of each stimulus exposure was 12 sec. The minimum interstimulus interval (ISI)--time from stimulus offset to onset--varied randomly among 20, 25, and 30 sec intervals. However, each stimulus was presented only when the infant's head was oriented toward the screen. This was achieved by the observer pressing a hand held button whenever the infant's head was facing the screen and in a quiet-awake state. Thus, when the ISI had elapsed on the timer and the infant was oriented toward the screen the slide projector advanced and presented the next stimulus.

Infants within each age and sex subgroup were randomly assigned one of six stimulus sequences from a 6 x 6 Latin square. Each stimulus was given a number and then a randomly selected Latin square determined the six sequences (one sequence per row). Occasionally an infant did not fixate a stimulus when it was presented. When this occurred the stimulus was presented again after the last trial. Two infants in the

final sample (one 3.5- and one 6-month old) had one stimulus repeated. Following the last stimulus trial the 60 sec post-baseline recording of EKG and respiration was obtained.

Data Scoring

Cardiac and respiration data were processed by playing the FM (analog) tapes into a PDP 11/10 computer in Dr. Porges laboratory at the University of Illinois at Champaign-Urbana.

Baselines

Heart periods (HP) were timed beat-by-beat to the nearest msec and respiration amplitude digitized 8 times per sec for the two 60 sec baseline samples per subject. To meet the assumption of spectral analyses that the data represent events that are equally spaced in time, a computer program established 125 msec windows and weighted mean HP for each successive window was calculated. The algorithm in this program computed the weighted mean HP as the sum of the HP(s) that occupied or partially occupied the window multiplied by the proportion of the window that it occupied. Mean HP and the variance of the HPs (HPV) for each baseline were then derived from these data when they were submitted to the spectral analysis program.

Spectral analyses. Spectral and cross-spectral analyses were then performed on the baseline cardiac and respiration data for each infant as follows: First, in order to meet the assumption of stationarity, linear trends (slow shifts) were removed by calculating successive difference scores. Spectral analyses were then calculated. Next, the primary respiratory frequency band was selected for each spectra of

respiration. The primary respiratory frequency band was defined as the frequency band of each spectrum that contained the respiratory frequency with the greatest power-density score (amplitude) as the midpoint and included four frequency components above and below this peak frequency. Cross-spectral analyses were then calculated on respiration and HP data within this frequency band and thus defined baseline-by-baseline for each infant. The following dependent variables were derived from the spectral and cross spectral analyses: (a) C_w or the percentage of RSA, (b) vagal tone defined as the total amount of HPV (shared and not shared with respiration) within the primary respiratory frequency band, (c) the absolute amount of RSA (ARSA) or the amount of HPV shared with respiration within the primary respiratory frequency band, (d) the peak respiratory frequency or the respiration frequency with the greatest power density score, and (e) the peak HP frequency.

All of these dependent variables as well as the mean HP and HPV per baseline were submitted to separate analyses of variance. The experimental design permitted the analyses of two between-subjects factors (age and sex) and one within-subjects factor (pre- vs post-baseline) as well as all possible interactions among these factors.

Visual Stimulus Trials

HP, respiration, and visual fixations were scored for each trial. As illustrated in Figure 2, each trial was divided into four subperiods: A Pre Period, consisting of the last 6 sec prior to stimulus onset; an Onset Period, consisting of the first 6 sec immediately following stimulus onset; a Tonic Period, consisting of the final 6 sec of a stimulus exposure; and an Offset Period consisting of the first 6 sec

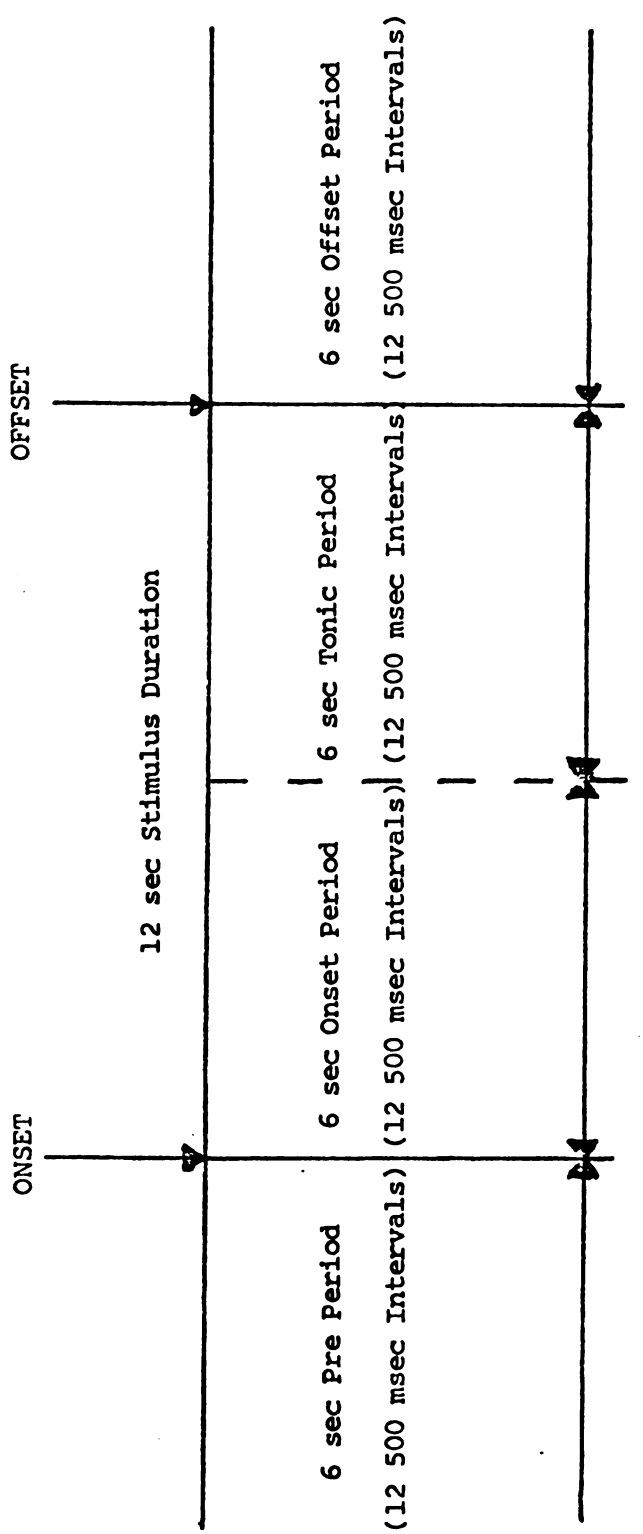

Figure 2. Illustration of visual stimulus presentation for a single trial and the four subperiods of a trial used in the data analyses.

immediately following stimulus offset. For the HP analyses, each of the four subperiods of a trial were divided into 12 500-msec intervals.

Heart period. Beat-by-beat HP was timed to the nearest msec and subsequently converted into weighted mean HP per 500 msec interval for each of the four subperiods of a trial. Three measures of HP were then derived: HPC/interval, mean HP/period, and HPV/period. For the interval analyses, HPC/interval difference scores were calculated as the difference between the HP score for the last 500 msec interval of the Pre Period and the HP score for each of the 500 msec intervals through the next three periods (18 sec) of a trial (Onset, Tonic, and Offset). For the period analyses, mean HP was the mean of the HP scores within each of the four subperiods of a trial and, similarily, HPV was the variance of the HP scores within each of the four subperiods of a trial.

Respiration amplitude variability (RAV). Respiration amplitude was digitized 8 times per sec for each trial. RAV was calculated as the variance of the amplitude scores within each of the four periods within a trial.

Visual fixation. Within each 12 sec stimulus exposure, four measures of visual fixation responses were derived: total fixation time, first fixation time, mean fixation time, and number of fixations. The distance between the onset and offset of each fixation (on the polygraph record) was measured in mm to the nearest mm (.1 sec). This distance was then divided by the polygraph paper speed (10 mm/sec) to yield the time in sec for each fixation. Total fixation time consisted of the sum of all fixation times per stimulus. First fixation time was the time of the first fixation of a stimulus. Mean fixation time was

the total fixation time per stimulus divided by the number of fixations per stimulus. Finally, the number of fixations was scored as the fixations per stimulus exposure.

Thus, from the visual stimulus trials the following dependent variables were submitted to separate analyses of variance: HPC/interval, mean HP/period, HPV/period, RAV/period, total fixation time, first fixation time, mean fixation time, and number of fixations. For the visual fixation measures, the experimental design permitted the analyses of two between-subjects variables (age and sex) and one within-subjects variable (stimulus) and all possible interactions among these variables. For the analyses of mean HP/period, HPV/period, and RAV/period an additional within-subjects variable, period, was analyzed. For the HPC/interval scores another within-subjects factor, interval, was incluced.

CHAPTER IV

RESULTS

Separate analyses of variance were computed for each of the dependent variables described in the previous chapter. Appendix C contains the analyses of variance summary tables. When significant interaction effects occurred, simple analyses of variance were performed on the conditions for the interacting variables. Where significant mean differences were found by either procedure, Duncan multiple comparisons were computed ($p = .05$). Finally, Pearson product moment correlations were computed among the dependent variables.

Baseline Cardiac and Respiratory Measures

Heart Period

Analysis of variance for the baseline levels of mean HP yielded a significant main effect of age (F (2, 30) = 3.49, $p < .05$). Figure 3 illustrates this age trend and shows a marked increase in mean HP (a decrease in HR) from the 1- to 3.5-month age periods which leveled off thereafter. Duncan tests indicated the mean HP for the 1 month group was significantly shorter than either of the two older age groups and that the mean HPs for the two oldest groups were not significantly different. There were no other significant main effects or interactions.

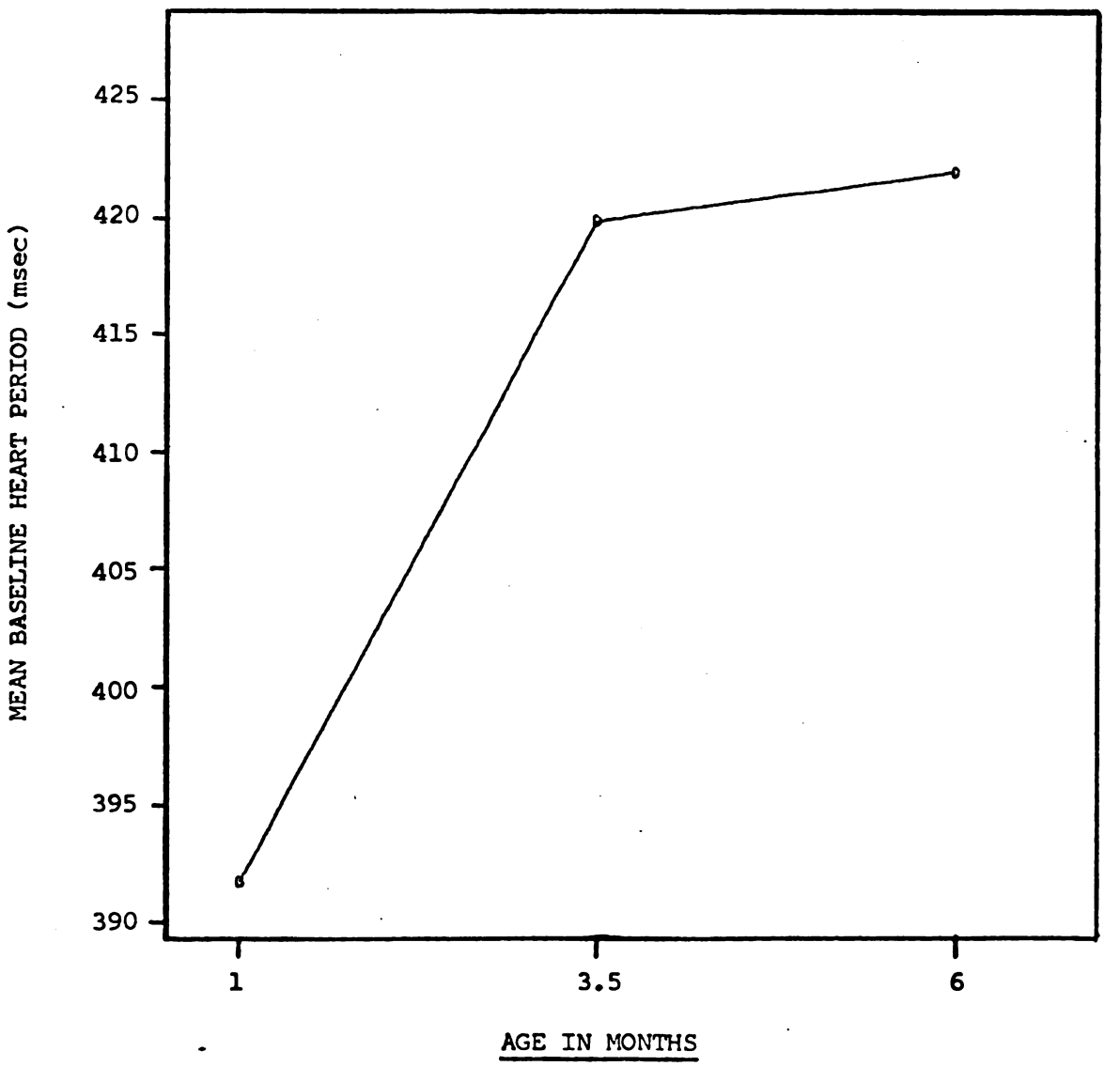

Figure 3. Mean baseline heart period as a function of age.

Heart Period Variance

The ANOVA for the baseline levels of ln HPV did not reveal any significant differences as a function of age, sex, baseline, nor were any of the interactions among these variables significant.

Measures Derived from the Spectral Analyses

Percentage of respiratory sinus arrhytmia. Table 2 shows the mean values of C_w for each of the age and sex subgroups. The ANOVA indicated there were no significant differences in C_w as a function of age, sex, baseline, nor were any of the interactions significant.

Amount of respiratory sinus arrhythmia. Although the ANOVA on C_w failed to reveal a significant age-related trend for the the percentage of RSA the possibility remains of age-related increases in the absolute amount of HPV that is shared (coherent) with respiration. Therefore, as a test of this possibility, an ANOVA was performed on the cross spectral analysis estimate of the ln amount of HPV shared with respiration (the numerator of the C_w equation, see Appendix A). The ANOVA yielded a significant main effect of age (F (2, 30) = 5.26, p = .011) and no other significant main effects or interactions. The top panel of Figure 4 illustrates this age trend. Duncan tests indicated the mean amount of RSA for the 1 month old group was significantly less than that for either of the two older groups and that the two oldest groups did not differ significantly on this measure.

Vagal tone. Recall that the measure used in this study as an estimate of peripheral vagal tone to the heart was the ln of the total amount of HPV within the primary respiratory band (i.e., the denominator of the C_w equation, see Appendix A). Although the ANOVA on this

Table 2

Mean Weighted-Coherence Scores[a]

	Age in Months		
	1	3.5	6
Males	.240 (.16)	.143 (.10)	.163 (.07)
Females	.199 (.13)	.179 (.11)	.216 (.10)
Group	.215 (.14)	.161 (.11)	.199 (.09)

[a]Numbers in parentheses indicate SDs.

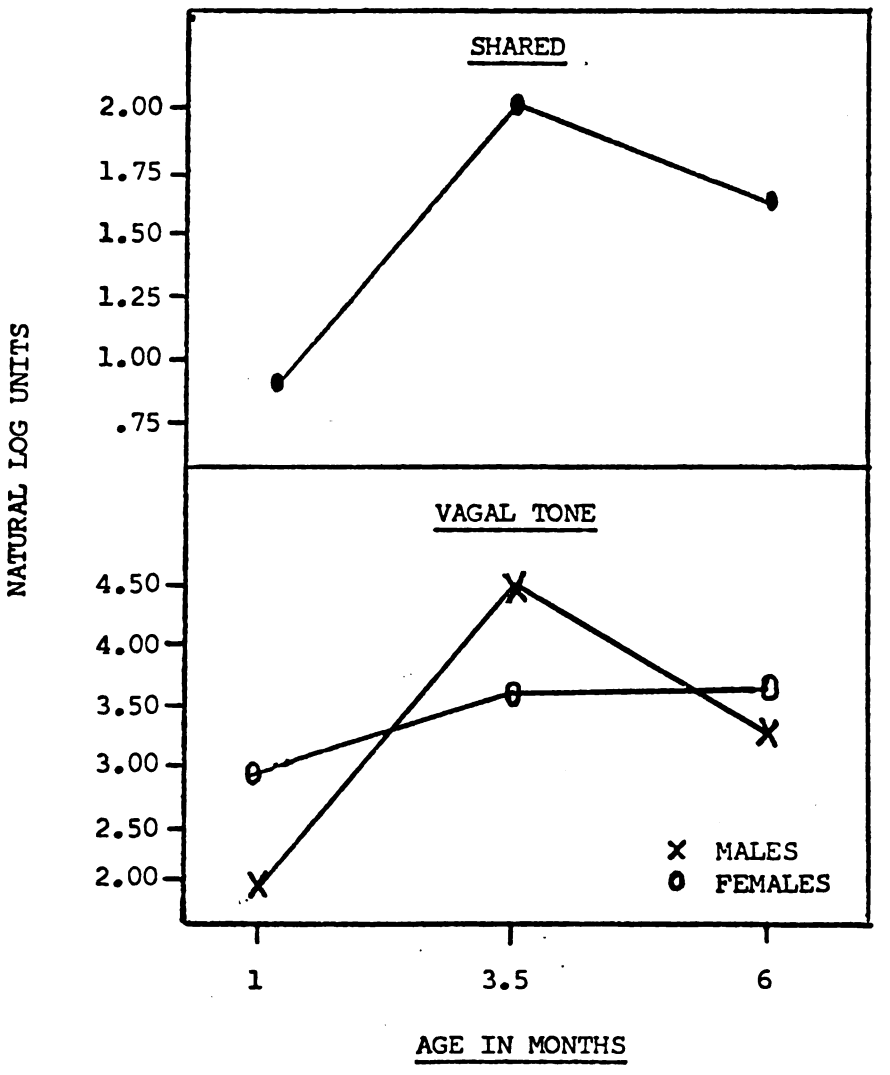

Figure 4. Mean changes in the absolute amount of respiratory sinus arrhythmia as a function of age (upper panel) and vagal tone as a function of age and sex (lower panel).

measure yielded a significant main effect of age (F (2, 30) = 10.93, p = .0003), the interaction of age x sex was also signficant, F (2, 30) = 3.78, p = .034. There were no other significant main effects or interactions. The lower panel of Figure 4 illustrates the interaction of age x sex for this dependent variable. Simple main effects tests indicated this interaction resulted from significant age differences in this estimate of vagal tone for the males (F (2, 30) = 14.12, $p <$ .01); there were no further significant simple main effects of age or sex. Duncan tests revealed that for the males, this estimate of vagal tone for the two oldest age groups was significantly greater than that for the 1 month old males. The means for this measure for the 3.5- and 6-month old males were not significantly different.

