THE RELATION OF SPONTANEOUS STARTLES TO CARDIAC AND RESPIRATORY ACTIVITY IN NEWBORN INFANTS

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(ABSTRACT)

Previous studies have reported that spontaneous startles occur most frequently in the quiet sleep states, and have posited an energy release model in which spontaneous startles occur to release energy which would otherwise wake the infant. An alternative suggestion is that startles serve a homeostatic function by increasing the activity of the infant during periods of low arousal. The purpose of the current study was to examine the function of spontaneous startles using the ongoing cardiac and respiratory activity as indices of arousal.

Twenty-six newborn infants were assigned to two groups. The first group was exposed to auditory stimulation which previously had been shown to decrease heart rate and respiratory rate for the first half of the one hour observation period. To the extent that spontaneous startles are related to periods of low arousal, decreasing the ongoing activity via the auditory intervention was expected to increase the rate of startles. The second group received no auditory stimulation.

The occurrence of spontaneous startles was preceded by

periods of lower than average heart rate and decreasing respiratory rate. Startles were followed by periods of increasing heart rate and further decreasing respiratory rate. In addition, the auditory intervention group reliably showed both a lower heart rate and an increased number of startles while exposed to the auditory stimulation, while the nonintervention group showed comparable rates of startles and heart rates in both halves of the observation period. Further, regardless of group status, most infants had their higher rate of startles in the period in which they had their lower heart rate. Finding lower heart rate and decreasing respiratory rate preceding startles, and lower heart rate and increased number of startles when exposed to the auditory stimulation, suggests that spontaneous startles modulate periods of low arousal in newborn infants.

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Finally, I would like to thank my parents,

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. To these people, I dedicate this paper.

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Introduction

Spontaneous behaviors, those behaviors which occur in the absence of any obvious external eliciting stimuli (Wolff, 1966), have been a subject of study for many years (Gilmer, 1933; Irwin, 1930; Preyer, 1880, in Kessen, Haith, & Salapatek, 1970). Some spontaneous behaviors, such as rapid eye movements (REM) and crying, have been used to define particular biobehavioral states (Wolff, 1966). Others, such as smiling, have been studied for the information which they can provide about the infant's development. For example, Emde and Gaensbauer (1981) based a model of infant emotional development on the change from spontaneous smiling in the newborn period to elicited smiling at two to three months of age.

Another perspective has emphasized the importance of studying spontaneous behaviors for their effects on the adaptation of the infant during the newborn period. Woodson (1982) suggested that the behavior an infant displays, whether in response to, or in the absence of external stimulation, plays an important part in the infant's adaptation to the extrauterine environment. Stratton (1982) discussed the importance of considering the energy requirements of various behaviors in relation to the benefits produced by the emission of those behaviors. Crying, for example, is a behavior which requires an energy

expenditure, but which also provides energy returns through attracting the attention of caregivers (Bell & Ainsworth, 1972; Bernal, 1972).

One spontaneous behavior is the spontaneous startle, a sudden, full-body motor movement resembling the Moro reflex which has been reported to occur in the absence of a specific external stimulus (Wolff, 1966). Spontaneous startles "begin suddenly from a position of rest as an abduction at the shoulders, an extension of the arms at the elbows and wrists, an abduction and dorsiflexion of the fingers at the carpal and metacarpal joints, a palmar flexion at the distal phalanges, an arching of the back and in some cases, a hyperextension of the head. After the initial extension movements, the arms slowly return to a partially abducted, partially flexed position, the hands relax into a partial fist, and the trunk and head resume their resting position" (Wolff, 1966; pp. 12-13). Korner (1969) described startles more briefly as "spontaneously occurring massive body jerks which resemble the Moro reflex and involve both sudden extensor and flexor movements of the limbs" (p. 104). While the studies of Wolff (1966) and Korner (1969) provided descriptions of the pattern of occurrence of spontaneous startles, little is known about their functional significance or the neural mechanisms which control them. For example, although they are referred to as

spontaneous startles, might there not be some internal stimulus which "evokes" them, thus making the distinction of spontaneous versus evoked unnecessary? If so, what is the significance of their occurrence in relation to the ongoing activity of the infant?

The purpose of this study was to examine the functional significance of spontaneous startles by observing the relation of spontaneous startles to ongoing cardiac and respiratory activity. The rationale for this study will be presented in the following sections. First, the literature review will describe the similarities between spontaneous startles, evoked startles, and the Moro response. Second, psychophysiological responses associated with evoked startles will be discussed in terms of the information that they can contribute to our understanding of spontaneous startles. Third, the relation of infant heart-rate responses to behavioral state will be discussed, as this relation might affect the function of spontaneous startles in different states. Fourth, previously proposed models of the function of spontaneous startles will be discussed, and an extension of these models relating spontaneous startles to the homeostatic activity of the infant will be proposed. Fifth, and finally, the specific hypotheses of this study will be presented.

The relation of spontaneous startles to evoked startles and

the Moro reflex

Similarities between spontaneous startles, startles evoked by loud, sudden onset auditory stimuli, and the Moro response have been noted at several levels. At the first level, spontaneous startles, evoked startles, and the Moro reflex share a striking similarity of appearance (Hunt, Clarke, & Hunt, 1936; Korner, 1969; Wolff, 1966). Hunt et al. (1936) described the evoked startle as similar in appearance to the Moro reflex, with both involving abduction of the upper arms, contraction of the abdomen, and bending of the knees, and suggested that the startle might be a later developmental form of the Moro reflex. Both Wolff (1966) and Korner (1969) described spontaneous startles in relation to the appearance of the Moro reflex.

At a second level, while the spontaneous startle can be distinguished from the evoked startle and the Moro reflex by the apparent lack of external eliciting stimuli, there appears to be some functional overlap between spontaneous and evoked startles. Wolff (1966) reported that the elicitation of a startle delayed the occurrence of a spontaneous startle, and conversely, the occurrence of a spontaneous startle made the elicitation of a startle more difficult. Finally, both the spontaneous startle and the Moro reflex show a similar developmental course, disappearing by the second to third month of life (McGraw,

1933; Wolff, 1966). Because of the similarities between spontaneous startles, evoked startles, and the Moro reflex, the results of studies of the psychophysiological responses to both startle and nonstartle eliciting stimuli may contribute to our understanding of the mechanisms and functions of spontaneous startles.

Psychophysiological responses to startle evoking stimuli

Studies investigating startle responses have demonstrated specific stimulus characteristics which reliably differentiate startle evoking stimuli (Graham, 1979). Fleschler (1965) demonstrated that the rise time, the rate of increase in the intensity of a stimulus, is critical in eliciting whole-body startle in rats, with 10- to 12-msec the optimal rise time. Berg (1973), using the blink response, demonstrated that the rise time of the stimulus is negatively correlated with the intensity required to elicit a startle response in humans. A 1 kHz tone of 87 dB with a rise time of less than 5 μmsec elicited a startle, whereas with a 30 msec rise time, an intensity of 96 dB was required.

The behavioral responses to intense, sudden stimuli are reliably paralleled by heart rate (HR) responses, which can be differentiated from responses to less intense and less sudden stimuli. Hatton, Berg, and Graham (1970) found that the direction of the HR response was dependent on the rise

time of the stimulus. A 90 dB tone elicited a deceleratory response followed by an acceleration after approximately 2 seconds. The same intensity tone with a 5 μmsec rise time elicited a large, short-latency acceleratory response. Hatton et al. (1970) suggested that this HR response was a component of the startle response and was dependent on the sudden stimulus onset, characterized by high-intensity, transient noise. Graham and Slaby (1973) supported this suggestion by demonstrating that an 85 dB white noise stimulus, beginning with high intensity transients, elicited a large HR acceleration accompanied by a gross body jerk, while an equal intensity tone without transients elicited an initial HR deceleration. The HR acceleration and the visible startle response showed a rapid decrement. This result is consistent with the report of Graham, Putnam, and Leavitt (1975) that the whole-body startle habituates rapidly, while portions of the response, such as the eyeblink, decrease more slowly. These results indicate a reliable parallel between the occurrence of the whole-body startle and HR accelerations.

Just as the whole-body startle differentiates the response to sudden intense stimuli, HR change reliably distinguishes between startle responses and responses to other stimuli. Graham (1979) reviewed unpublished evidence of the differences in these HR responses. Low or moderate

intensity stimuli, around 50 dB, elicit HR decelerations. Higher intensity stimuli, around 90 dB, with rise times greater than 30 msec elicit a biphasic response with an initial short deceleratory component followed by a longer latency, large acceleratory component. These responses are differentiated from the short-latency acceleratory response to high-intensity, short rise time stimuli (Graham, 1979). These responses are also differentiated by the pattern of response to repeated stimulation. Both the HR deceleration to low and moderate intensity stimuli and the short-latency acceleratory response to short rise time stimuli show rapid habituation over repeated stimulus presentations. The response to the high-intensity, long rise time stimuli, however, shows a sensitization effect over repeated presentations, with the initial deceleration disappearing and the late acceleration becoming predominant (Graham, 1979).

These differential HR responses have been viewed within the framework of an attentional theory (Sokolov, 1963),which posits an orienting reflex (OR), occurring in response to low- to moderate-intensity stimuli or changes in stimulus characteristics and facilitating the organism's receptivity to stimuli, and a defensive reflex (DR), elicited by intense or painful stimuli, and decreasing the organism's sensitivity. Graham and Clifton (1966) related Sokolov's

theory to work by Lacey (Lacey, 1959; Lacey & Lacey, 1958) which indicated that increases in HR and blood pressure were related to inhibition of cortical activity, rather than facilitation of this activity. Graham and Clifton (1966) suggested that HR accelerations and decelerations might be related to the OR and DR, respectively. Lacey and Lacey (1967, 1970) proposed that the mechanism of this effect is the cortical inhibitory effect of stimulation of the carotid baroreceptors, a mechanism which has since been substantiated (Joy, 1975).

While this theory has affected the direction of psychophysiological research, it is not entirely accepted. Obrist (1976, 1981, 1982), for example, has repeatedly emphasized the oversimplification of assuming that the primary purpose of cardiovascular responses is the modulation of cognitive activity, while overlooking the importance of metabolic and homeostatic inputs to cardiac functions. Obrist, Langer, Grignolo, Light, and McCubbin (1980) suggested that the HR deceleration found concomitant with the orienting response might be related to the cessation of bodily activity which is also associated with orienting. Conversely, the HR acceleration found concomitant with the defensive response might be related more to increased muscular activity and oxygen consumption than to inhibition of cortical activity.

Obrist et al. (1980) also suggested that the HR acceleration is not merely a byproduct of the concurrent muscular activity, but is mediated by a parallel, central, system. This suggestion is supported by the work of Freychuss (1970), which demonstrated that the intent to grasp an object was accompanied by HR accelerations in subjects whose grasp was inhibited by neuromuscular blockade. Clearly, there is still some debate as to the functional significance of these HR changes. Thus, for the purposes of this study, the HR changes will be referred to descriptively as acceleratory and deceleratory changes, without reference to the theoretical constructs of orienting and defensive reflexes.