Peak respiratory frequency. Figure 5 illustrates the age-related decline in the baseline mean of the peak respiratory frequency. The ANOVA on this measure yielded a significant main effect of age (F (2, 30) = 3.81, p = .034) and no other significant main effects or interactions. Duncan tests indicated the mean peak respiratory frequency for the 1 month group was significantly greater than that for the 6 month group. No other mean differences were significant.

Peak heart period frequency. The ANOVA on the baseline peak HP frequency indicated there were no significant differences in this measure as a function of age, sex, baseline, nor were any of the interactions significant. (The main effect of age approached significance, F (2, 30) = 3.26, p = .052.)

Correlational Analyses

Pearson product moment correlations were calculated (for all

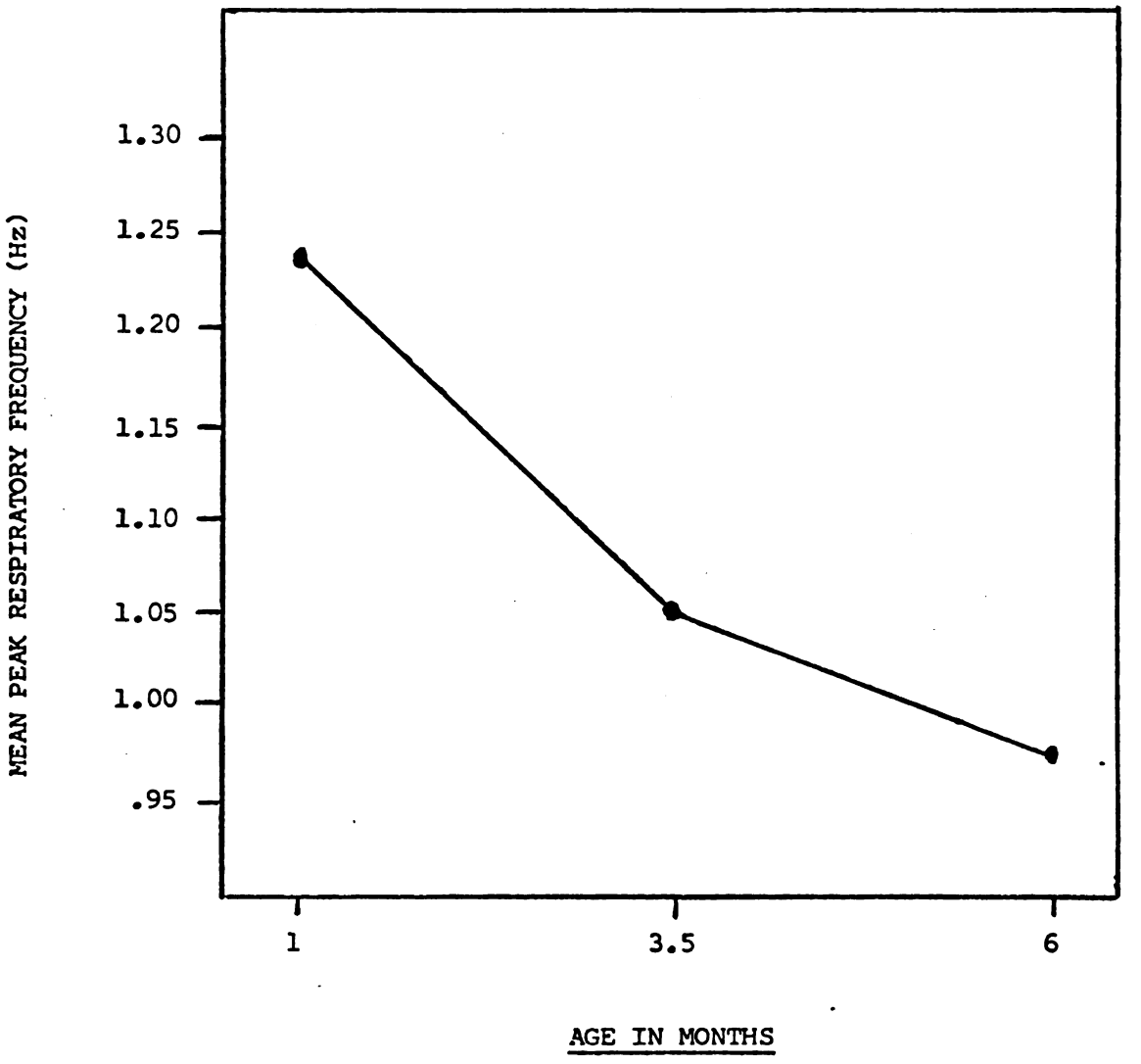

Figure 5. Mean baseline peak respiratory frequency as a function of age.

subjects combined) between the physiologic measures within each baseline sample to examine their relationships (see Table 3). Unexpectedly, C_w was not found to be correlated with mean HP (MHP), nor was it consistently correlated with the estimate of vagal tone or HPV. On the other hand, C_w was positively correlated with the amount of RSA in the pre-baseline sample and approached significance in the post-baseline. Amount of RSA was also positively correlated with the estimate of vagal tone as well as positively correlated with mean HP and HPV in the pre-baseline sample and approached significance with these measures in the post-baseline. In addition to the correlation with the amount of RSA, the estimate of vagal tone was positively correlated with mean HP and HPV in the pre-baseline and in the same direction in the post-baseline. There was also an inverse relation between the peak respiratory frequency and mean HP but only the post-baseline correlation was significant. Finally, peak HP frequency was negatively correlated with mean HP in both baseline samples and negatively correlated with HPV in only the post-baseline.

Summary of Baseline Analyses

The hypotheses of a developmental increase in the relative vagal influences on the heart during the first six months of life as estimated by the Porges' C_w function was clearly not supported. On the other hand, it was found that the amount of RSA did show a significant age-related trend suggesting increasing vagal influences over the heart with age. An age-related trend of increasing peripheral vagal tone with increasing age was also found. However, there was a significant sex difference in this latter trend: Both sexes showed mean increases

Table 3

Pearson Product Moment Correlations for the Baseline Physiologic Measures

	HPV	C_w	ARSA	VT	PRF	PHPF
			PRE-BASELINE			
MHP	.51*	-.02	.38*	.48*	-.27	-.45*
HPV		-.08	.07	.42*	-.04	.03
C_w			.55*	.04	.38*	-.05
ARSA				.63*	.02	.06
VT					-.09	.13
PRF						.36*
			POST-BASELINE			
MHP	.41*	-.09	.36*	.24	-.41*	-.37*
HPV		-.43*	.28	.31	-.03	-.40*
C_w			.30	-.31	.07	-.23
ARSA				.64*	.02	.03
VT					.16	.17
PRF						.41*

*$p < .05$

Abbreviations: MHP, mean heart period; HPV, heart period variance; C_w, weighted coherence; ARSA, amount of respiratory sinus arrhythmia; VT, vagal tone; PRF, peak respiratory frequency; PHPF, peak heart period frequency.

over the first six months but this trend was only significant for the male infants. The apparently parallel age-related trends in mean HP, amount of RSA, and peripheral vagal tone appear consistent with the hypothesis of developmental changes in the vagal control of the heart during early human infancy. In addition, the age-related monotonic decline in the peak respiratory frequency is consistent with the above developmental trends.

The significant intercorrelations among these parameters of cardiac-respiratory functioning, although in the low to moderate range, lend additional support to this hypothesis in that although two distributions may have the same mean and variance, it does not necessarily follow that they will be correlated. These significant intercorrelations also suggest that individual differences in these parameters may be consistent with the observed developmental differences. Thus, although C_w was not correlated with mean levels of HP, mean levels of HP were positively correlated with the other two estimates of vagal control over the heart. Moreover, higher levels of peripheral vagal tone were associated with higher amounts of RSA. Finally, the negative correlations of peak respiratory frequency and peak HP frequency with mean HP were also consistent with the overall developmental hypothesis that higher respiratory and HP frequencies were associated with shorter mean heart periods (faster HRs).

Visual Fixation and Physiologic Measures of Attention

Visual Fixation Performance

Total fixation time. The ANOVA on the total amount of time the infants fixated each stimulus yielded a significant main effect of

stimulus, F (5, 150) = 6.01, $p < .01$. However this effect was qualified by a significant interaction of age x stimulus, F (10, 150) = 2.24, $p < .02$. Means for this interaction are shown in Table 4. The simple effects ANOVA for these data indicated there was only on significant simple main effect of age and this was for the landscape stimulus, F (2, 180) = 3.49, $p < .05$. Duncan tests indicated that for this stimulus mean total fixation time for the 3.5 month group was significantly greater than that for the 1- and 6-month groups. The simple effects ANOVA for this interaction also revealed that there were significant differences in mean total fixation time among the stimuli by the 3.5- (F (5, 150) = 3.53, $p < .01$) and 6-month groups (F (5, 150) = 5.40, $p < .01$) but not by the 1 month group. For the 3.5 month group, Duncan tests indicated four (out of 12) significant differences in mean total fixation time: sunset> baby, woman, landscape; triangles <landscape. For the 6-month group, Duncan tests revealed seven significant differences in mean total fixation time to the stimuli: sunset <landscape, checkerboard, woman, baby; baby> triangles, landscape, checkerboard.

Mean fixation time. The ANOVA on the mean fixation time per stimulus yielded a significant interaction of age x stimulus (F (10, 150) = 2.03, $p = .026$) and no other significant main effects or interactions. Means for this interaction are shown in Table 5. The simple effects ANOVA for this interaction yielded simple age effects for the landscape (F (2, 180) = 3.80, $p < .05$) and checkerboard stimuli (F (2, 180) = 4.50, $p < .05$) as well as significant simple effects of stimulus for the 1-month group (F (5, 150) = 2.28, $p < .05$) and 3.5-month group (F (5, 150) = 2.30, $p < .05$). Subsequent Duncan tests revealed that for the

Table 4

Mean Total Fixation Time Scores (sec) from the Interaction of Age x Stimulus

	Stimulus					
Age (mo.)	Baby[a]	Woman	Landscape	Sunset	Checkerboard	Triangles
1	8.15 (4.21)	6.90 (3.62)	7.40 (4.23)	7.07 (4.86)	9.46 (2.92)	8.41 (3.63)
3.5	9.04 (2.69)	9.60 (1.80)	9.93 (2.14)	6.18 (4.25)	8.31 (3.49)	7.43 (3.23)
6	9.50 (2.17)	7.83 (2.10)	6.62 (2.47)	4.28 (2.58)	7.52 (1.76)	6.05 (2.71)

Note Maximum Score = 12.0

[a]Numbers in parentheses indicate SDs.

Table 5

Mean Fixation Time Scores (sec) from the Interaction of Age x Stimulus

Age (mo.)	Stimulus					
	Baby[a]	Woman	Landscape	Sunset	Checkerboard	Triangles
1	6.66 (4.64)	3.80 (3.25)	5.04 (4.45)	5.19 (4.54)	7.77 (3.90)	6.40 (4.80)
3.5	6.42 (3.74)	7.17 (3.59)	7.42 (4.29)	4.31 (4.07)	5.89 (4.87)	4.08 (3.37)
6	5.68 (3.50)	5.06 (3.15)	3.18 (1.83)	2.40 (2.35)	3.17 (2.14)	3.00 (2.83)

Note Maximum Score = 12.0

[a]Numbers in parentheses indicate SDs.

landscape stimulus, the mean fixation time for the 3.5-month group was significantly longer than for the 6-month group but no other significant age comparisons for this stimulus. For the checkerboard stimulus, Duncan tests indicated the mean fixation time for the 1-month group was significantly longer than that for the 6-month group and no other significant age comparisons. Duncan comparisons between stimuli for the 1-month group revealed only one significant difference with the mean fixation time for the checkerboard stimulus greater than that to the woman stimulus. For the 3.5-month group, Duncan comparisons revealed there were three significant differences in mean fixation time between the stimuli, i.e., landscape> sunset, triangles; woman < triangles.

First fixation time. The ANOVA on the infants' time of their first fixation to the stimuli yielded a significant main effect of stimulus (F (5, 150) = 3.19, $p < .01$) and no other significant main effects or interactions. The mean first fixation time for each stimulus is shown in Table 6. Duncan tests indicated there were four significant differences between the stimuli in mean first fixation time: baby > sunset, triangle, landscape; checkerboard > sunset.

Number of fixations. The ANOVA on the $\log_{10}(x + 1)$ number of fixations per stimulus failed to reveal any significant differences as a function of age, sex, stimulus, nor were any of the interactions among these variables significant.

Summary

The hypothesis of increasing visual fixation times with increasing age was not supported. Age-related differences in terms of the visual fixation measures (total fixation time and mean fixation time) were

Table 6

Mean First Fixation Time Scores (sec) for Each Stimulus

Stimulus					
Baby[a]	Woman	Landscape	Sunset	Checkerboard	Triangles
7.27 (3.76)	5.66 (3.63)	5.37 (4.09)	4.19 (4.22)	5.98 (4.17)	5.24 (4.04)

Note Maximum Score = 12.0

[a]Numbers in parentheses are SDs.

primarily restricted to differences among the stimuli within the age groups rather than between age groups. No significant age-related differences were found in the mean first fixation times. Thus, these results do not support the hypothesis of age-related increases in sustained visual attention when visual stimuli are presented for a duration similar to that employed in the foreperiod of warned reaction time tasks.

Physiologic Measures

Heart period/interval. For each infant during each trial HP difference scores (HPC/interval) were calculated as the difference between the weighted mean HP for the last 500 msec interval of the pre-period and the weighted mean HP for each of the subsequent 500 msec intervals through the next three subperiods of a trial (i.e., onset, tonic, offset). The ANOVA on these scores yielded significant main effects of sex (F (1, 30) = 5.74, p = .02), period (F (2, 60) = 3.93, p = .02), and interval (F (11, 330) = 11.40, $p < .001$) as well as significant interactions of period x interval (F (22, 660) = 2.54, $p < .00001$), sex x period x interval (F (22, 660) = 1.99, p = .005), age x sex x period x interval (F (44, 660) = 2.69, $p < .0001$), age x stimulus x period x interval (F (220, 3300) = 1.29, p = .003), and sex x stimulus x period x interval (F (110, 3300) = 1.30, p = .02).

Although there are obvious and quite often insurmountable difficulties in interpretating four-way interactions, several interesting findings emerge from these results. Figures 6 through 11 from the interaction of age x stimulus x period x interval illustrate the mean HP responses to each of the stimuli by the three age groups. As can

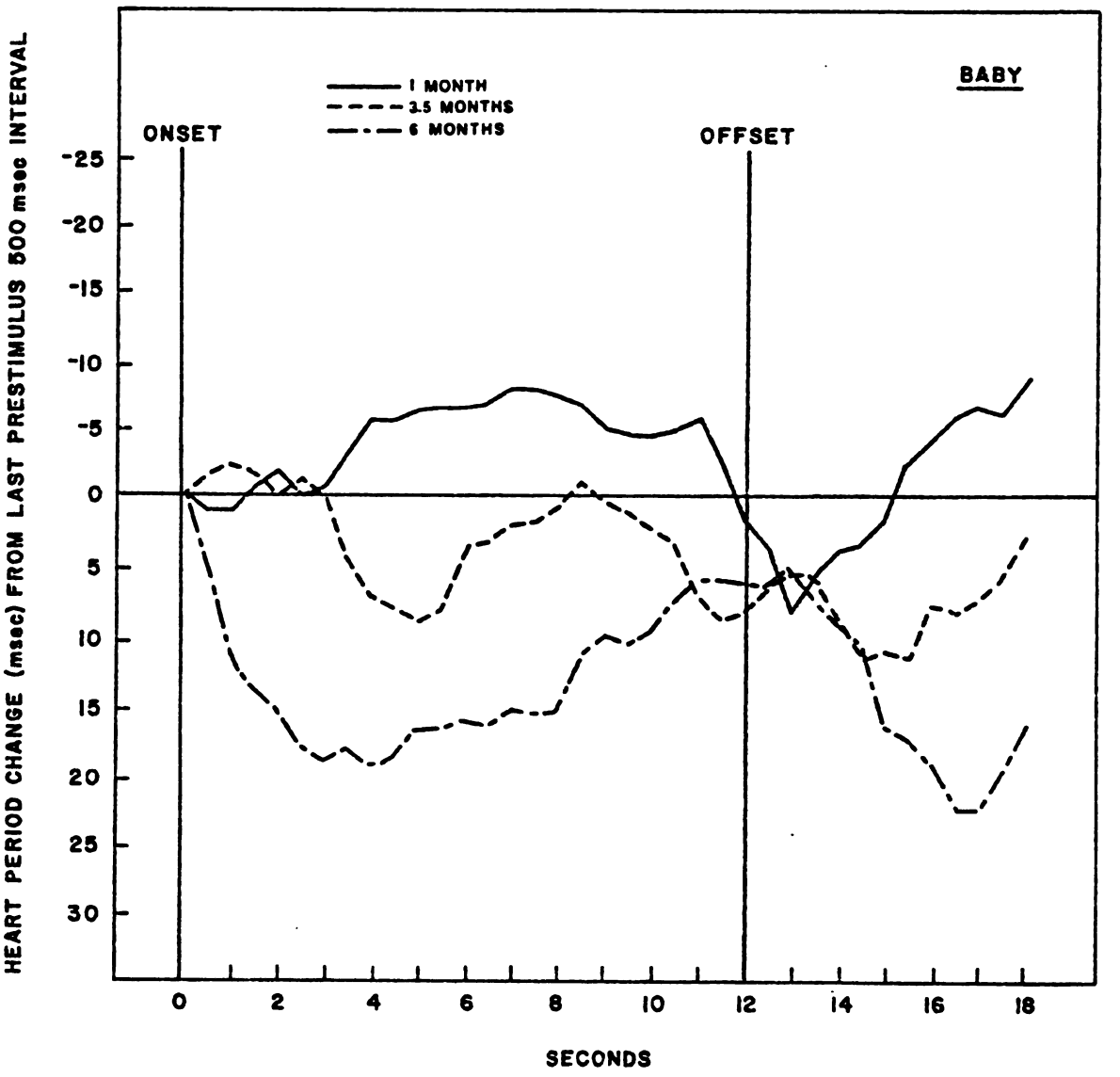

Figure 6. Mean heart period/interval changes to the baby stimulus for the three age groups.

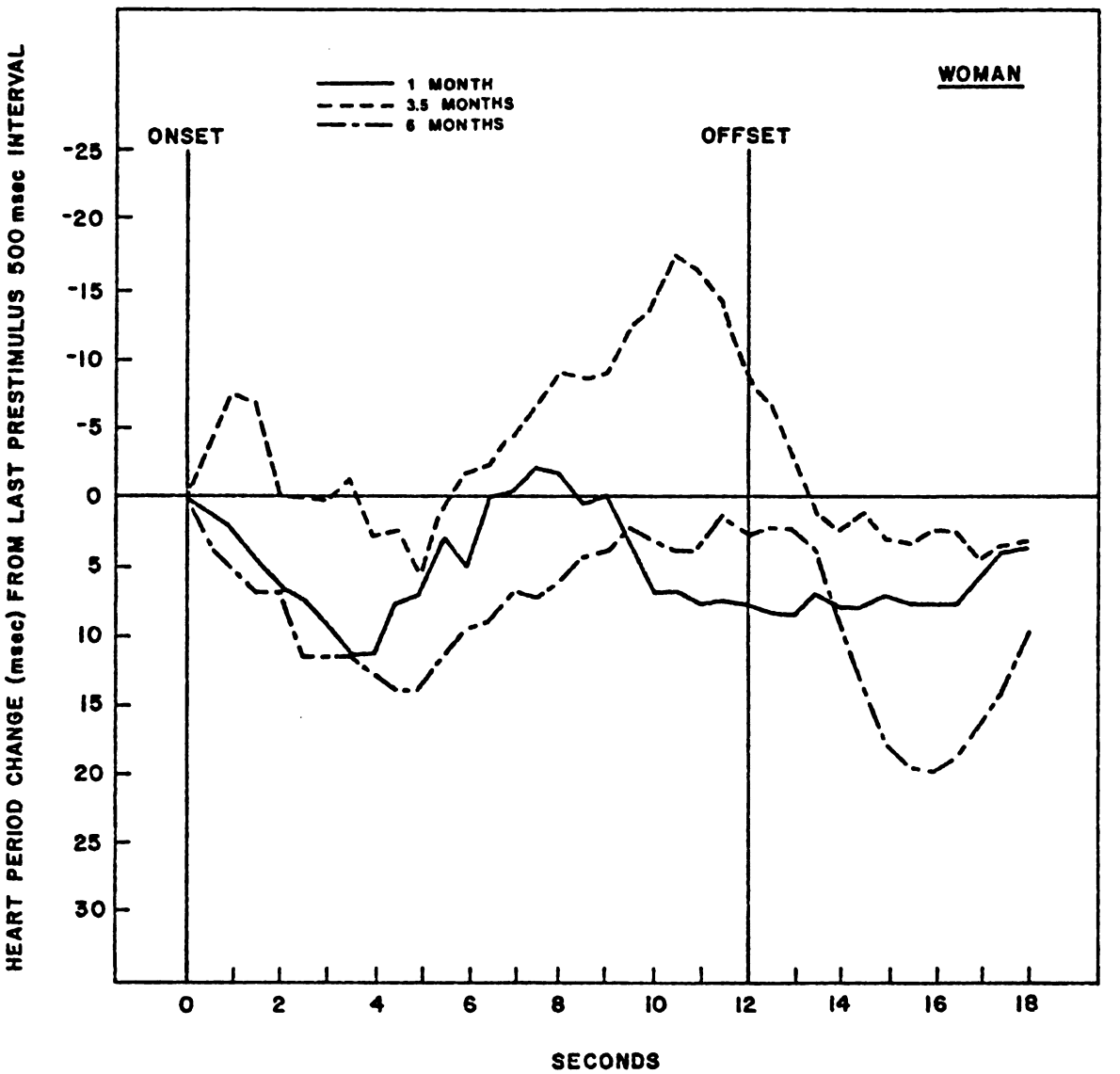

Figure 7. Mean heart period/interval changes to the woman stimulus for the three age groups.

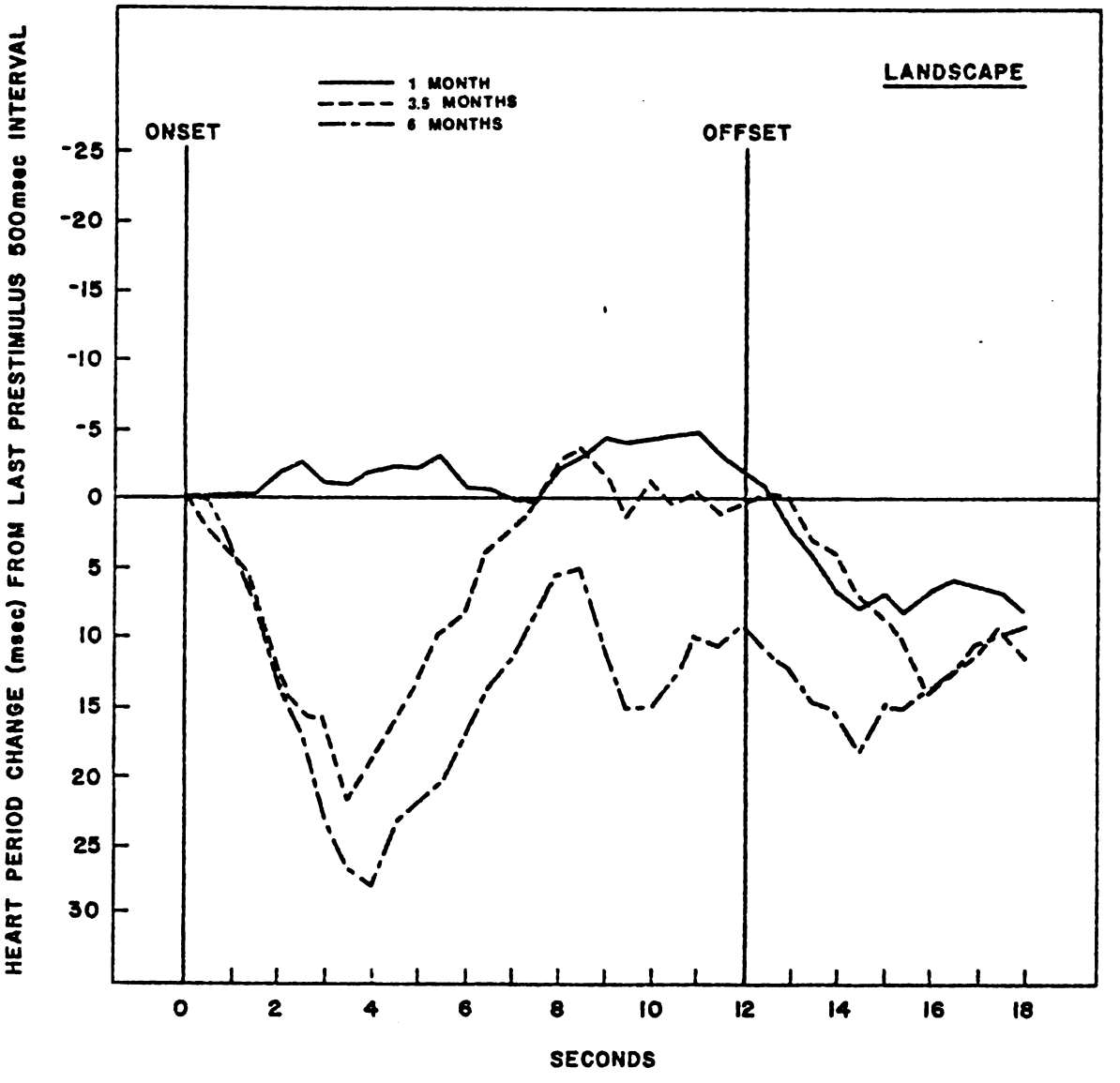

Figure 8. Mean heart period/interval changes to the landscape stimulus for the three age groups.

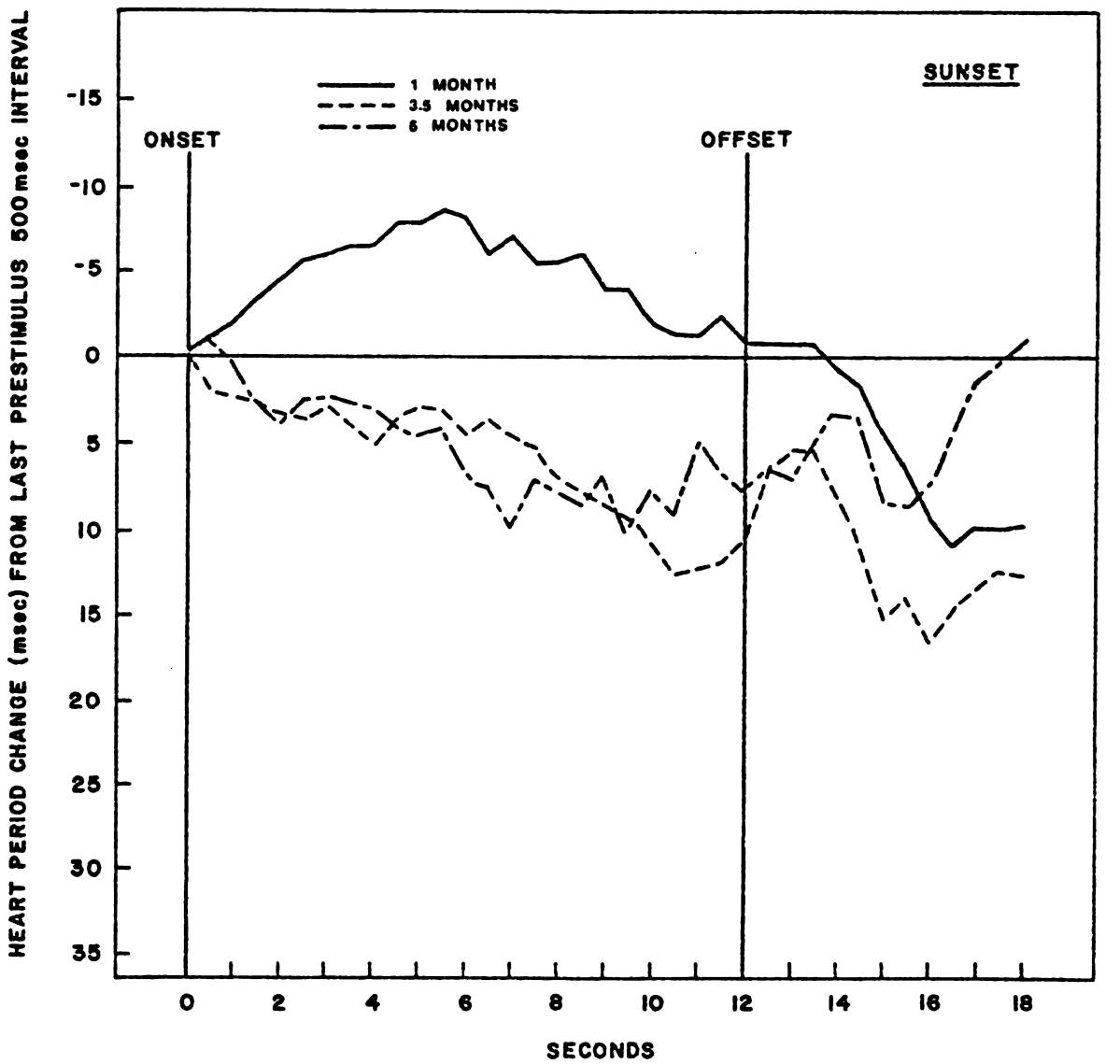

Figure 9. Mean heart period/interval changes to the sunset stimulus for the three age groups.

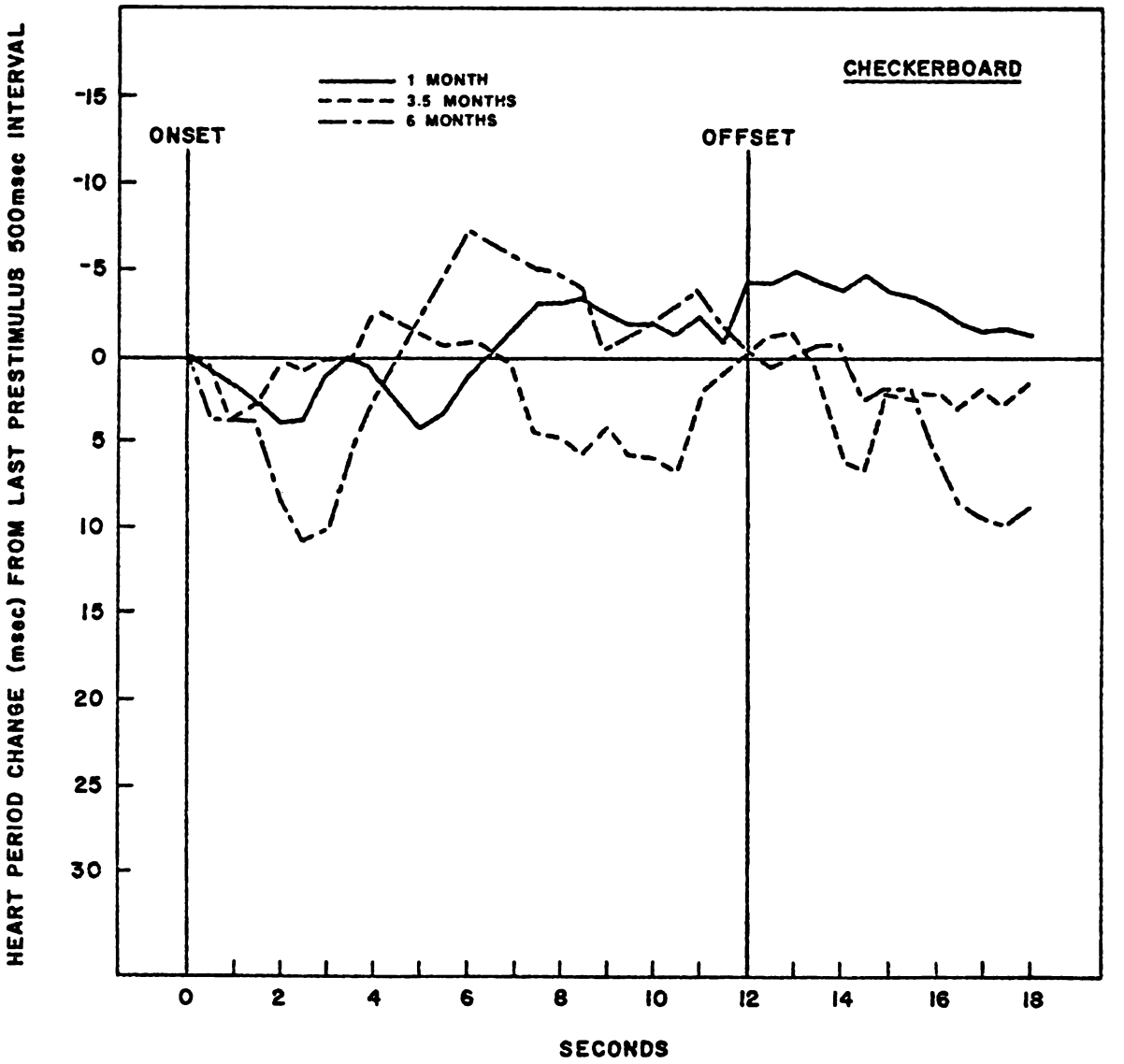

Figure 10. Mean heart period/interval changes to the checkerboard stimulus for the three age groups.