While studies of adults' behavioral and cardiac responses have found different responses to different types of stimuli, early studies of infants' responses indicated that newborns typically show acceleratory HR responses to stimulation with no decrease below baseline (Bridger & Reiser, 1959; Graham, Clifton, & Hatton, 1968; Lipton & Steinschneider, 1964). Bridger and Reiser (1959) demonstrated that the behavioral startle response to an intense air puff was reliably accompanied by HR acceleration. Graham et al. (1968), in a study of 10 newborns, found a significant HR acceleration to a 70 dB stimulus which would not elicit a startle response in an

adult subject. Lipton and Steinschneider (1964) demonstrated that the adult type responses develop between two and three months of age. In one of the few studies that have examined respiratory responses to stimuli, Steinschneider (1968) found that the infant's initial response was a slowing of respiration, followed by a significant increase in respiratory rate. Graham et al. (1968) suggested that a possible explanation for the predominance of acceleratory responses in infants is the relative immaturity of higher brain structures. One factor which these early studies overlooked, however, was the effect of behavioral state on infants' responses to stimulation.

The relation of infant state to behavioral and cardiac responses to stimulation

The concept of infant state has developed over the last twenty-five years as a framework for the organization of information pertaining to infant behavior (Prechtl, 1982). Behavioral and psychophysiological observations have revealed cyclic patterns within the behavior of infants (Aserinsky & Kleitman, 1955; Korner, 1969; Petre-Quadens, 1967; Parmelee, Wenner, Akiyama, Schultz, & Stern, 1967; Roffwarg, Muzio, & Dement, 1966; Thoman, 1975; Wolff, 1966). For example, Aserinsky and Kleitman (1955) described two distinct sleep states, REM and non-REM sleep, in human infants. Petre-Quadens (1967) traced the development of

these states between the 28th week of gestation and one month post-term, showing that the ratio of non-REM to REM sleep increased across this period, at the same time as the ratio of sleep to waking was decreasing. Jouvet-Mounier, Astic, and Lacote (1970) employed both ontogenetic and phylogenetic comparisons to relate this trend to the maturity of the developing organism, reporting that those species and individuals which are relatively mature at birth show higher ratios of non-REM to REM sleep.

Behavioral observations have aided in further subdividing the sleep periods. Wolff (1966) employed a state scale which divided sleep into regular sleep, irregular sleep, and periodic sleep. Korner (1969) employed much the same scale, with the inclusion of periodic sleep in the active sleep state, and the addition of an indefinite state for those periods in which an infant could not be classified as sleeping, awake, or drowsy. The distinction between the sleep states on these state scales is based on the amount of gross bodily movement, regularity of respiration, and occurrence of REM activity. Quiet sleep is characterized by regular respiration and absence of gross body movement or REM activity, and active sleep is characterized by irregular respiration and either gross body movement or REM activity (Korner, 1969; Wolff, 1966). These state scales are represented in Table 1.

In addition to the subdivision of the sleep states, the waking period has been divided into states which vary in the quality and quantity of the observed activity. Alert inactivity is characterized by open "shining" eyes, visual pursuit of objects, and relatively little body movement (Wolff, 1966). The characteristics of waking activity include brief periods of diffuse motor activity, sobs or groans, and highly irregular respiration (Wolff, 1966). The defining criterion for the state of crying is the crying vocalization, which is accompanied by vigorous, diffuse motor activity (Wolff, 1966). During the drowsy state, the infant is relatively inactive, the eyes have a dull, glazed appearance, and the eyelids appear heavy (Wolff, 1966). These definitions correspond to those employed by Korner (1969), with the addition that the indeterminate state was defined as any period "in which the infant's state did not clearly meet the criteria of any of the above states" (p 1044).

Thoman (1975) further subdivided both the sleep and waking states in order to examine individual differences in the state regulation of infants. Quiet sleep was divided into quiet sleep A and quiet sleep B, distinguished from each other by depth and regularity of respiration, with respiration in quiet sleep A being more regular. Active sleep was subdivided into active sleep without REM and two

REM activity states, active sleep with light REM and active sleep with dense REM. Thoman (1975) continued to use Wolff's (1966) and Korner's (1969) definitions for the states of drowse, alert inactivity, waking activity and the indefinite state. The crying state was divided into fussing, characterized by motor activity and vocalizations but not including sustained crying vocalizations, and crying, characterized by sustained crying vocalizations. A comparison of Thoman's 11-point state scale with the scales of Wolff (1966) and Korner (1969) is included in Table 1.

Studies of the relation of state to psychophysiological activity indicate that HR and respiratory rate vary between states (Ashton & Connolly, 1971; Campos & Brackbill, 1973; Harper, Hoppenbrowers, Sterman, McGinty, & Hodgeman, 1976), with higher means and increased variability in the "higher" states. The Law of Initial Values (LIV) (Wilder, 1958), states that the level of the prestimulus HR can affect both the amplitude and the direction of the poststimulus response, with large, acceleratory responses found when prestimulus HR is low and large deceleratory responses found to the same stimulus when HR is high. For example, Campos and Brackbill (1973) examined the effect of between-state HR differences on the HR response to an 85 dB white noise stimulus. The HR response in all states was acceleratory, but was larger in active sleep than in the quiet awake

Table 1

The correspondence between state scales

	Author	
Type of State	Korner (1969)	Thoman (1975)
Sleep	Regular Sleep	Quiet Sleep A Quiet Sleep B
	Irregular Sleep	Active Sleep (No REM) Light REM Dense REM
Transition	Drowsiness	Drowsy
	Alert Inactivity	Alert Inactivity
Waking	Waking Activity	Waking Activity
	Crying	Fussing Crying
	Indeterminate	Indefinite

Note: Because of the similarity between the scales employed by Wolff (1966) and Korner (1969), only Korner's scale is represented here.

state. Campos and Brackbill (1973) stated that a significant amount of the variance in HR response between the two states was accounted for by the prestimulus HR levels. When prestimulus levels were controlled, the active sleep response was still larger than the quiet awake response.