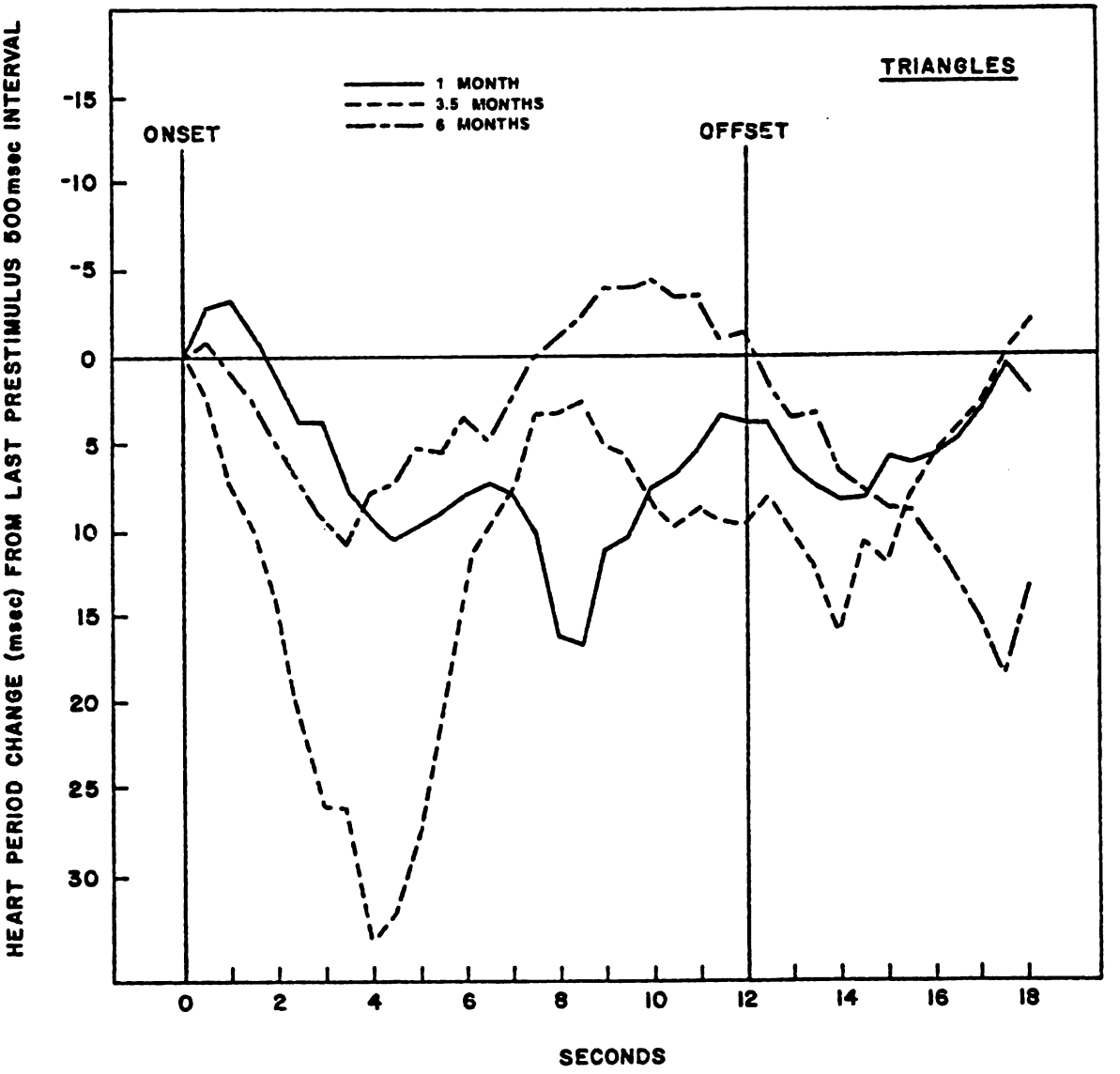

Figure 11. Mean heart period/interval changes to the triangles stimulus for the three age groups.

be seen in comparing these figures, there appears to be within each stimulus trial a general trend consisting of an initial HR decelerative response during the onset-period (sec 1 - 6) which tends to return toward the prestimulus level during the tonic-period (sec 7 - 12) and then another HR decelerative pattern following stimulus offset. However, it is equally clear from these figures that these response topographies fluctuate among the stimuli as well as between the age groups. Regarding the latter, differences in the direction of the HP responses during the stimulus presentations between the 1-month group and the two oldest groups appears to replicate the previously cited developmental shift in directional HR responses. Thus, in contrast to the more typical HR decelerative pattern by the 3.5- and 6-month groups, the 1-month group shows a general HR accelerative pattern to three of the stimuli (see Figures 6, 8, and 9).

Simple effects tests for this interaction (age x stimulus x period x interval) indicated that when the groups were partitioned according to age, there were significant differences in the HP responses among the stimuli for the two oldest groups but not for the 1-month group in that the simple interaction of stimulus x period x interval was significant for only the two oldest groups (3.5-month, F (110, 3300) = 1.37, $p < .01$; 6-month, F (110, 3300) = 1.41, $p < .01$). The lack of a simple interaction of stimulus x period x interval for the 1-month group suggests there were marked individual differences in the HP responses to the stimuli and that the differences in their response patterns across stimuli depicted in Figures 6 through 11 were not consistent among the infants within this group. Further simple effects tests for the oldest

two groups indicated there were significant differences in their HP responses only during the onset-period and not during the tonic- and offset-periods, i.e., the simple interaction of stimulus x interval was only significant for the onset-period for these two groups (3.5-month, F (55, 4950) = 1.84, $p < .05$; 6-month, F (55, 4950) = 1.46, $p < .05$). For the 3.5-month group, Duncan comparisons among the stimuli of the mean HP difference score at the nadir of the response during the onset period revealed that the HR decelerative response to the triangle was significantly greater than that to all of the other five stimuli and that the decelerative response to the landscape stimulus was significantly greater than that for the baby, woman, sunset, and checkerboard stimuli. For the 6-month group, Duncan tests for these scores indicated that this mean HPC/interval score to the landscape stimulus was significantly greater than that to the woman, sunset, checkerboard, and triangles stimuli and that this decelerative response to the baby stimulus was significantly greater than that to the sunset stimulus.

It thus appears that the interaction of age x stimulus x period x interval resulted from significant differences in the HP responses to the stimuli only during the onset-period by the 3.5- and 6-month groups and not by the 1-month group. However, there is a potential confound in this analysis which makes interpretations of the apparent age differences depicted in Figures 6-11 as well as the other two four-way interactions problematic. That is, it is possible that the apparent age differences in the direction and/or magnitude of the HP changes may have been a function of the peak changes in HP occurring at different

intervals during the stimulus presentations. Inspection of the HP response curves for each infant suggested that this might be the case. Moreover, inspection of the visual fixation records indicated that the infants' initial fixation of the stimuli only rarely occurred immediately upon stimulus onset. Therefore, an age (3) x sex (2) x stimulus (6) ANOVA was calculated on the HP response magnitude to each stimulus which was defined for each infant in each trial as the difference between the weighted mean HP for the last prestimulus 500 msec interval and the maximum change in the weighted mean HP (either acceleratory or deceleratory) for the 500 msec interval following the infant's initial fixation of the stimulus within the onset or tonic period. This ANOVA yielded significant main effects of age (F (2, 30) = 8.32, $p < .01$) and sex (F (1, 30) = 7.07, $p < .05$). The mean change scores for these significant main effects indicated that all three age groups and both sexes showed mean HR decelerations (increases in HP). Duncan tests revealed that the mean decelerative response for the 3.5-month (M = 22.3, SD = 36.6) and 6-month (M = 26.5, SD = 24.1) groups were significantly greater than that for the 1-month group (M = 7.2, SD = 34.5). The significant sex effect indicated that the decelerative response for the females (M = 24.1, SD = 35.2) was significantly greater than that for the males (M = 13.3, SD = 34.5).

These results are in line with previous findings of increasing magnitude of HR decelerative responses with increasing age. Nevertheless, it is possible, and inspection of the individual HP response curves suggests, the lower mean magnitude scores for the 1-month group resulted from their exhibiting both accelerative and decelerative responses.

Table 7 shows the number of infants in each age and sex group which exhibited at least one HR accelerative response and the mean number of these responses per infant within these groups. Tests on the proportion of infants within the age groups which exhibited at least one accelerative response showed that the 1-month group had significantly ($p < .05$) higher proportion of infants exhibiting HR accelerative responses than did either of the two older groups. The difference between the oldest two groups in this proportion was not significant. These findings suggest that although all three age groups may respond with an accelerative or decelerative response, the 1-month old infants were more likely to respond with a HR accelerative response than the older infants. Table 8 shows the mean HP change scores for each type of directional response for the age and sex groups and indicates that the magnitude of the HR decelerative responses for the 1-month group are clearly within the range of the older two groups.

Mean heart period/period. The ANOVA on the mean HP/period scores indicated a significant main effect for age (F (2, 30) = 4.43, p = .02), stimulus (F (5, 150) = 2.62, p = .03), period (F (3, 90) = 5.03, p = .003), and a significant interaction of age x stimulus (F (10, 150) = 2.62, p = .007). Figure 12 illustrates the differences in these scores as a function of period. Duncan tests revealed there were significant increases in mean HP from the: (a) pre- to onset-period, (b) pre- to offset-period, and (c) tonic- to offset-period.

Table 9 shows the mean HP scores for the interaction of age x stimulus. The simple main effects ANOVA for this interaction yielded significant simple main effects of age for the baby (F (2, 180) = 6.71,

Table 7

Number of infants with at Least One HR acceleratory Response and Mean Number of HR Acceleratory Responses for Each Subject per Age and Sex Groups

Sex	Age in Months		
	1	3.5	6
Males			
No. of Subjects[a]	6	4	3
Mean No. of HRAs[b]	2.67	1.50	.50
Females			
No. of Subjects	6	2	2
Mean No. of HRAs	2.33	.33	.33
Column			
No. of Subjects	12	6	5
Mean No. of HRAs	2.50	.92	.42

Note. HRA = Heart Rate Acceleration Response.

[a] n = 6 per cell

[b] maximum score = 6.0

Table 8

Mean HP Change Scores for HR Decelerative and HR Accelerative Responses per Age and Sex Group

Group	HRD		HRA	
	M	SD	M	SD
1 month				
Males	30.9	12.7	-31.9	15.8
Females	38.1	34.1	-30.4	15.8
3.5 month				
Males	30.7	22.7	-44.1	27.3
Females	37.0	26.8	-43.5	31.8
6 month				
Males	28.7	22.3	-18.7	6.5
Females	31.4	19.5	-24.0	17.0

Note. HRD = Heart Decelerative Response, HRA = Heart Rate Accelerative Response (decreased HP).

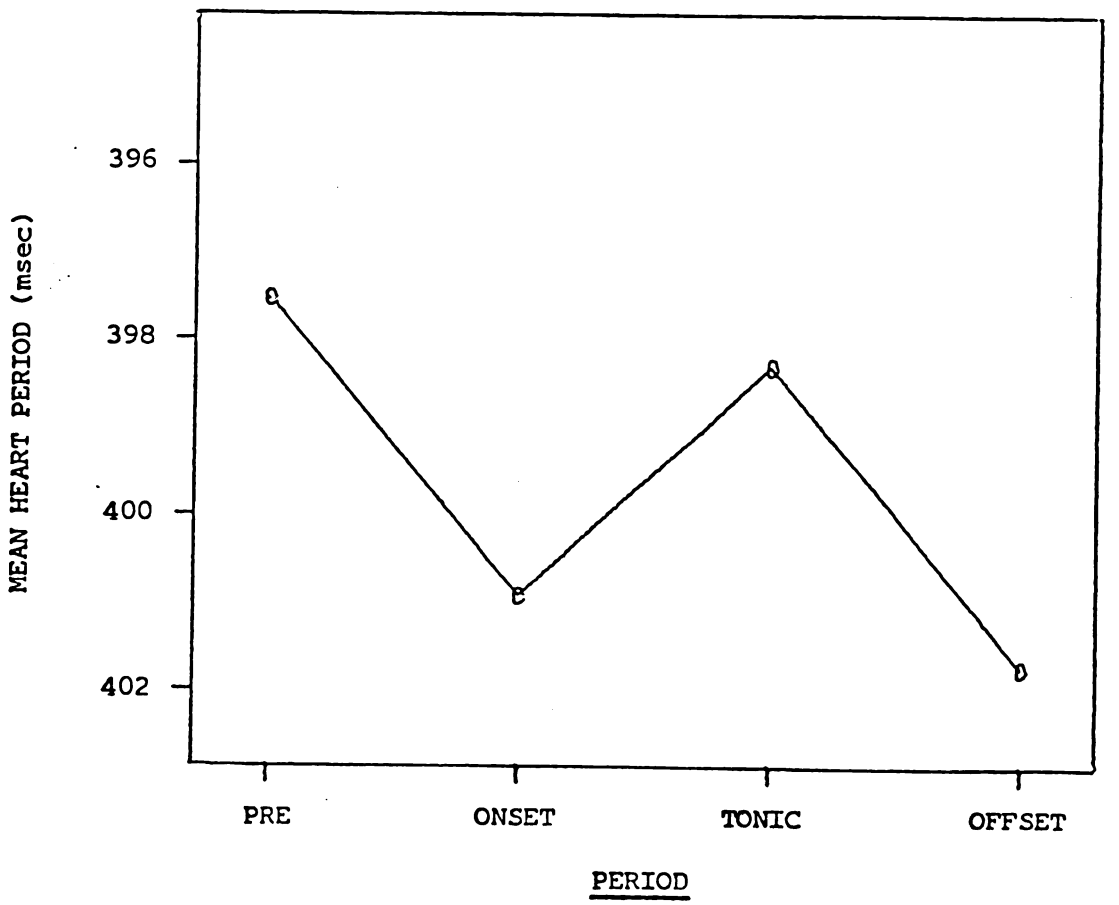

Figure 12. Mean heart period scores as a function of period.

Table 9

Mean Heart Period Scores from the Interaction of Age x Stimulus

	Stimulus					
Age (mo.)	Baby	Woman	Landscape	Sunset	Checkerboard	Triangles
1	380.6	379.0	376.8	378.8	386.3	387.2
3.5	402.3	411.8	402.1	401.4	407.5	415.7
6	422.5	407.5	410.5	401.1	410.0	405.2

$p < .01$), woman (F 2, 180) = 4.84, $p < .01$), landscape (F (2, 180) = 4.71, $p < .01$), and triangles (F (2, 180) = 3.17, $p < .05$) stimuli as well as significant simple stimulus effects for the 3.5-month (F (5, 150) = 2.56, $p < .05$) and 6-month group (F (5, 150) = 3.83, $p < .01$). Duncan tests yielded the following significant age trends: for the baby stimulus, 1-month < 6-month; for the woman and landscape stimuli, 1-month < 3.5-month, 6-month; for the triangles stimulus, 1-month < 3.5-month. Within the 3.5-month group, Duncan tests indicated three significant differences in the mean HP/period scores among the stimuli: triangles > sunset, landscape, baby. Within the 6-month group, Duncan tests revealed there were five significant differences in mean HP/period scores among the stimuli: baby > woman, landscape, sunset, checkerboard, triangles.

Heart period variance/period. The ANOVA on the $\log_{10}$ HPV scores per period yielded a significant main effect of period (F (3, 90) = 3.38, $p = .02$) and no other significant main effects or interactions. Figure 13 illustrates the changes in this variable as a function of period. Duncan tests indicated that the mean $\log_{10}$ HPV score was greater in the offset-period than for either of the pre- or tonic-periods. There were no other significant differences.

Respiratory amplitude variance/period. The ANOVA on the $\log_{10}$ respiratory amplitude variance scores per period yielded a sigificant main effect of period (F (3, 90) = 2.97, $p = .04$) and no other significant main effects or interactions. Figure 14 shows the decline in this measure across the periods within a trial. Duncan tests indicated significant differences in this measure between the pre- and tonic-

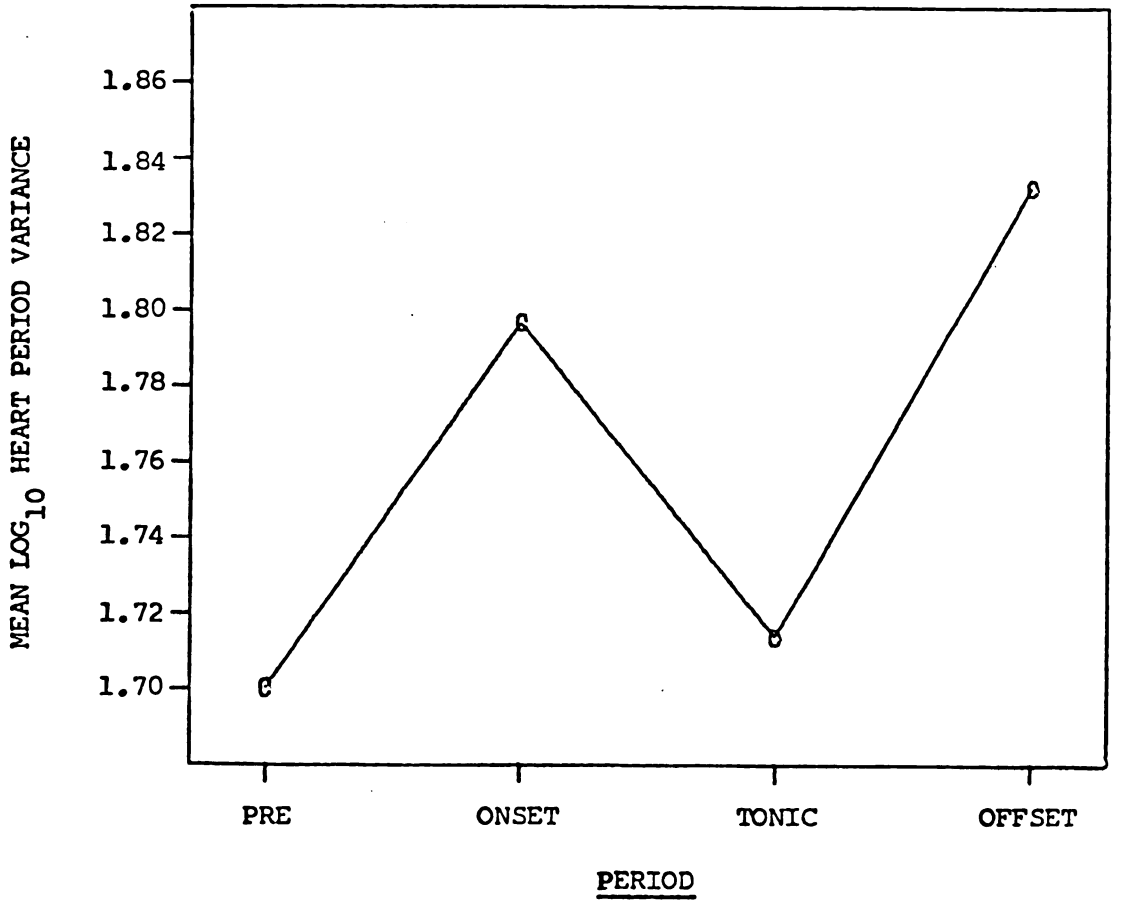

Figure 13. Mean heart period variance scores as a function of periods.

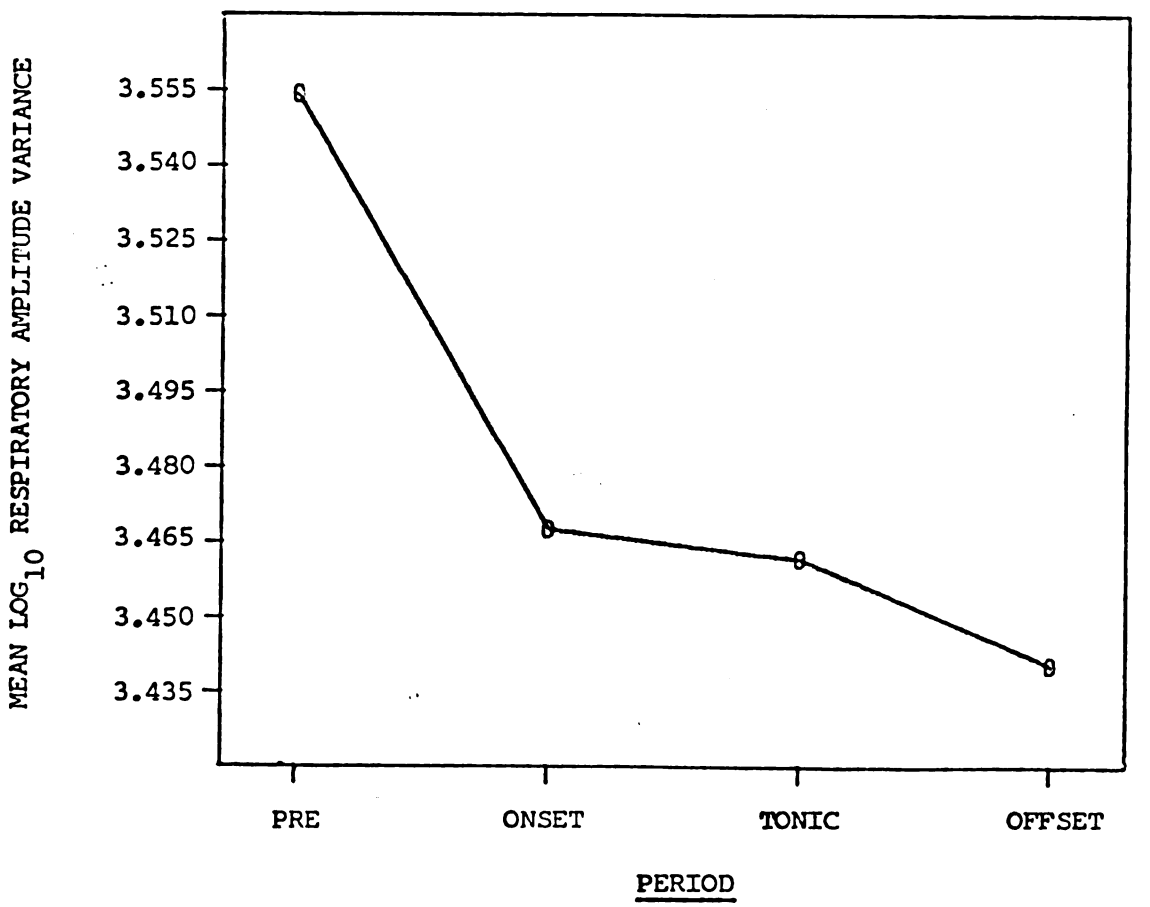

Figure 14. Mean respiratory amplitude variance scores as a function of period.

periods as well as between the pre- and offset-periods. No other comparisons were significant.

Correlational Analyses

Pearson product moment correlations (between subjects) were computed between the various physiologic and visual fixation response measures (see Table 10). Unexpectedly, as can be seen in Table 10, virtually none of these correlations were significant. However, six out of six of the correlations between mean HPV changes and the visual fixation measures were in the expected direction, i.e., greater mean decreases in HPV were associated with greater mean visual fixation scores. Four out of the six correlations between the mean RAV scores and the visual fixation measures were also in the expected direction, i.e., greater mean decreases in RAV were associated with greater visual fixation scores. Nevertheless, it is clear that these correlations do not support the hypothesis that these physiologic response parameters are predictive of behavioral response measures which can be reasonably assumed to reflect orienting and sustained attention to visual stimuli.

Summary

The results from the analyses of the HPC/interval responses replicated previous findings of a developmental change in the direction of HR responses in that there was a significant decline from 1- to 3.5-months in the proportion of infants showing at least one HR accelerative response to the stimuli. The initial results also seemed to support the hypothesis of increases in the magnitude of HR decelerative responses to stimulus onset with increasing age. However, the mean

Table 10

Pearson Product Moment Correlations Between Visual Fixation and Physiologic Response Measures

	Total Fixation	Mean Fixation	First Fixation
HPC/Interval	.03	-.13	-.02
MHP/Period			
Onset	-.02	-.17	-.06
Tonic	.00	-.04	-.04
HPV/Period			
Onset	-.08	-.20	-.22
Tonic	-.07	-.05	-.09
RAV/Period			
Onset	.09	.04	-.06
Tonic	-.07	-.20	-.16

HPC/interval scores for both types of directional HR responses shown in Table 8 suggests that the magnitude of the decelerative responses for the 1-month group were clearly in the range of those for the two older groups.

The hypothesis of increasing magnitude of the suppression of HRV and RAV responses with increasing age was not supported. However, the infants did appear to show suppression of RAV in a fashion quite similar to that reported for adults and older children.

Finally, the lack of any significant intercorrelations between the visual fixation and physiologic response measures did not support the hypothesis that these physiologic response parameters index the degree to which the infants oriented to or sustained their attention to the stimuli. These negative findings, however, seem to provide indirect support for the hypothesis that the development of the neural control of the heart may be responsible for the observed developmental changes in the direction of the HR responses rather than the development of orienting and/or sustained attention. The results of relatively more direct tests of this latter hypothesis are presented in the next section of this chapter.

Respiratory Sinus Arrhythmia and Attention

Since there were no significant differences as a function of age in the baseline levels of C_w, the hypothesis that individual differences in the neural control of the heart may be related to individual differences in orienting and sustained attention was initially examined by subdividing all of the infants into high- and low-C_w groups and then

testing whether or not the measures of orienting and sustained attention varied as a function of these levels of C_w. Thus, all infants were ranked from the highest to lowest on the basis of their mean C_w scores (mean of the pre- and post-baseline estimates of C_w) and divided into high- and low-C_w groups at the median. Mean C_w scores ranged from .496 to .020 (Md = .173) and the means and standard deviations of C_w for the high- and low-C_w groups were, respectively, M = .279, SD = .092; M = .103, SD = .046. Table 11 summarizes the distribution of infants within these C_w groups.

C_w and Visual Fixation Responses

Separate C_w group (2) x stimulus (6) ANOVAs for each of the visual fixation dependent variables failed to yield any significant main effects of C_w group nor was the C_w group x stimulus interaction significant. The stimulus main effect was significant for the total fixation measure (F (5, 180) = 5.61, p = .0001) and for the first fixation time measure (F (5, 180) = 3.04, p = .012).

C_w and Physiologic Responses

HPC/interval. The ANOVA on the HPC/interval scores yielded significant main effects of period (F (2, 68) = 4.10, p = .02), and interval (F (11, 374) = 10.43, $p < .0001$) as well as significant interactions of period x interval (F (22, 748) = 2.21, p = .001), C_w group x stimulus x period (F (10, 340) = 1.99, p = .03), and C_w group x stimulus x interval (F (55, 1870) = 1.92, $p < .0001$).

Figures 15 through 20 illustrate the mean HPC/interval responses to the stimuli by the two C_w groups. As can be seen in comparing these

Table 11

Distribution of Infants Within C_w Groups

Age	C_w Group			
	High		Low	
	Males	Females	Males	Females
1 month	4	3	2	3
3.5 month	2	2	4	4
6 month	3	4	3	2

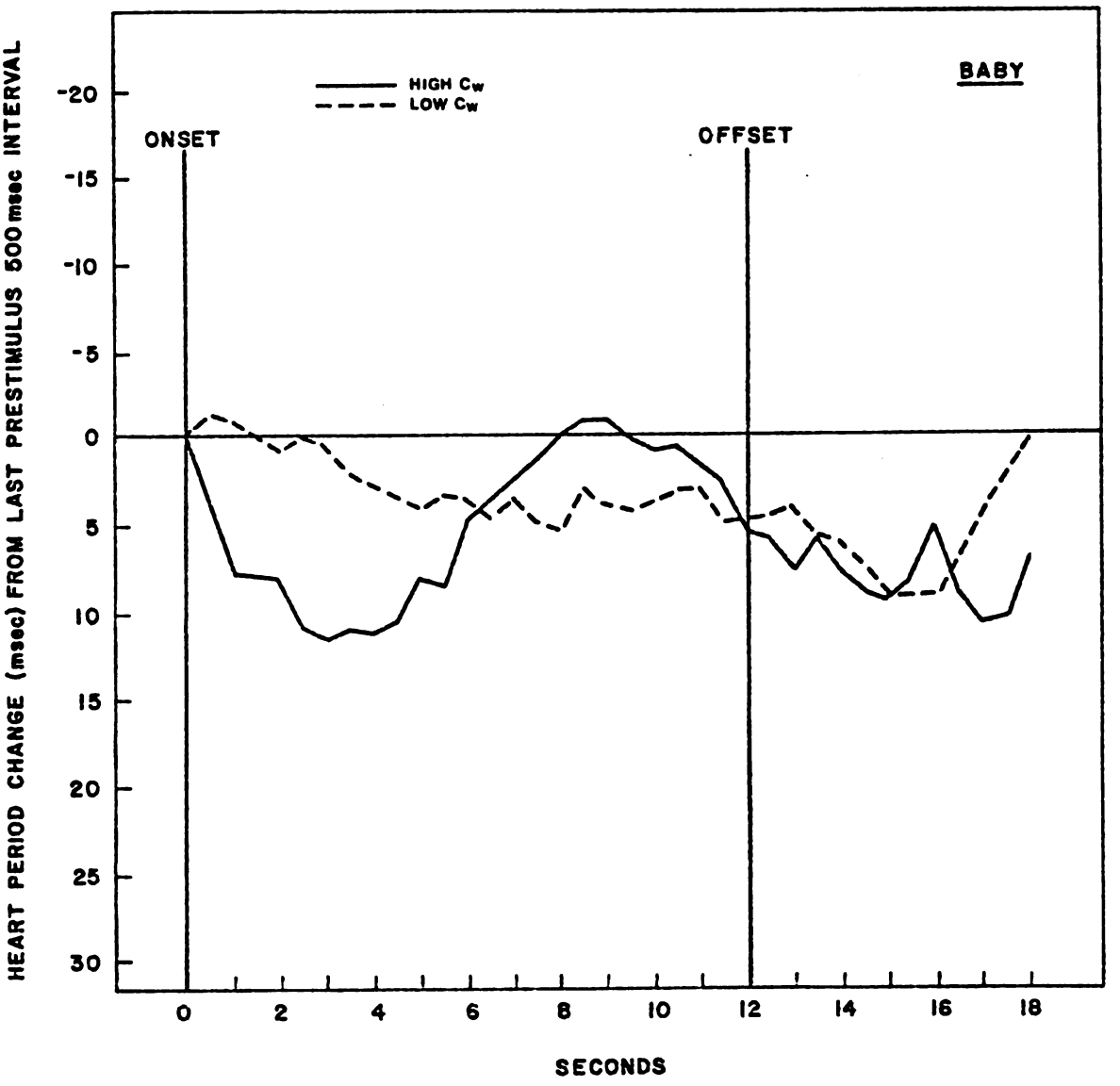

Figure 15. Mean heart period/interval changes to the baby stimulus as a function of C_w group.

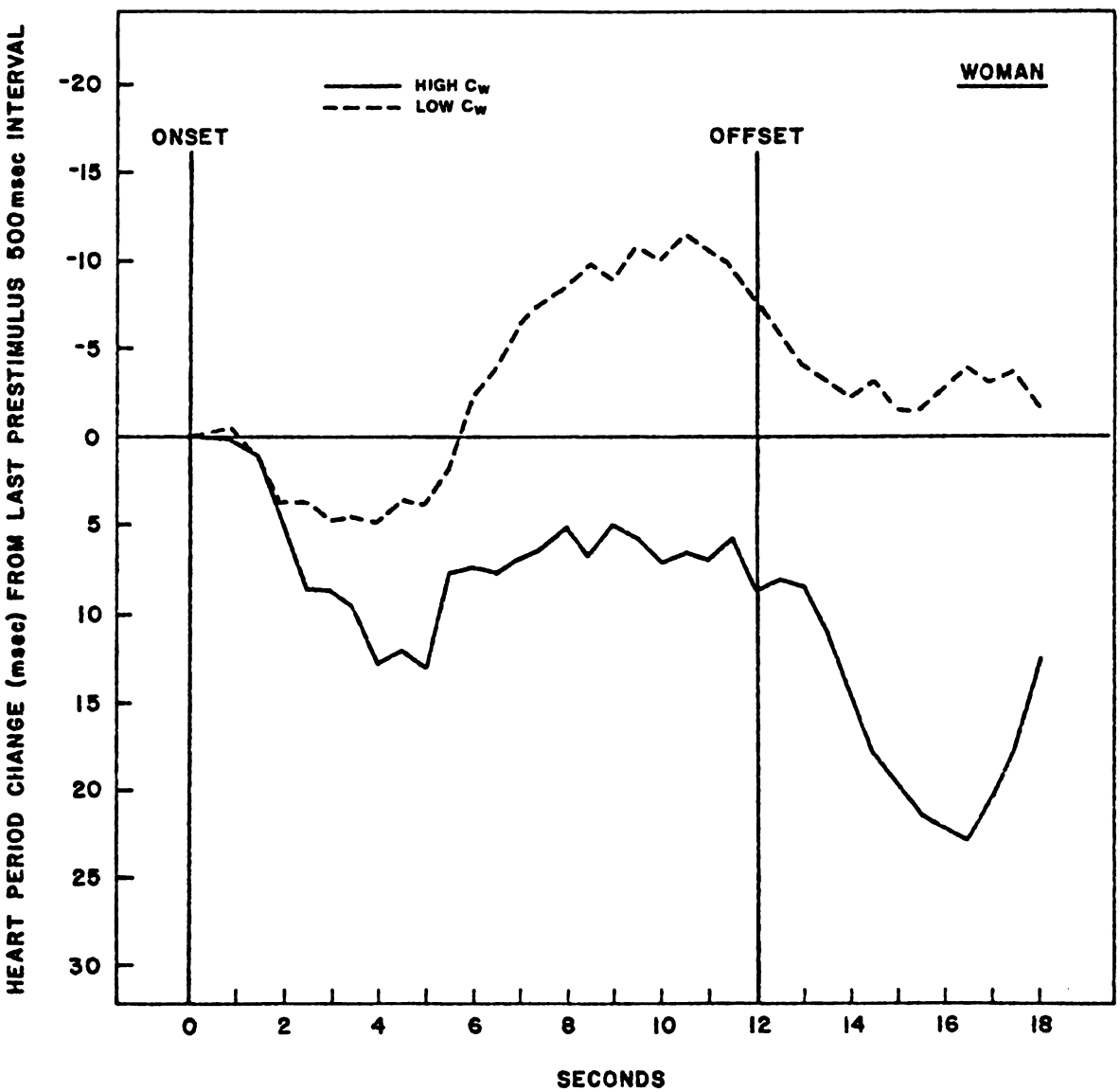

Figure 16. Mean heart period/interval changes to the woman stimulus as a function of C_W group.