While early studies of neonatal responses to stimulation revealed no deceleratory HR responses (Bridger & Reiser, 1959; Graham, Clifton, & Hatton, 1968; Lipton & Steinschneider, 1964), more recent studies have employed distinctions in state behavior to demonstrate that the infant's response to stimulation is highly dependent on the infant's state (Adkinson & Berg, 1976; Jackson, Kantowitz, & Graham, 1971; Pomerleau-Malcuit & Clifton, 1973). Jackson et al. (1971) found that when moderate intensity stimuli were presented to infants without monitoring state, HR showed significant acceleratory responses, while presentation to awake infants did not elicit clear changes in either direction. Pomerleau-Malcuit and Clifton (1973) demonstrated deceleratory responses to auditory and vestibular stimulation when infants were awake, but unfed. Adkinson and Berg (1976) also showed clear deceleratory responses in infants over 36 hours old.

The activity of the infant during stimulation is also an important determinant of the direction of the HR response (Gregg, Clifton, & Haith, 1976; Nelson, Clifton, Dowd, &

Field, 1978; Pomerleau & Malcuit, 1981). Gregg et al. (1976) examined the effects of sucking during stimulus presentation on the HR response and showed that there was a reliable HR acceleration on those trials during which the infant sucked. Trials on which the infant was not sucking, however, were accompanied by a non-reliable HR deceleration. Gregg et al. (1976) also noted that previous studies which reported acceleratory responses or no reponses (Jackson, et al, 1971) had used pacifiers to maintain the infants in an alert state, whereas those reporting deceleratory responses had not (Adkinson & Berg, 1976; Pomerleau-Malcuit & Clifton, 1973). The effects of sucking on HR responses were demonstrated more clearly by Nelson et al. (1978) in a study of the responses of 16 awake newborns. Nelson et al. (1978) found that the direction of HR response to a stimulus depended on the ongoing HR change related to a burst of sucking. If the stimulus was presented before a burst, or early in the burst, the response was acceleratory, but if the stimulus was presented at the end of a burst or after a burst, the response was deceleratory.

Consideration of the combination of the infant's state and activity within that state is also important. Pomerleau and Malcuit (1981) demonstrated that, in the waking state, the HR response was directly related to the behavioral response to stimulation. An increase in behavior in response

to a stimulus was accompanied by an acceleratory HR response, while a deceleratory response accompanied quieting. In the sleep states, the response to stimulation was acceleratory, regardless of whether the infant increased or decreased its movement. The results of this study, combined with the results of previous studies, suggest a general conclusion that it is possible to elicit a deceleratory HR response from an infant by presentation of a moderate intensity, slow rise-time stimulus to an alert infant (Clifton & Nelson, 1976). Because of the similarities between spontaneous startles and evoked startles, it is important to remember that intense, sudden stimuli elicit HR accelerations in all states (Campos & Brackbill, 1973; Clifton & Nelson, 1976).

While these studies indicate that deceleratory responses can be elicited from the newborn, they do not entirely contradict Graham et al.'s (1968) suggestion that the predominantly acceleratory responses are the product of a relatively immature organism. Support for this suggestion can be derived from conceptualizations of infant development. Major changes have been noted in the pattern of infant behavior between the second and fourth month of life. For example, many of the reflexes that the neonate displays, including the Moro reflex, dissappear during this period (McGraw, 1933). Other behaviors, such as smiling, do not

disappear, but lose their spontaneous character and come under the control of environmental stimuli (Emde & Gaensbauer, 1981). These changes have long been considered demonstrative of the descent of cortical control mechanisms (Emde & Gaensbauer,1981; Graham et al., 1968; McGraw, 1933), and physiological and anatomical evidence supports this concept (Bronson, 1982).

Recent investigations using animal preparations provide evidence that the startle response is mediated by brainstem and midbrain mechanisms. Davis, Gendelman, Tischler, and Gendelman (1982) demonstrated, through both lesion and stimulation manipulations, that the circuit mediating the acoustic startle response in rats consists of the auditory nerve, ventral cochlear nucleus, nuclei of the lateral lemniscus, nucleus reticularus pontis caudalus, spinal interneuron, lower motor neuron, and muscle. These results indicate that startle responses in the rat are mediated by brainstem and midbrain structures which, in the human infant, are relatively mature in comparison with cortical structures.

Although care must be taken when generalizing from animal models, the results of these studies, combined with the pattern of disappearance observed in the development of both the Moro reflex and spontaneous startles (McGraw, 1933; Wolff, 1966), suggest involvement of brainstem mechanisms in

these behaviors. Chusid (1979) refers to the "mass reflex", a spread of response in spinal humans and animals which includes many reflexes, includes autonomic outflow, and may at times appear "spontaneously and without obvious stimulation" (p. 79). The resemblance of this phenomenon to spontaneous startles provides further support for the mediation of startles by brainstem mechanisms.

The possibility that spontaneous startles are controlled by a brainstem mechanism is important because it relates this behavior to other crucial processes in the newborn. Brainstem and midbrain activity is related to the maintenance of homeostasis via regulation of cardiac and respiratory activity, and temperature control, all of which are relatively immature in the newborn (Adolph, 1968; Harper et al, 1976). Spontaneous startles thus might play a role in neonatal adaptation by interacting with other processes to affect the infant's homeostatic balance.

Spontaneous startles as a homeostatic mechanism

The term homeostasis refers to the balance of an organism's internal physiological processes, which can be maintained in a number of ways, one of which is via intake and output of energy (Adolph, 1968). Based on several characteristics of startle behavior observed in 12 infants, Wolff (1966) provided an "energy overflow" model to describe the generation of spontaneous startles. These

characteristics include 1) more frequent occurrence in the sleep states, in which the infant is least accessible to either internal or external sources of stimulation, 2) occurrence in inverse proportion to the amount of gross bodily activity observed in the same period, 3) a regular periodicity in the occurrence of startles, and 4) a refractory period following a spontaneous startle in which a startle response is more difficult to elicit. According to Wolff, the energy release model is also supported by observations that other spontaneous behaviors, such as sucking or smiling, "substitute" for startles. As the infant's feeding time approaches, startles become less frequent and sucking becomes the predominant spontaneous behavior, suggesting that as internal changes occur, the energy is released via different behaviors (Korner, 1969; Wolff, 1966).

Korner (1969) supported Wolff's (1966) findings and further suggested that spontaneous behaviors such as startles may serve the homeostatic function of providing stimulation to the developing nervous system in those periods in which it is "deafferenated", or inaccessible to other forms of stimulation. In support of this suggestion, Korner suggested a parallel between spontaneous startle activity and REM activity in neonates. The sleep periods of newborns show a high proportion of REM activity relative to

periods of quiet sleep (Parmalee et al., 1967). Roffwarg et al. (1966) suggested that these REM periods provide stimulation which assists the processes of maturation and differentiation of the nervous system. Korner proposed that spontaneous behaviors such as startles might contribute to this process.

Huntington, Zeskind, and Weiseman (1985) reported analyses of data from a larger study of the effects of prenatal conditions on the organization of infant state behavior (Zeskind, Goff, Huntington, & Weiseman, 1983) that both support and extend previously reported results. Similar to the findings of previous studies, Huntington et al. found higher rates of startles in the sleep states relative to the waking states. Contrary to previous reports, however, Huntington et al. found significant rates of startles across the entire range of states, including the waking states. When these data were analyzed using the 11-pt. scale developed by Thoman (1975), the rate of startles in the "lowest" state, quiet sleep A, was significantly lower than that in quiet sleep B and did not differ significantly from that in the waking states. According to the model proposed by Wolff (1966) and Korner (1969), because the infant is most deafferented in quiet sleep A, the rate of startles should be highest in this state.

An important difference between Huntington et al.'s

(1985) study and previous reports is the focus on a wider range of infants in the newborn nursery. Korner (1969) excluded infants if their prenatal histories included such common conditions as multiple birth, extensive labor, abnormal presentation, or the use of high forceps. Huntington et al. suggested that the focus on this restricted sample might limit the range of individual differences observed. For example, those infants who were characterized by nonoptimal fetal growth showed a significantly higher rate of starles during the drowse state than other infants (Huntington et al., 1985). Because of the wide variability in newborn behavior, it is important to remember that these infants were considered normal, healthy newborns by hospital standards.

Among infants in the normal newborn nursery, individual differences in spontaneous and elicited behaviors have been related to differential exposure to nonoptimal obstetric conditons such as those excluded by Korner (1969) and included by Huntington et al. (1985) (Sepkoski, Garcia-Coll, & Lester, 1982). Infants exposed to high numbers of nonoptimal conditions display higher thresholds for elicitation of a cry response and unusually high pitched cries (Zeskind & Lester, 1978), as well as extreme heart rate (HR) variability (Zeskind & Field, 1982). These behavioral differences have been associated theoretically

and empirically with individual differences in the functional integrity of the infant's autonomic nervous system (ANS) (Lester & Zeskind, 1982; Zeskind, 1983), the final common pathway for homeostatic mechanisms.

Combining the results of studies of spontaneous and evoked startles with the results of studies of the effects of prenatal conditions suggests an extension of the homeostatic model proposed by Korner (1969). Spontaneous startles might act to modulate the biobehavioral state of the infant. This suggestion is supported by the reports of differential rates of startles in different states (Huntington et al., 1985; Korner, 1969; Wolff, 1966). In addition, infants with histories of nonoptimal conditions which have been shown to disrupt ANS functioning show a different pattern of startles than has been reported previously (Huntington et al., 1985). For example, the unusually high rate of startles displayed in the drowsy state by infants with nonoptimal fetal growth may indicate an attempt by the infant to maintain the aroused state it had achieved. These infants have been shown in the past to be lethargic, having difficulty maintaining the arousal necessary to remain awake for sustained periods (Als, Tronick, Adamson, & Brazelton, 1976).

Startles could modulate the biobehavioral state of the infant by decreasing the infant's arousal, increasing the

infant's arousal, or by maintaining a given level of arousal. The energy overflow model proposed by Wolff (1966) posits that startles release energy which might otherwise wake the infant, suggesting that startles either lower arousal or maintain a particular level of arousal. One way of examining this suggestion is to observe the prestartle cardiac and respiratory activity of the infant. Given the energy overflow model, startles should be preceded by a period of increasing cardiac and/or respiratory activity and followed by activity which either decreases, or increases briefly and then returns to a level lower than the level preceeding the startle.

An alternative suggestion is that spontaneous startles modulate periods of low arousal. In this case startles might increase cardiac and respiratory activity. This suggestion is supported by consistent reports of heart-rate acceleration following the elicitation of a startle (Graham, 1979), and by the reports of Wolff (1966) and Korner (1969) that the highest rates of startles are seen in the sleep states in which the lowest average cardiac and respiratory rates are found (Adkinson & Berg, 1976; Ashton & Connolly, 1971; Campos & Brackbill, 1973; Harper et al., 1976; Jackson et al., 1971; Pomerleau-Malciut & Clifton, 1973). Further support for this suggestion comes from the finding that, in the sleep states, the infant's predominant mode of HR

response to stimulation is acceleratory (Bridger & Reiser, 1959; Graham et al., 1968; Lipton & Steinschneider, 1964).