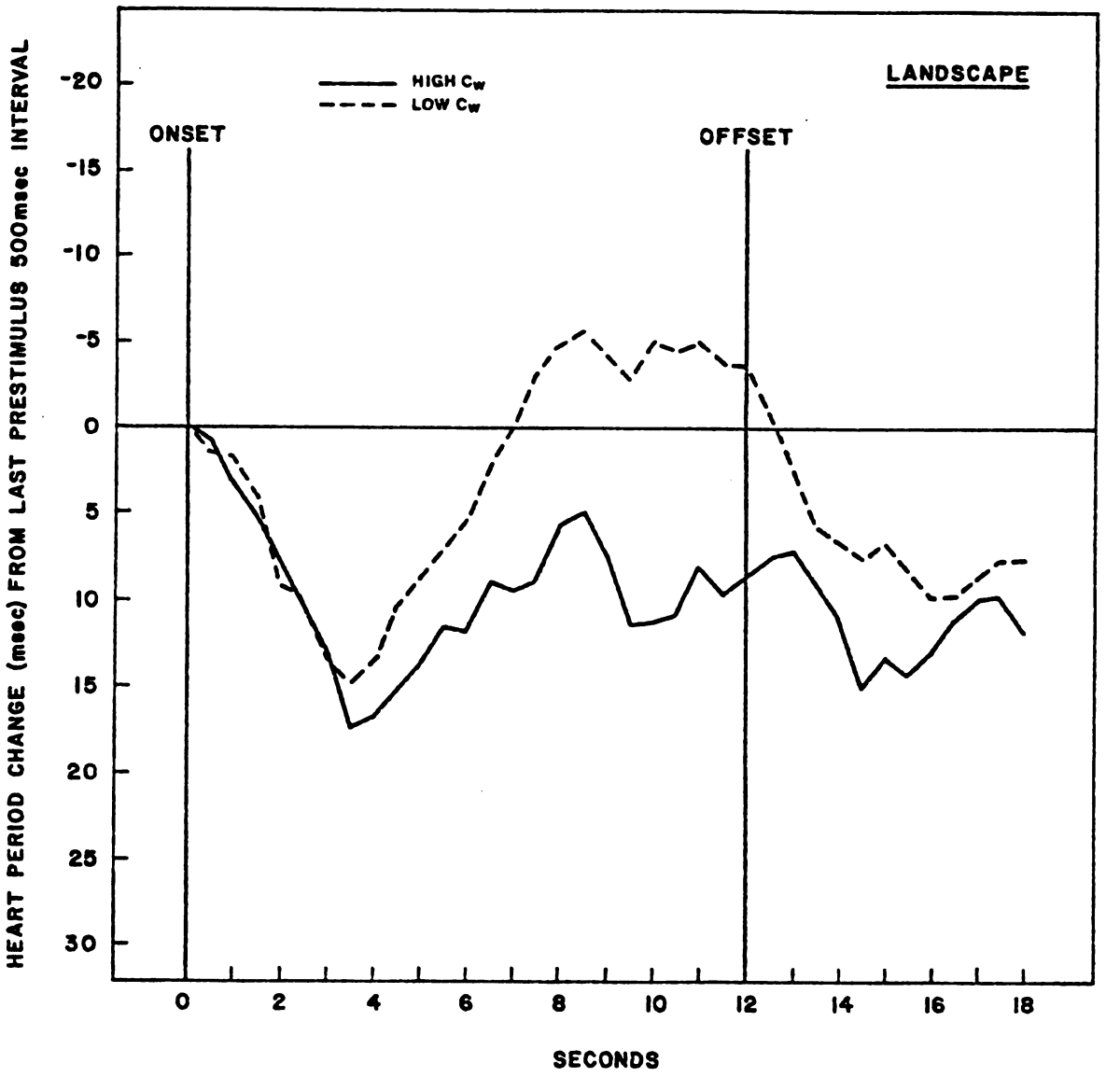

Figure 17. Mean heart period/interval changes to the landscape stimulus as a function of C_W group.

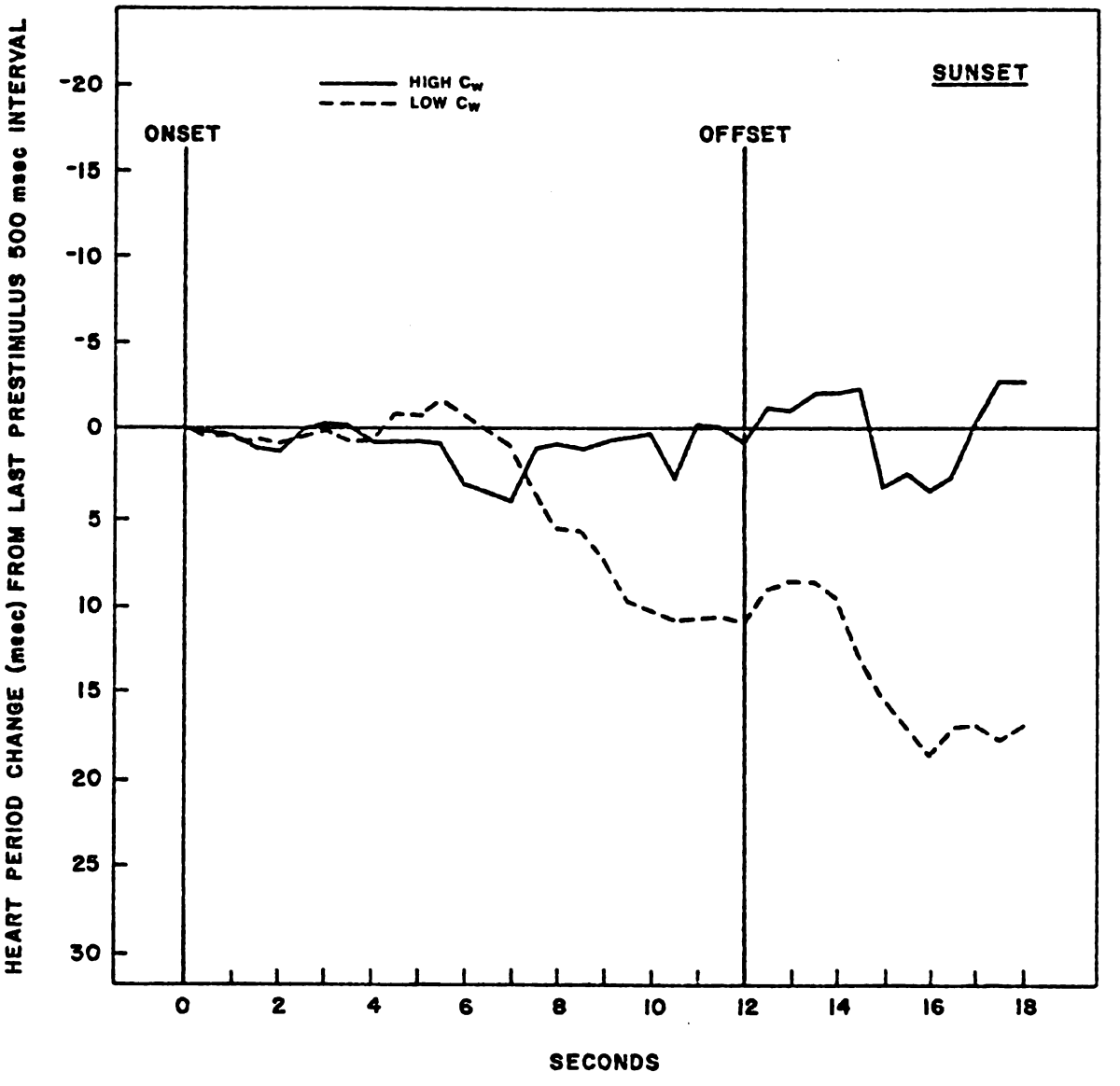

Figure 18. Mean heart period/interval changes to the sunset stimulus as a function of C_w group.

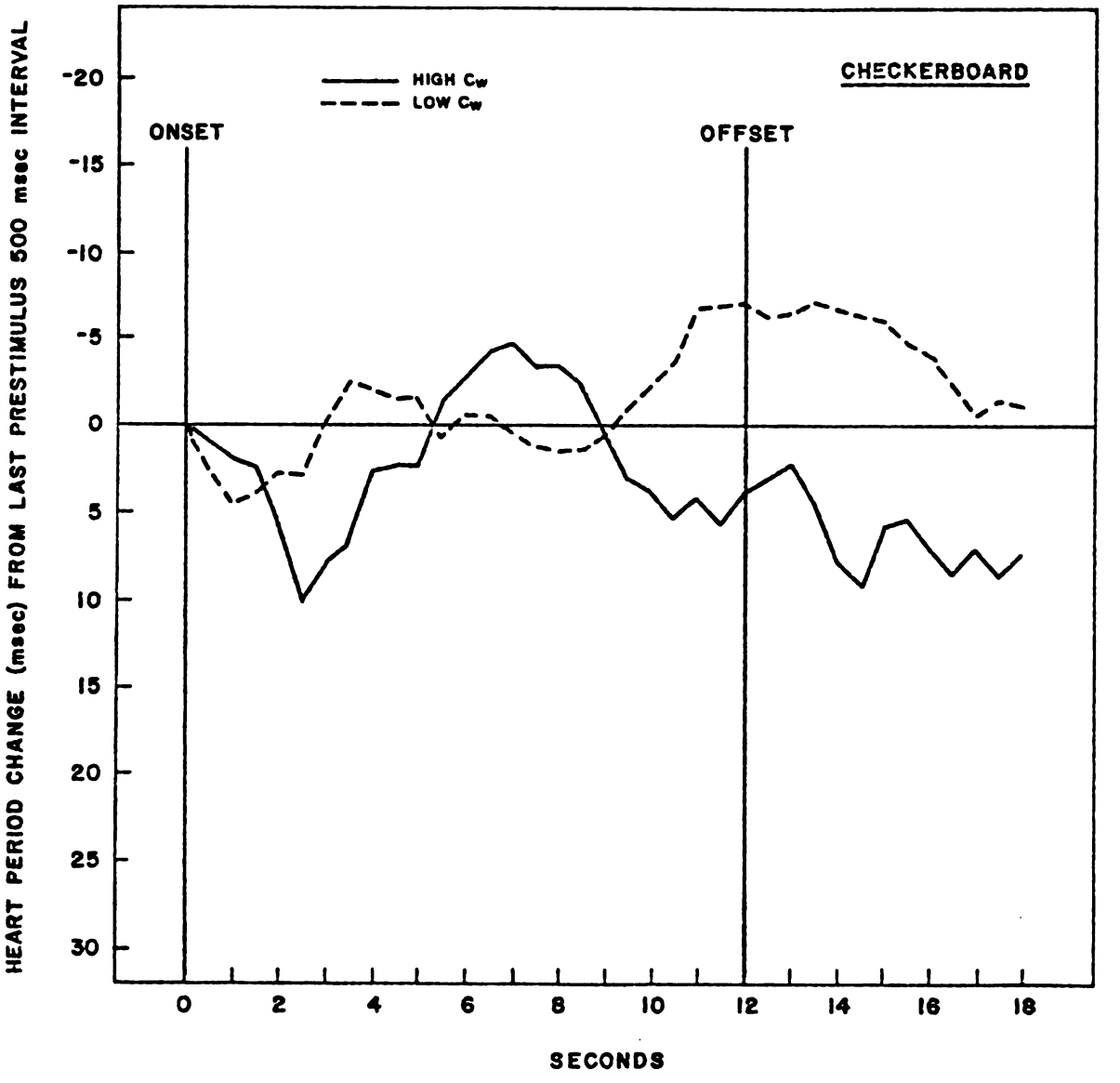

Figure 19. Mean heart period/interval changes to the checkerboard stimulus as a function of C_w group

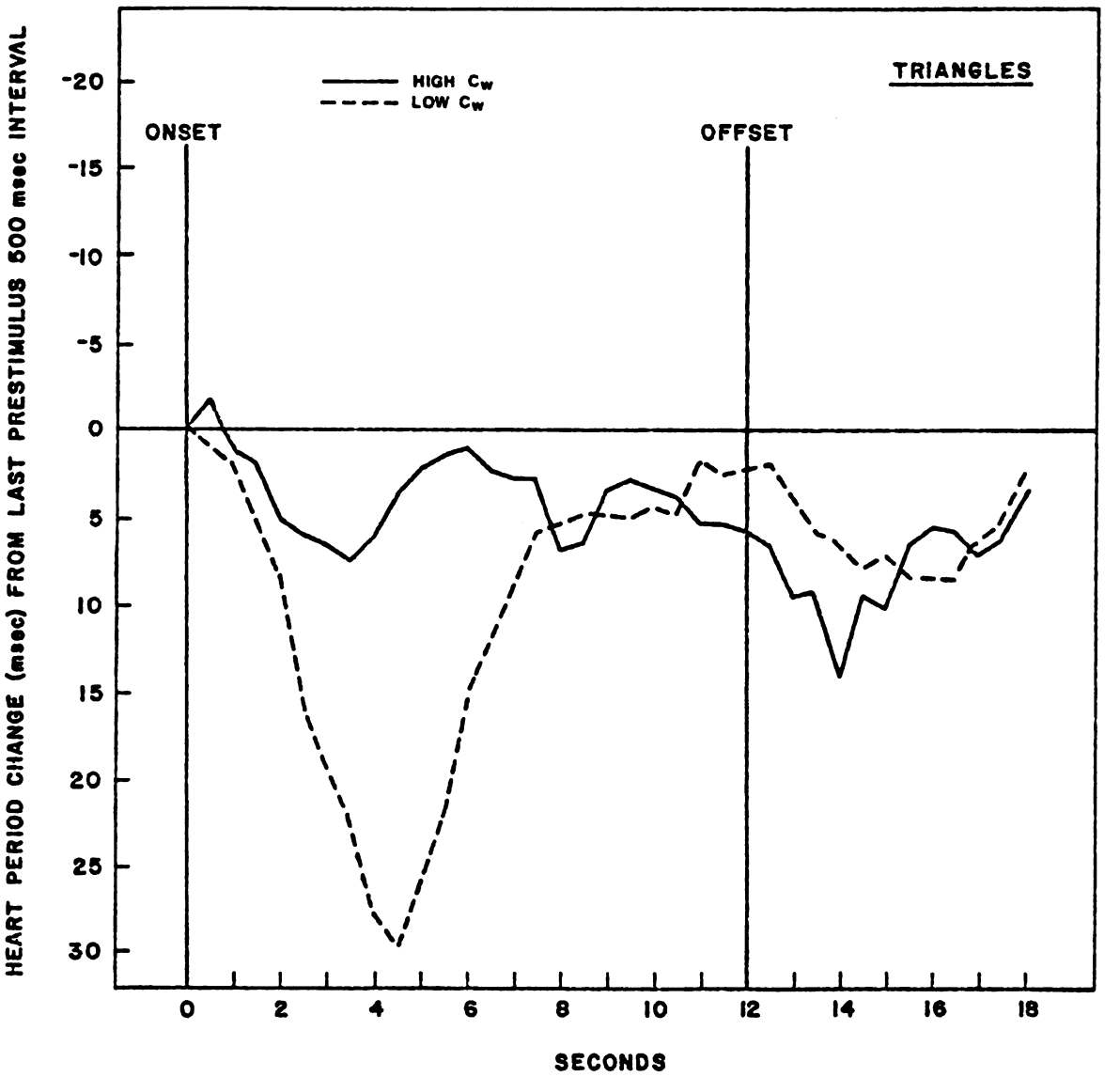

Figure 20. Mean heart period/interval changes to the triangles stimulus as a function of C_w group.

figures, the magnitude of the HR decelerative pattern to stimulus onset to four of the six stimuli (see Figures 15, 16, 17, and 19) appears to be greater for the high-C_w group than for the low-C_w group. Since this analysis was subject to the same confound as the previous age-ANOVA of the HPC/interval scores, further statistical tests for this ANOVA did not seem warrented. Instead, a C_w group (2) x stimulus (6) ANOVA was calculated for the HPC/interval magnitude scores (defined as before in the age-analysis). This latter ANOVA failed to yield a significant main effect of C_w group nor was the interaction of C_w x stimulus significant. These results suggest that the apparent differences in the HP responses depicted in Figures 15-20 were most likely due to differences in the intervals during which the peak changes in the HP responses occurred rather than due to differences in the magnitude of the responses. Moreover, the proportion of infants within each C_w group which exhibited at least one HR accelerative response did not differ significantly between the groups (high-C_w = .61 versus low-C_w = .67). The mean number of HR accelerative responses per infant did not differ between the C_w groups (high-C_w = 1.28, low-C_w = 1.11).

Period analyses. The ANOVA on the mean HP/period scores yielded significant main effects of stimulus (F (5, 170) = 2.36, p = .04) and period (F (3, 102) = 4.82, p = .004) as well as a significant interaction of C_w group x stimulus x period (F (15, 510) = 1.76, p = .04). The interaction of C_w group x stimulus x period is illustrated in Figure 21. However, given the results of the analysis of the HPC/interval scores discussed above, this interaction is best interpreted in light of those findings.

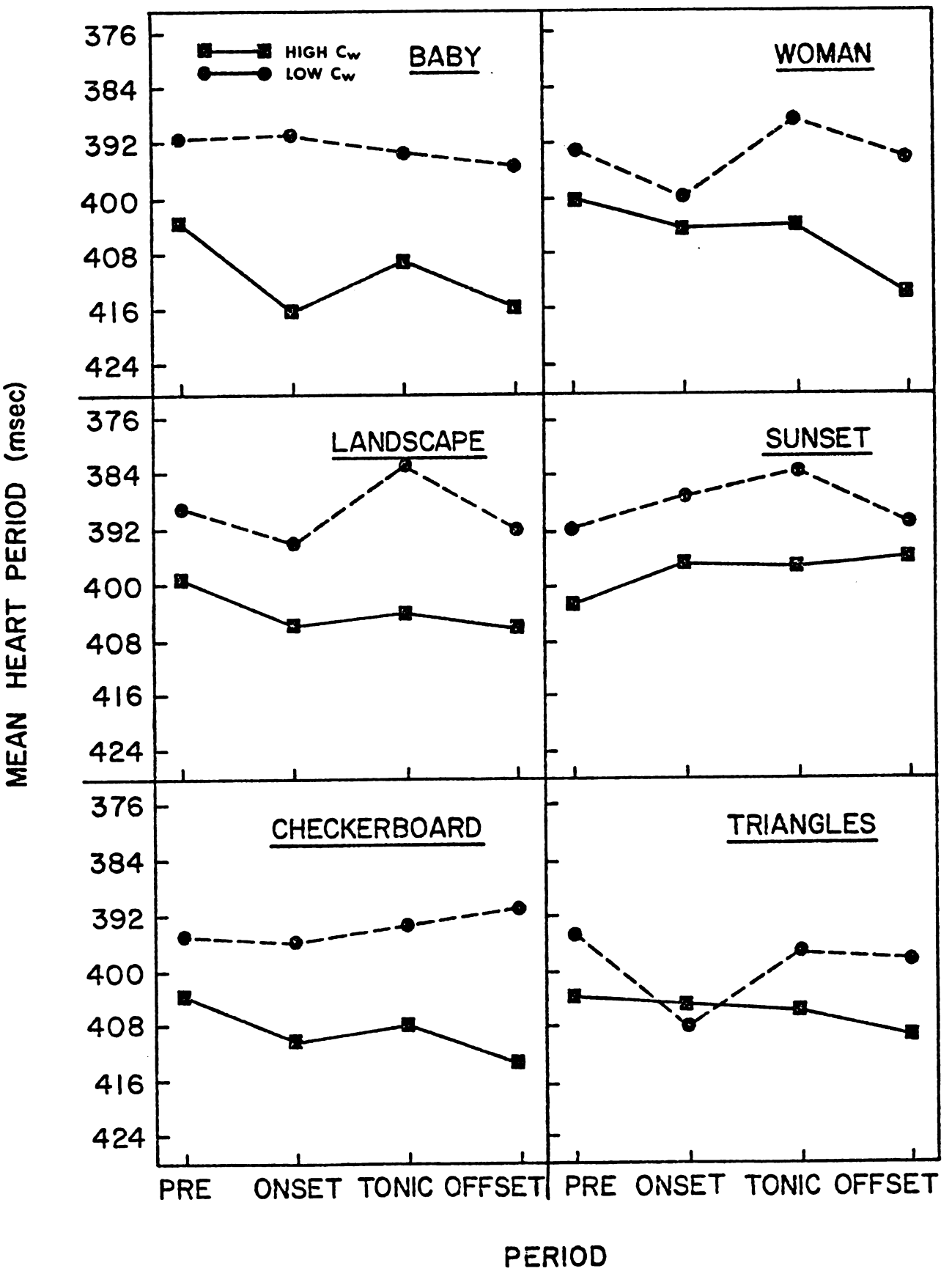

Figure 21. Mean heart period scores from the interaction of C_w x stimulus x period.