The results of Huntington et al. (1985) at first appear to conflict with the suggestion that startles modulate low arousal. The rate of startles in quiet sleep A was significantly lower than that in quiet sleep B, and did not differ significantly from the rates in the waking states. This result suggests that startles are not related to lower cardiac and respiratory rate. Startles may still, however, be related to cardiac and respiratory activity. In addition to the mean rates differing, cardiac and respiratory variability differ between quiet sleep A and B (Thoman, 1975), with greater variability associated with quiet sleep B. Spontaneous startles may be associated with the variability in this activity, rather than with the average rate. For example, Zeskind and Field (1982) showed that while the mean heart rate of risk infants did not differ from that of controls, their cardiac activity showed greater variability, with periods of greater excitation and inhibition.

One way of examining modulation of cardiac and respiratory activity by spontaneous startles is by observing trends in cardiac and respiratory activity preceding the occurrence of spontaneous startles. In addition, the relation of startles to these indices of ANS activity may be

demonstrated through the use of a manipulation. Brackbill, Adams, Crowell, and Gray (1966), using heartbeat sound, lullabye, and metronome, demonstrated the inhibitory effect of auditory stimulation on crying, motor activity, and cardiac and respiratory activity in neonates. This effect pas also been demonstrated for moderate intensity (85 dB) white noise (Brackbill, 1973), but not for lower intensities (65-75 dB) (Murray & Campbell, 1971). The important point for this study is that the auditory stimulation reduced both the mean and the variability of the cardiac and respiratory activity.

This study had two major purposes. The first purpose was to examine the connection between the systems mediating spontaneous and evoked startles by examination of the pattern of HR change following spontaneous startles. The second purpose was to examine the relation of spontaneous startles to ongoing homeostatic processes. This relation was examined in two ways: 1) by observation of the cardiac and respiratory activity preceding and following the occurrence of spontaneous startles and 2) by the examination of changes in this activity concommitant with the application of an auditory intervention. The hypotheses of this study were:

1) To the extent that the system controlling spontaneous startles is related to that controlling

evoked startle responses, the occurrence of spontaneous startles would be followed by significant heart rate accelerations resembling those associated with evoked startles.

2) To the extent that spontaneous startles are related to the maintenance of the infant's homeostatic balance, spontaneous startles would be preceded by some combination of the following: low heart rate, low respiratory rate, declining trends in heart rate and/or respiratory rate, and possibly apneic pauses of longer than 15 seconds.

3) To the extent that spontaneous startles are related to the occurrence of slowed respiration or apneic pauses, startles would be followed by an increase in the respiratory activity of the infant.

4) To the extent that spontaneous startles are related to the ongoing homeostatic activity of the infant, modification of this activity by application of an auditory stimulus would increase the frequency of spontaneous startles.

Methods

Subjects

The subjects were 26 1-2 day old infants (12 male, 14 female), residing in the normal newborn nursery at Roanoke Memorial Hospital. Because of hospital policy constraints, subjects were selected from those infants born to mothers attending a social services prenatal care clinic. All subjects were healthy newborns as judged by routine pediatric examinations. Infants were not observed within 18 hours of either caesarean section or circumcision. Demographic characteristics of the infants are provided in Table 2.

Informed consent was collected from the mothers of all infants in their rooms. The information provided to the mothers will consist of a brief explanation of the purpose and procedure of the study. Because the letter of consent used for this study had been used previously in other studies, it contained information regarding the study of the infant's cry. Mothers were informed that the procedure for this study did not include making their infant cry. Emphasis was placed on the fact that this study would not provide information about the health of any individual infant, but is designed to provide information about general aspects of infant development. A copy of the letter of consent is included in Appendix 1.

Table 2

Means and Standard Deviations of Infant Demographics

Variable	Total Sample Mean(SD)	Auditory Intervention Mean(SD)	Non-Intervention Mean(SD)
Age(hr)	27.6(13.1)	24.5(6.9)	30.7(17.0)
1 Min Apgar	7.6(1.7)	8.3(.85)	6.9(2.1)*
5 Min Apgar	9.0(1.7)	9.1(.37)	8.9(.28)
Weight(gm)	3360(414.0)	3302(436.7)	3418(399.8)
Length(cm)	49.9(3.23)	49.9(3.18)	49.8(3.40)

* $p < .05$

Apparatus

A continuous record of cardiac and respiratory waveforms was recorded on a Grass Model 7 Polygraph via Hewlett Packard electrodes attached to a Cavitron KDC monitor. Pilot data for this study indicated that the occurrence of a spontaneous startle caused an overload of the polygraph channel recording the respiratory waveform. Dr. Leon J. Arp developed a circuit which was attached to the light output of the respiration channel of the monitor (see Appendix 2). This channel was not as sensitive to the movement artifact caused by the spontaneous startle and allowed the continuous recording of the poststartle respiratory activity. The cardiac and respiratory waveforms were manually transformed to heart rate and respiratory rate using a Tektronix 4956 digitizer and graphics tablet to measure intervals to within .10 mm.

A Dakin "Rock-a-Bye Bear" provided the auditory stimulus for the intervention period. This bear contains a tape recording of womb sounds and is routinely used in the nursery to calm infants. The sound that this bear generates is a pink noise with predominant frequencies between 120 and 320 Hz. The intensity of the sound, measured at 6 inches from the source, sweeps between 83 and 86 db with a cycle length of .7 seconds.

Procedure

Each infant was observed for 60 minutes, between scheduled feedings. The infant was taken to a quiet room adjoining the nursery and placed in an Armstrong isolette which maintained the ambient temperature at 32 °C and attenuated sound. Hewlett Packard 40426 disposable electrodes were placed over the left clavicle and over the ribs below the right nipple. The observation period began ten minutes after the infant was placed in the isolette. This period allowed the electrode paste to stabilize, and the infant to calm after the stimulation of being connected to the physiograph.

The infants were randomly assigned to one of two groups: 1) an auditory intervention group, which recieved auditory stimulation during the first half hour of the observation period, and 2) a no intervention control group, which was not exposed to the auditory stimulation. This design is represented in Table 3. The auditory stimulation began concurrent with the observation for the infants in the intervention group. A comparison of the groups' demographic characteristics is provided in Table 2. As represented in the table, the only characteristic on which the groups differed was the one minute Apgar scores.

Table 3

Research Design

Group	n	Periods 1	2
Auditory Intervention	13	Intervention	No Intervention
Control	13	No Intervention	No Intervention

Note: Each period was one half hour in length.

The 11-pt. state scale developed by Thoman (1975)(See Table 1) was used to rate the biobehavioral state of the infant. This scale was used because it can be converted to the 7-pt. scale for comparison with previous studies, whereas the converse transformation is not possible. The infant's state was determined every 30-sec by the consensus of two observers, based on the 10-sec epoch immediately preceding the recording point (Zeskind et al., 1983). The reliability of the state observations was determined for the observations of a subset of six infants (23% of the sample) on which the observers were blind to each others state ratings. The reliability coefficient for this data, calculated as the number of observations on which the observers agreed divided by the total number of observations equaled .95 for the 11-pt. scale and .98 for the 7-pt. scale. Because of the amount of time that the infants spent in the sleep states, these reliability coefficients are based predominantly on judgements between the sleep states.

For the purposes of this study, spontaneous startles were defined according to the criteria of Korner (1969), as "spontaneously occurring massive body jerks which resemble the Moro reflex and involve both sudden extensor and flexor movements of the limbs" (p. 104). The frequency of spontaneous startle activity was determined by one trained observer, who recorded the occurrence of a startle via an

event marker connected to the polygraph. This observer was not blind to the hypotheses of the study but was maintained blind to the group membership of the infant by listening to music played on a Sony D-6 cassette player during the first half of the observation period. Reliability of the observation of startles was determined on a subsample of five infants (19% of the sample) on which two of the observers recorded startle occurrences, while blind to each others recordings. The reliability coefficient for this data was .73. The reliability of the first occassion on which this reliability check was used was .33, which might have had as much to do with instituting the new procedure as with the actual reliability of the observations, and which adversely affected the overall reliability coefficient. The average reliability of the remaining four checks was .83.

Results

For the purposes of this study, only those startles occurring in the sleep states were used. Although Huntington et al. (1985) demonstrated the occurrence of startles across the range of states, there is no reason to assume that startle activity should take the same form or have the same function in all states. Evidence from studies of infant's responses to stimuli indicate that the same stimulus might elicit a different response in different states. Further, in the waking states visual stimuli or the infant's own activity might be the startle eliciting stimulus.

There were two reasons that startle activity was examined across the combined sleep states, rather than examining the differences between these states. First, the quiet sleep state occurs relatively less frequently in newborns (Petre-Quadens, 1967). In this sample, only 11 of 26 infants were observed in both active and quiet sleep in both halves of the observation period. Further, only 5 of 26 infants showed startle activity in both sleep states in both periods. Seven infants who were observed were not included in the sample for this study because they either did not show startles or did not sleep in one of the periods.

The second reason that startles occurring in both sleep states were used is based on previous studies of evoked startle responses in infants (Campos & Brackbill, 1973;

Clifton & Nelson, 1976; Graham et al., 1968). These studies demonstrated that although there are differences in the heart rate responses to stimulation which are dependent on the state of the infant, startle evoking stimuli were associated with heart rate accelerations regardless of state. Thus, these analyses examine the relation of cardiac and respiratory activity to spontaneous startles in the sleep states.

One important question is whether spontaneous startles occur frequently enough in newborn infants to warrant analysis. If this behavior is infrequent, only occuring in a few infants, although it may have a functional significance it may not be related to homeostatic functioning in the majority of infants. In this sample of 26 infants, the mean number of startles was 15.7 across the whole one hour observation period, with a standard deviation of 10.7. The minimum number of startles emitted by an infant was 3, and the maximum was 46, for a range of 43 startles. Thus, rather than being a rarity, startles occur with some frequency in most infants.

The results of the analyses for this study will be presented in three sections:

1) The results of the analyses of the pre- and poststartle cardiac and respiratory activity and supporting analyses.

2) The results of analyses of the mean and variability of heart rate, respiratory rate, and number of startles.

3) The results of further supporting analyses.

A conservative alpha level of .025 was designated as the significance level for this study because of the need to perform multiple analyses.

Analyses of pre- and poststartle cardiac and respiratory activity

The dependent measures for these analyses were the raw score heart rate and respiratory rate. Raw scores were employed because they are less susceptible to problems of nonnormality of distributions than are change scores (Graham, 1978a). For each second, both heart rate and respiratory rate were calculated as the weighted average of the portions of interbeat or interbreath intervals which occurred during that second, converted to rate.(Graham, 1978b).

For each infant, from one to seven startles were used in each half of the one hour observation period, chosen from those startles which were free of movement artifact. Because of the circuit that allowed the recording of the respiratory activity from the monitor light, respiratory activity was rarely affected by movement artifact. The movement sometimes affected the recording of the cardiac activity however. This

disruption took the form of either adding noise to the recording or losing the cardiac waveform, both of which obscure the R-R intervals, making the calculation of heart rate imposible. For this reason, recordings which were affected by movement artifact were not used in these analyses. The groups did not show a differential distribution of the number of startles excluded, $F(1,24) = 1.54$, $p = .26$.

Previous studies of physiological responses to stimulation have found that most changes occur within the first eight seconds following stimulus onset (Graham, 1979). For the analyses in this study, heart rate and respiratory rate were calculated for each of the eleven seconds preceding and eight seconds following the occurrence of a spontaneous startle and then averaged across startles. Thus, each infant received one score for each second preceding and following startles which was an average of the rates for that second based on one to seven startles per period (See Table 4). The eleven seconds preceding spontaneous startles were further subdivided into two epochs. The first eight seconds were used as the prestartle recording epoch. The three seconds directly preceding the startle were used as the baseline recording epoch. The heart rates and respiratory rates were averaged across the three seconds as

Table 4

Calculaton of Average Heart by Second Across Startles

Startle	Second																		
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
2	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
3	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
4	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
5	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
6	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
7	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
Mean	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.