Only significant main effects for period emerged out of the ANOVAs for the HRV/period (F (3, 102) = 2.36, p = .02) and RAV/period (F (3, 102) = 2.84, p = .04) dependent variables.

Correlational Analyses

The previous analyses failed to provide any support for the hypothesis that individual differences in the relative vagal control of the heart, as measured by the Porges C_w function, are related to these sorts of differences in the behavioral and physiologic measures of orienting and sustained attention used in this study. Recall, however, there were age-related differences in the other two estimates of vagal influences on the heart, i.e., the amount of RSA and peripheral vagal tone. Thus, one might expect that these parameters, rather than C_w, might be related to the behavioral and physiologic measures of orienting and sustained attention. Therefore, Pearson product moment correlations were computed between the three estimates of vagal influences on the heart and the measures of orienting and sustained attention (see Table 12). As can be seen in Table 12, the only significant correlation to emerge out of these analyses was a positive correlation between mean first fixation time and mean C_w such that longer mean first fixations were associated with greater mean levels of C_w. It is also interesting to note several of the other correlations which approached significance (i.e., r (35) = .275, $p < .10$). For example, the mean amounts of RSA were positively related with mean first fixation times and the magnitude of the mean HPC/interval change scores whereas none of the visual fixation measures were correlated with the estimates of peripheral vagal tone. Moreover, the estimate of vagal tone was

Table 12

Pearson Product Moment Correlations Between Measures of the Neural Control of the Heart and Measures of Attention

	C_w	ARSA	Vagal Tone
Total Fixation	.13	.22	.08
Mean Fixation	.13	.18	.07
First Fixation	.36*	.30	.12
HPC/Interval	-.03	.32	.29
Mean HP/Period			
Onset	-.16	.21	.27
Tonic	.04	.21	.13
HPV/Period			
Onset	-.13	.10	.13
Tonic	-.09	-.13	-.17
RAV/Period			
Onset	.09	.02	-.26
Tonic	.14	.06	-.15

*$r(35) = .325$, $p < .05$

positively correlated ($p < .10$) with the mean magnitude of the HPC/interval scores such that greater mean levels of peripheral vagal tone were associated with greater mean HR decelerations.

CHAPTER V

DISCUSSION

The present study was designed to investigate developmental aspects of cardiac functioning during early human infancy. Two aspects of cardiovascular function were investigated. The first had to do with the development of the neural control of the heart, particularily in terms of parasympathetic-vagal influences, and the second was concerned with the question of HRV and the directional HR response as a measure of orienting and sustained attention. While the results concerning the parasympathetic-vagal influences on the heart did provide support for the hypothesis of increasing vagal control with increasing age during the first six postnatal months, the results bearing on the use of HR as a dependent variable in infant attention research were less clear-cut and were generally negative. Nevertheless, the results from both parts of the study provided indications for further research.

Development of the Neural Control of the Heart

The hypothesis provided by Porges (1976a; Reese & Porges, 1976) of a developmental increase in the relative vagal control of the heart during early human infancy as measured by the C_W function was not supported. One possible explanation for this may be that the data of the present study were cross-sectional and, hence, individual differences within the age groups were completely completely confounded

with between age group differences. Thus, if a sample of infants were followed longitudinally thereby eliminating the confounding of individual differences with possible age differences, a developmental increase in C_w might have been observed. However, recent data reported by Harper et al. (1978) suggest that the data for the C_w function in the present study provide a reasonably accurate description of its developmental function. In a longitudinal design (n = 16) using cross spectral techniques, Harper et al did not find significant age differences in the "coherence of sinus arrhythmia" (CSA) in the awake state (the infants were seen in this study at 1 week, 1-, 2-, 3-, 4-, and 6-months of age). Although CSA values reported by Harper were higher than those found in the present study (e.g., at 1, 3, and 6 months the respective mean CSA values were .387, .38, and .413), this may have been due to several differences in the algorithms used in computing these values or due to the fact that the means for CSA were based on minute-by-minute values over a 12-hour recording session.

From a broader developmental perspective, the lack of significant increases in C_w during the first six months does not, of course, rule out the possibility of increases in C_w after this age period. Mean C_w values reported in the previously cited studies by Porges with hyperactive and nonhyperactive controls (Porges, 1976a; Porges, et al., Note 5) as well as unpublished data with adults (Porges, Note 6) suggests there may be increases in C_w after the first six postnatal months in that the mean C_w values for these children and adults were approximately double those found for the infants in the present study.

Although developmental changes in C_w were not found in the present

study, significant age-related trends suggesting increasing vagal influences on the heart were found in the other two estimates of vagal functioning analyzed, i.e., the absolute amount of RSA (the amount of HPV shared with respiration) and peripheral vagal tone (the total amount of HPV within the primary respiratory frequency band). The results for both of these measures indicated that the significant developmental changes occurred from 1 to 3.5 months of age. However, there was a significant sex difference in the age-related increase in peripheral vagal tone. Both males and females showed mean increases in this measure, but only the increase for the males was significant. Whether or not this reflects a "true" developmental sex difference or was due to the complete confounding of individual differences with sex in this study remains an open question. Additional data from the Harper et al. (1978) study suggest that the latter may be the case. In their study Harper et al. also found significant increases in vagal tone from 1 to 3 months of age but did not find a significant sex difference in this trend. (Vagal tone in their study was referred to as the extent of sinus arrhythmia or XSA but corresponds to the vagal tone measure in the present study in that it was the total amount of HPV within the primary respiratory frequency band.)

It is also interesting to note that Harper et al. found significant differences in CSA as a function of state with CSA the highest in the quiet sleep state, lower in the REM sleep state, and lowest in the awake state. They suggested that these differences in CSA between the sleep and waking states may be due to varying central control of respiration and HR and to the involvement of competing sources of

cardiac variation in the awake state. Harper also found that in contrast to the REM sleep and awake states, there were significant age differences in CSA in the quiet sleep state. There was a significant drop in CSA from 1 week to 1 month and then a significant increase from 1 to 4 months of age. Their measure of vagal tone (XSA) also showed a significant decline from 1 week to 1 month in the quiet sleep state. It was suggested that these decreases in CSA and XSA might be a response to the stress of immediate postnatal life. However, this does not explain why these differences are restricted to the quiet sleep state.

Orienting and Sustained Attention

The second part of the present study was concerned with the question of the utility of cardiac responses as measures of orienting and sustained attention in early infancy. The results of this part of the study suggest several provocative implications.

The results of the analyses of the HP per interval responses during the stimulus presentations replicated previous findings of a shift in the direction of HR responses in that from 1 to 3.5 months there was a significant decline in the proportion of infants exhibiting HR accelerative responses. However, the results did not support the hypothesis of increasing magnitude of decelerative responses over the same age period. The results of the analyses of the visual fixation responses and the HPV and RAV responses did not lend strong support for the hypothesis of an increase in the capacity to sustain attention over the first six postnatal months. However, the most provocative finding was that the HP per interval change scores were not correlated with the

visual fixation response measures. This finding suggests that the HR responses do not reflect the degree to which the infants were orienting and sustaining their visual fixations to the stimuli. In fact, none of the physiologic measures thought to index orienting or sustained attention were correlated with the visual fixation measures. To the extent that one is willing to grant that the visual fixation responses reflect the infants' orienting and sustained attention to the stimuli, the implication of these findings would be that these physiologic response measures do not appear to be particularily good indices of these psychological processes.

The alternative hypothesis that the direction and/or magnitude of these HR responses are dependent upon the development of vagal influences on the heart did not receive entirely convincing support either. Significant differences were not found between the high- and low-C_w groups for the visual fixation nor for the physiologic responses. Within the context of the present study these negative findings do not provide support for the continuity model as advanced by Porges (see Porges & Smith, Note 3). However, C_w was significantly correlated with mean first fixation times such that greater levels of mean C_w were associated with longer mean first fixation times. Since C_w was not significantly correlated with any of the physiologic response measures, it is tempting to speculate that perhaps this function is sensitive to centrally mediated processes such as those associated with orienting and sustained attention. Of course, the fact that C_w was not correlated with the other visual fixation measures certainly challenges this view.

The hypothesis that individual differences in the development of

the vagal control of the heart are related to individual differences in the cardiac responses thought to reflect orienting received only marginal support in that there was a tendency for peripheral vagal tone to be positively correlated ($p < .10$) with the HP per interval changes, such that greater levels of vagal tone were associated with greater decreases in HR (increases in HP). Interestingly enough, there was a tendency for the amount of RSA to be positively correlated ($p < .10$) with first fixation times and greater HP per interval changes such that greater levels of RSA were associated with longer mean first fixation times and greater increases in HP per interval scores. Since the amount of RSA is mediated by the CNS, these results provide limited support for the continuity hypothesis. Admittedly these correlations were low and do not account for much of the variance but at least they are consistent with the hypothesis.

Therefore, there appear to be two main conclusions which can be drawn from the results of the present study. The first is that during the first six months cardiac responses do not appear to be particularily good measures of orienting, nor for that matter, of sustained attention. Second, even though significant developmental changes in vagal influences on the heart were found during this age period, the evidence was not convincing with respect to the hypothesis that these developmental changes are related to developmental shifts in the direction of HR responses or to the magnitude of these HR responses. However, several methodological considerations suggest that these conclusions may be in need of qualification. First, although it seems reasonable to assume that visual fixation responses may be logical criterion variables against

which the validity of cardiac responses may be judged, they are not without problems. Second, the manner in which the stimuli were presented in this study may also undermine the above assumption. Regarding the first point, there is the problem of the "blank" or "empty" stare. The observers reported that on some trials even though the infants were looking at the stimulus they did not appear to be actively scanning it, i.e., there appeared to be few if any eye movements. The subjective impression was that while the infant's eyes have fixated the stimulus, they were not "processing" it. Thus similar fixation times may not reflect similar levels of orienting or sustained attention. The observers also reported that these "blank" stares seemed to occur more often with the younger infants. Unfortunately, the observers were not always aware of an infant's age nor were the fixation responses coded in a fashion which would allow an analysis of this potential confound. Lewis et al. (1966) suggested that "infants with equally long fixation times but who appear to be systematically scanning the stimulus are more likely to manifest cardiac deceleration" but he did not report the data upon which this suggestion was based. Nevertheless, future research should explore this problem and the possibility remains that blank stares may have contributed to the negative results of the present study.

Another methodological point has to do with the fixed duration of the stimulus presentations. Recall that it was pointed out in the results chapter that the infants only rarely fixated a stimulus immediately upon its presentation. Similarily, there were a fair number of trials in which the infants were fixating a stimulus when it

went off. Although this may have been related to the finding of no significant age differences in the HP responses to stimulus offset, it suggests that the infants' capcity to sustain attention may have been seriously underestimated. A more appropriate procedure would have been an "infant control technique" similar to that employed in recent infant habituation studies (see Cohen & Gelber, 1975; Horowitz, 1975). In this technique, a stimulus onset may be controlled by the experimenter (Horowitz, 1975) or by the infant (Cohen & Gelber, 1975) but the trial lasts as long as the infant fixates the stimulus, typically until there is a 1- or 2-sec period of no fixation of the stimulus. Combining this technique with one which would allow for coding of blank stares should provide a more adequate test of the possible relations between the behavioral and physiologic responses measured in this study as well as their possible relations to the measures of the development of the vagal influences on the heart.

APPENDICES

APPENDIX A

THE PORGES' WEIGHTED COHERENCE (C_w) FUNCTION

APPENDIX A

THE PORGES' WEIGHTED COHERENCE (C_w) FUNCTION

The C_w measure is based on the time series statistical procedure cross-spectral analysis. The C_w function is used to identify the percentage of spontaneous HR activity shared with respiration and, as such, is the operational definition of the relative parasympathetic-vagal influence on the heart or the relative degree of cardiac-respiratory coupling. The C_w function is defined by the following equation:

$$C_w = \frac{\sum\left[C^2_{(\lambda)}F_{H(\lambda)}\right]}{\sum\left[F_{H(\lambda)}\right]}$$

where

C^2 = coherence squared, an estimate of the shared variance of HR and respiration

F_H = the power density of HR (a measure of variance) at each frequency (λ) respiration normally occurs (e.g., in the adult human .2 to .5 Hz or 12 to 30 breaths a minute)

The product $C^2_{(\lambda)}F_{H(\lambda)}$ is summed over the dominant frequencies of respiration. The natural log of this product is viewed as the absolute amount of cardiac-respiratory coupling. In order to obtain a proportion of shared to total variance over the dominant frequency band of respiration, this quantity is divided by the total accumulation of the HR processes occurring within this band or $\sum\left[F_{H(\lambda)}\right]$. On the basis of pharmacologic and surgical studies with rats and rabbits (Porges, Note 6), Porges now considers the denominator of the above equation or $\sum\left[F_{H(\lambda)}\right]$ the operational definition of vagal tone or the absolute amount of vagal influences on cardiac pacemaking.

APPENDIX B

FORMS

MICHIGAN STATE UNIVERSITY

DEPARTMENT OF PSYCHOLOGY
OLDS HALL

EAST LANSING · MICHIGAN · 48824

Infant Learning Unit
Developmental Psychobiology Laboratory
Psychology Research Building

Dear Parents:

For the past several years we have been conducting research on how people attend and process information from their environment. In the very near future we will be starting a study with 1, 3½, and 6 month old infants. We will be examining the early development of attentional processes. We would like your permission for your baby to participate in this project.

The experiment itself will be quite simple. The infant will be seated in an infant seat in front of a screen. We will project a series of slides on the screen and measure your baby's reactions to these pictures. The pictures will be of human faces, landscape, and geometric objects. We will measure how much time your baby spends looking at the pictures as well as how your infant's heart rate and respiration changes while he/she is looking at the pictures.

In order to measure heart rate it will be necessary to attach three small surface sensors on your baby's chest. To measure respiration we will place a small infant rubber bellows around your baby's chest. These procedures are completely safe and painless, involving no discomfort or danger to your infant.

The experiment usually lasts about ten minutes. We ask that one parent accompany the infant to our laboratory on the MSU campus, and remain in the testing booth during the study. If at any time during the study you desire to end the testing we will do so. If you are interested in participating in this project, please sign the enclosed card. When we receive a signed permission card, we will contact you to set up an appointment and answer any additional questions you may have.

Thank you very much for your time and interest. We look forward to hearing from you in the near future.

Sincerely,

Patrick K. Ackles, Research Assistant

Hiram E. Fitzgerald, Ph.D., Professor of Psychology

PERMISSION FORM FOR TESTING INFANT

Date: _____

Dear Parents:

This form is to request permission for us or members of our staff to examine your infant in tests of attention. The tests have have been approved by Michigan State University's Human Subjects Committee and will help us to understand normal development of infant attention.

You may withdraw permission at any time simply by informing us or our staff members that you wish to do so. The information collected is confidential; it will be available only to qualified personnel, and information on individual infants is identified by number only. If you have any questions about the procedures to be used, please feel free to ask. The tests will not disrupt the infant or in any way be harmful; however, participation in the study will not guarantee you or your infant any beneficial results.

Your signature on this form verifies that the specific tests and procedures to be used with your infant have been explaind to your satisfaction, and that you have voluntarily agreed to allow us to test your infant. If at any time you wish to have the data from your baby withdrawn from the experiment, simply advise us and we will destroy all records relevant to your baby.

Sincerely,

Patrick K. Ackles
Research Director

Hiram E. Fitzgerald, Ph.D.
Professor of Psychology

_____

Parent's signature

_____

Experimenter's signature

FOLLOW-UP INFORMATION

The experiment your infant is participating in is part of a larger project on the mental development of children. Our work is routinely reported in various professional journals and we like to have the parents of the children participating aware of the kind of work we are doing. Thus, if you would like to receive follow-up information on the results of the experiment in which your infant participated, check the line below and provide a mailing address that will be good for the next twelve to twenty-four months.

_____ Please send a general summary of the findings (available within about four to nine months)

Mailing address:

Name _____

Address _____

City or town _____

State _____ Zip _____

BACKGROUND INFORMATION SHEET

The information requested in this form will be used to report the general charateristics of the infants used in our research. Only groups results will be reported, and the identity of individual infants will remain anonymous. All information you provide on this form will be kept strictly confidential.

Subject Number _____ Slide Order _____

Date of Test _____ Time of Test _____ A.M. P.M. (circle)

Tape Number _____

Experimenters:

Date of Birth _____ Sex: Male ___ Female ___
Month Day Year

Place of Birth _____
City or Town State Country

Weight at Birth _____ lb. _____ oz. Length at birth _____ inches

Due Date_____ Present Weight _____lb. _____oz.

Present length _____ inches

Any complications during pregnancy?_____ If so, please briefly describe them.

Was medication used during labor and/or delivery (for example, local anesthetic, general anesthetic, gas, saddle block)?_____ If so, please describe briefly.

Is your infant _____breast fed _____ bottle fed

_____ some combination, with bottle feeding____75%___50%___25%

Has your infant had any prolonge or general illness since birth? _____
If so, please describe briefly.

Any special problems with ( ) colic, ( ) rashes, ( ) feeding, ( ) sleeping.
If so, please describe briefly.

Is there anything else special about your infant that you think it would be important for us to know about for this project? _____ If so, please describe.

APPENDIX C

ANALYSES OF VARIANCE SUMMARY TABLES

Table 13

Analysis of Variance Summary Table for Baseline Mean Heart Period (msec)

Source	df	MS	F	p
Between Subjects				
Age (A)	2	6659.785	3.49	.043
Sex (B)	1	8.134	.004	ns
A x B	2	2333.510	1.22	ns
Sub. w. gps.	30	1906.309		
Within Subjects				
Baseline (C)	1	1.681	.011	ns
A x C	2	38.961	.151	ns
B x C	1	876.409	3.41	.07
A x B x C	2	65.223	.25	ns
C x Sub. w. gps.	30	256.709		

Table 14

Analysis of Variance Summary Table for Baseline ln Heart Period Variance

Source	df	MS	F	p
Between Subjects				
Age (A)	2	2.1906	1.68	ns
Sex (B)	1	1.2086	.93	ns
Sub. w. gps.	30	1.3064		
Within Subjects				
Baseline (C)	1	.1036	.29	ns
A x C	2	.2025	.56	ns
B x C	1	.4497	1.24	ns
A x B x C	2	.1735	.48	ns
C x Sub. w. gps.	30	.3631		

Table 15

Analysis of Variance Summary Table for C_w

Source	df	MS	F	p
Between Subjects				
Age (A)	2	.023611	1.11	ns
Sex (B)	1	.001830	.10	ns
A x B	2	.017517	.95	ns
Sub. w. gps.	30	.018389		
Within Subjects				
Baseline (C)	1	.001005	.11	ns
A x C	2	.012047	1.26	ns
B x C	1	.000210	.02	ns
A x B x C	2	.000766	.08	ns
C x Sub. w. gps.	30	.009548		

Table 16

Analysis of Variance Summary Table for ln Amount of Heart Period Variance Shared with Respiration

Source	df	MS	F	p
Between Subjects				
Age (A)	2	8.1047	5.26	.011
Sex (B)	1	.9500	.61	ns
A x B	2	.2855	1.85	ns
Sub. w. gps.	30	.153969	.02	
Within Subjects				
Baseline (C)	1	.0143	.02	ns
A x C	2	2.3806	2.93	.07
B x C	1	.7246	.89	ns
A x B x C	2	.5367	.66	ns
C x Sub. w. gps.	30	.811978		

Table 17

Analysis of Variance Summary Table for ln Amount of Heart Period Variance Shared and Not Shared with Respiration

Source	df	MS	F	p
Between Subjects				
Age (A)	2	12.8312	10.93	.0003
Sex (B)	1	.2542	.22	ns
A x B	2	4.4349	3.78	.034
Sub. w. gps.	30	1.1743		
Within Subjects				
Baseline (C)	1	.06171	.14	ns
A x C	2	1.1107	2.53	ns
B x C	1	.7975	1.82	ns
A x C	2	.3669	.83	ns
C x Sub. w. gps.	30	.4376		

Table 18

Analysis of Variance Summary Table for
Baseline Peak Respiratory Frequency

Source	df	MS	F	p
Between Subjects				
Age (A)	2	.4432	3.81	.034
Sex (B)	1	.2048	1.76	ns
A x B	2	.2035	1.75	ns
Sub. w. gps.	30	.1165		
Within Subjects				
Baseline (C)	1	.0089	.18	ns
A x C	2	.0161	.33	ns
B x C	1	.1881	3.85	.059
A x B x C	2	.0774	1.58	ns
C x Sub. w. gps.	30	.0489		

Table 19

Analysis of Variance Summary Table for
Baseline Peak Heart Period Frequency

Source	df	MS	F	p
Between Subjects				
Age (A)	2	.231822	3.26	.052
Sex (B)	1	.032089	.45	ns
A x B	2	.0952889	1.34	ns
Sub. w. gps.	30	.071164		
Within Subjects				
Baseline (C)	1	.096800	1.62	ns
A x C	2	.055467	.93	ns
B x C	1	.000089	.002	ns
A x B x C	2	.176356	2.94	.07
C x Sub. w. gps.	30	.059929		

Table 20

Analysis of Variance Summary Table for Total Fixation Time (sec)

Source	df	MS	F	p
Between Subjects				
Age (A)	2	38.7935	1.41	ns
Sex (B)	1	10.2704	.37	ns
A x B	2	10.7168	.39	ns
Sub. w. gps.	30	27.5937		
Within Subjects				
Stimulus (C)	5	41.7745	6.01	.00004
A x C	10	15.5849	2.24	.018
B x C	5	9.9396	1.45	ns
A x B x C	10	5.4330	.78	ns
C x Sub. w. gps.	150	6.9465		

Table 21

Analysis of Variance Summary Table for Mean Fixation Time (sec)

Source	df	MS	F	p
Between Subjects				
Age (A)	2	105.7211	3.17	.057
Sex (B)	1	2.9822	.09	ns
A x B	2	4.1129	.12	ns
Sub. w. gps.	30	33.3961		
Within Subjects				
Stimulus (C)	5	23.7610	2.27	.051
A x C	10	22.2959	2.13	.026
B x C	5	12.5037	1.19	ns
A x B x C	10	11.6985	1.12	ns
C x Sub. w. gps.	150	10.4906		

Table 22

Analysis of Variance Summary Table for First Fixation Time (sec)

Source	df	MS	F	p
Between Subjects				
Age (A)	2	50.7246	1.39	ns
Sex (B)	1	1.2513	.03	ns
A x B	2	11.4397	.31	ns
Sub. w. gps.	30	36.4586		
Within Subjects				
Stimulus (C)	5	36.9232	3.19	.009
A x C	10	16.5257	1.43	ns
B x C	5	20.0962	1.73	ns
A x B x C	10	11.1113	.96	ns
C x Sub. w. gps.	150	11.5924		

Table 23

Analysis of Variance Summary Table for Log_{10} (x + 1) Number of Fixations

Source	df	MS	F	p
Between Subjects				
Age (A)	2	.12978	3.08	.06
Sex (B)	1	.00264	.06	ns
A x B	2	.00083	.02	ns
Sub. w. gps.	30	.04217		
Within Subjects				
Stimulus (C)	5	.02774	1.51	ns
A x C	10	.01545	.84	ns
B x C	5	.01781	.97	ns
A x B x C	10	.01801	.09	ns
C x Sub. w. gps.	150	.01832		

Table 24

Analysis of Variance Summary Table for Heart Period (msec) Difference Scores by 500 msec Intervals

Source	df	MS	F	p
Between Subjects				
Age (A)	2	29297.64	1.90	ns
Sex (B)	1	88425.06	5.74	.02
A x B	2	6443.00	.42	ns
Sub. w. gps.	30	15395.95		
Within Subjects				
Stimulus (C)	5	7591.71	.54	ns
A x C	10	10298.99	.73	ns
B x C	5	14316.85	1.01	ns
A x B x C	10	9083.84	.64	ns
C x Sub. w. gps.	150	14158.59		
Period (D)	2	11856.49	3.93	.02
A x D	4	884.03	.29	ns
B x D	2	1619.86	.53	ns
A x B x D	4	3112.05	1.03	ns
D x Sub. w. gps.	60	3016.32		
Interval (E)	11	1220.68	11.40	<.0001
A x E	22	150.76	1.41	ns
B x E	11	218.83	2.04	ns
A x B x E	22	137.28	1.28	ns
E x Sub. w. gps.	330	107.07		
C x D	10	2257.23	.85	ns
A x C x D	20	1974.93	.74	ns
B x C x D	10	3533.40	1.33	ns
A x C x D	20	1302.02	.49	ns
CD x Sub. w. gps.	300	2651.98		
C x E	55	76.67	.70	ns
A x C x E	110	106.08	.96	ns
B x C x E	55	68.44	.62	ns
A x B x C x E	110	109.57	.99	ns
CE x Sub. w. gps.	1650	110.25		
D x E	22	438.01	2.54	.0001
A x D x E	44	194.71	1.13	ns
B x D x E	22	342.58	1.99	.005
A x B x D x E	44	464.31	2.69	<.0001
DE x Sub. w. gps.	660	172.53		
C x D x E	110	185.24	1.20	.08
A x C x D x E	220	198.38	1.29	.003
B x C x D x E	110	200.01	1.30	.02
A x B x C x D x E	220	113.36	.74	ns
CDE x Sub. w. gps.	3300	153.86		

Table 25

Analysis of Variance Summary Table for the Magnitude of the Heart Period per Interval Change Scores (msec)

Source	df	MS	F	p
Between Subjects				
Age (A)	2	7459.93	8.32	.01
Sex (B)	1	6337.50	7.07	.05
A x B	2	1342.01	1.50	ns
Sub. w. gps.	30	896.84		
Within Subjects				
Stimulus (C)	5	976.97	<1	ns
A x C	10	2100.48	1.77	ns
B x C	5	994.27	<1	ns
A x B x C	10	852.93	<1	ns
C x Sub. w. gps.	150	1188.78		

Table 26

Analysis of Variance Summary Table for Mean Heart Period (msec)

Source	df	MS	F	p
Between Subjects				
Age (A)	2	668862.79	4.43	.02
Sex (B)	1	7814.04	.50	ns
A x B	2	4806.53	.31	ns
Sub. w. gps.	30	15558.69		
Within Subjects				
Stimulus (C)	5	1722.13	2.62	.03
A x C	10	1677.35	2.55	.007
B x C	5	639.50	.97	ns
A x B x C	10	924.71	1.41	ns
C x Sub. w. gps.	150	656.57		
Period (D)	3	1203.22	5.03	.003
A x D	6	362.98	1.52	ns
B x D	3	398.46	1.67	ns
A x B x D	6	191.58	.80	ns
D x Sub. w. gps.	90	239.00		
C x D	15	179.93	.72	ns
A x C x D	30	203.66	.67	ns
B x C x D	15	279.06	.28	ns
A x B x C x D	30	152.49	.92	ns
CD x Sub. w. gps.	450	234.66		

Table 27

Analysis of Variance Summary Table for Log_{10} Heart Period Variance (msec)

Source	df	MS	F	p
Between Subjects				
Age (A)	2	3.4438	1.56	ns
Sex (B)	1	.6790	.31	ns
A x B	2	.2762	.12	ns
Sub. w. gps.	30	2.2137		
Within Subjects				
Stimulus (C)	5	.2958	.74	ns
A x C	10	.1816	.45	ns
B x C	5	.5382	1.34	ns
A x B x C	10	.3415	.85	ns
C x Sub. w. gps.	150	.4009		
Period (D)	3	.8853	3.38	.02
A x D	6	.1509	.58	ns
B x D	3	.3360	1.28	ns
A x B x D	6	.3780	1.45	ns
x Sub. w. gps.	90	.2621		
C x D	15	.1648	.76	ns
A x C x D	30	.2385	1.11	ns
B x C x D	15	.2523	1.17	ns
A x B x C x D	30	.2108	.98	ns
CD x Sub. w. gps.	450	.2156		

Table 28

Analysis of Variance Summary Table for Log_{10} Respiratory Amplitude Variance

Source	df	MS	F	p
Between Subjects				
Age (A)	2	11.5507	2.65	.09
Sex (B)	1	.0026	.98	ns
A x B	2	5.3013	1.22	ns
Sub. w. gps.	30	4.3619		
Within Subjects				
Stimulus (C)	5	.8465	2.06	.07
A x C	10	.5743	1.40	ns
B x C	5	.6225	1.52	ns
A x B x C	10	.7388	1.80	.07
C x Sub. w. gps.	150	.4106		
Period (D)	3	.5455	2.97	.04
A x D	6	.3150	1.72	ns
B x D	3	.1990	1.09	ns
A x B x D	6	.3688	2.01	.07
D x Sub. w. gps.	90	.1834		
C x D	15	.1794	.87	ns
A x C x D	30	.1605	.78	ns
B x C x D	15	.1853	.90	ns
A x B x C x D	30	.1580	.77	ns
CD x Sub. w. gps.	450	.2055		

Table 29

Analyses of Variance Summary Table for Total Fixation Time (sec) for Median C_W Groups

Source	df	MS	F	p
Between Subjects				
C_W (A)	1	1.98	<1	ns
Sub. w. gps.	34	27.50		
Within Subjects				
Stimulus (B)	5	41.77	5.61	.0001
A x B	5	7.22	<1	ns
B x Sub. w. gps.	170	7.45		

Table 30

Analysis of Variance Summary Table for Mean Fixation Time (sec) for Median C_w Groups

Source	df	MS	F	p
Between Subjects				
C_w (A)	1	.56	<1	ns
Sub. w. gps.	34	35.99		
Within Subjects				
Stimulus (B)	5	23.76	2.08	.07
A xB	5	6.00	<1	ns
B x Sub. w. gps.	170	11.45		

Table 31

Analysis of Variance Summary Table for First Fixation Time (sec) for Median C_w Groups

Source	df	MS	F	p
Between Subjects				
C_w (A)	1	10.50	<1	ns
Sub. w. gps.	34	35.55		
Within Subjects				
Stimulus (B)	5	36.92	3.04	.012
A x B	5	10.17	<1	ns
B x Sub. w. gps.	170	12.14		

Table 32

Analysis of Variance Summary Table for Number of Fixations For Median C_w Groups

Source	df	MS	F	p
Between Subjects				
C_w (A)	1	.0001	<1	ns
Sub. W. gps.	34	.0450		
Within Subjects				
Stimulus (B)	5	.0277	1.52	ns
A x B	5	.0143	<1	ns
B x Sub. w. gps.	170	.0182		

Table 33

Analysis of Variance Summary Table for Heart Period (msec) Difference Scores by 500 msec Intervals for Median C_w Groups

Source	df	MS	F	p
Between Subjects				
C_w (A)	2	14536.96	.81	ns
Sub. w. gps.	34	17860.23		
Within Subjects				
Stimulus (B)	5	7591.71	.56	ns
A x B	5	16747.66	1.23	ns
B x Sub. w. gps.	170	13561.57		
Period (C)	2	11856.49	4.10	.02
A x C	2	1823.25	.63	ns
C x Sub. w. gps.	68	2890.54		
Interval (D)	11	1220.68	10.43	.0001
A x D	11	27.80	.24	ns
D x Sub. w. gps.	374	117.04		
B x C	10	2257.23	.91	ns
A x B x C	10	4957.33	1.99	.03
BC x Sub. w. gps.	340	2490.86		
B x D	55	76.67	.72	ns
A x B x D	55	203.67	1.92	.0001
BD x Sub. w. gps.	1870	105.99		
C x D	22	438.01	2.21	.001
A x C x D	22	99.28	.50	ns
CD x Sub. w. gps.	748	198.16		
B x C x D	110	185.24	1.19	ns
A x B x C x D	110	153.40	.99	ns
BCD x Sub. w. gps.	3740	155.47		

Table 34

Analysis of Variance Summary Table for the Magnitude of Heart Period/Interval for Median C_w Groups (msec)

Source	df	MS	F	p
Between Subjects				
C_w (A)	1	384.00	<1	ns
Sub. w. gps.	34	1484.20		
Within Subjects				
Stimulus (B)	5	976.97	<1	ns
A x B	5	1611.37	1.34	ns
B x Sub. w. gps.	170	1204.50		

Table 35

Analysis of Variance Summary Table for Mean Heart Period (msec) for Median C_w Groups

Source	df	MS	F	p
Between Subjects				
C_w (A)	1	33255.99	1.92	ns
Sub. w. gps.	34	17313.45		
Within Subjects				
Stimulus (B)	5	1722.13	2.36	.04
A x B	5	712.70	.98	ns
B x Sub. w. gps.	170	730.23		
Period (C)	3	1203.22	4.82	.004
A x C	3	189.19	.76	ns
C x Sub. w. gps.	102	249.66		
B x C	15	179.93	.80	ns
A x B x C	15	395.67	1.76	.04
BC x Sub. w. gps.	510	224.57		

Table 36

Analysis of Variance Summary Table for Log_{10} Heart Period Variance for Median C_w Groups

Source	df	MS	F	p
Between Subjects				
C_w (A)	1	4.8358		
Sub. w. gps.	34	2.0498		
Within Subjects				
Stimulus (B)	5	.2958	.76	ns
A x B	5	.3648	.94	ns
B x Sub. w. gps.	170	.3896		
Period (C)	3	.8853	3.33	.02
A x C	3	.2206	.83	ns
C x Sub. w. gps.	102	.2659		
B x C	15	.1648	.77	ns
A x B x C	15	.3064	1.40	ns
BC x Sub. w. gps.	510	.2152		

Table 37

Analysis of Varince Summary Table for Log_{10} Respiratory Amplitude Variance for Median C_w Groups

Source	df	MS	F	p
Between Subjects				
C_w (A)	1	.0576	1	ns
Sub. w. gps.	34	4.9259		
Within Subjects				
Stimulus (B)	5	.9003	1.97	ns
A x B	5	.3731	1	ns
B x Sub. w. gps.	170	.4562	1	
Period (C)	3	.5947	2.84	.04
A x C	3	.0594	1	ns
C x Sub. w. gps.	102	.2095		
B x C	15	.1830	1	ns
A x B x C	15	.2499	1.23	ns
BC x Sub. w. gps.	510	.2032		

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