I_____I_____I_____I

Prestartle Epoch | Baseline | Poststartle Epoch

Recording Epochs

a base level with which to compare pre- and poststartle trends.

The three second averaged base level was chosen because of response latency in recording the occurrence of startles. When a spontaneous startle occurred the observer recorded it, however there was always some latency in the recording of the occurrence. As there was no way to know with certainty the length of that latency it was assumed that it could be as much as a full second. If only the last recording second before a startle was used, it could contain changes in rate due to the startle, and weight those changes too heavily in relation to the preceding and following activity. The three second period was chosen because it insured that the baseline would include information from the second during which the startle occurred, averaged with information from the seconds before and after the startle occurred, giving a more accurate representation of the activity at the time of the startle than any one of these seconds alone would give.

Heart rate and respiratory rate across the eight seconds preceding and eight seconds following spontaneous startles were analyzed in four separate 9 Seconds (eight seconds and the baseline) x 2 Period (1st and 2nd half of the observation period) x 2 Group (auditory intervention - nonintervention) ANOVAs. Seconds and Period were

repeated-measures factors. Pre- and poststartle activity were analyzed separately to ensure that any lack of effects in one epoch would not obscure effects in the other epoch.

There is some disagreement about the appropriateness of the ANOVA for the analysis of repeated-measures designs because of problems of inequality of the variance-covariance matrices of measures taken at varying time intervals (McCall & Appelbaum, 1973). These inequalities violate the basic assumption of the ANOVA model, and lead to a spuriously high rate of rejection of the null hypothesis, increasing the probability of Type I error. There are correction methods, such as the Greenhouse-Geisser method, which correct the degrees of freedom for the tests of the repeated-measures factors, based upon the degree of inequality of the variance-covariance matrices. McCall and Appelbaum (1973) stated that this method of correction, while useful, is overly conservative, and have recommended the use of Multiple Analysis of Variance (MANOVA) for analysis of repeated-measures data.

Hertzog and Rovine (1985) suggested that the decision of whether to use ANOVA or MANOVA analytical methods should include consideration of the specification of particular a priori hypotheses about the nature of the effects expected on the repeated-measures factors. For example, if hypotheses about trends or differences can be translated into

orthogonal comparisons, these single degree-of-freedom tests are not affected by the inequality of the variance-covariance matrices, and thus provide adequate tests of the hypotheses. The ANOVA model was thus considered appropriate for these analyses because the main hypotheses pertain to trends in the pre- and poststartle heart rate and respiratory rate. In order to examine the Periods and Groups factors effects and interactions, the results of both the traditional tests of these effects and the Greenhouse-Geisser corrected results were employed. Where the results of these tests disagree, the implications of both are considered and conclusions from the traditional tests are examined with caution.

The analyses of cardiac and respiratory activity for this study were not performed in the traditional stepwise manner recommended by Wilson (1974, 1980) and Richards (1980). These authors recommend an initial analysis of covariance (ANCOVA) to examine the efects of differences in baseline activity levels and remove those effects if significant. Significant covariance indicates the operation of the Law of Initial Values (Wilder, 1958), that the nature of the change in activity is affected by differences in the baseline activity levels of the groups. This analysis was not conducted on the heart rate and respiratory rate data because a major hypothesis of this study was that auditory

stimulation would change the baseline cardiac and respiratory activity levels. These effects were examined in the 3-way ANOVAs as effects of the Group factor and interactions of this factor with the Seconds and Periods factors.

The prestartle heart rate data are represented in Table 5 and the analysis of these data is represented in Table 6. There were no reliable effects on the Seconds, Periods, or Groups factors. The trends contrasts for the Seconds factor indicate that only the Seconds(quartic) trend approaches the significance at the specified .025 alpha level. These results are represented in Figure 1.

The means and standard deviations of the poststartle heart rate activity are represented in Table 7 and Table 8 represents the analysis of these data. There was a reliable Seconds main effect, $F(8,192)=11.21$, $p < .001$ (Greenhouse-Geisser $p < .001$), which was associated with significant quadratic, $F(1,24)=20.07$, $p < .001$, and cubic, $F(1,24),=31.71$, $p <.001$, trends. The lack of a reliable linear trend indicates that the final heart rate did not differ from the baseline heart rate. These data are represented in Figure 2.

In addition to the Seconds effects, there was a reliable main effect for Periods, $F(1,24)=8.48$, $p < .01$. The Seconds(lin) x Periods x Groups interaction approached

Table 5

Means and Standard Deviations of Prestartle Heart Rate

	Seconds								
	1	2	3	4	5	6	7	8	Base
Intervention									
Period 1									
Mean	120.1	119.8	120.0	119.6	119.3	118.1	118.2	119.3	121.7
(SD)	10.8	10.7	11.6	11.9	12.2	12.3	12.4	10.4	8.9
Period 2									
Mean	121.8	121.2	122.2	120.2	121.6	120.8	119.0	119.9	123.4
(SD)	15.4	18.0	15.9	16.2	15.3	14.3	14.8	13.0	12.3
Nonintervention									
Period 1									
Mean	119.1	118.6	118.5	119.8	119.4	119.9	118.8	119.2	119.8
(SD)	10.1	10.4	12.2	11.0	13.2	12.0	11.3	9.2	10.1
Period									
Mean	119.1	119.4	119.0	120.0	124.7	118.0	119.3	118.7	121.6
(SD)	12.0	12.6	13.0	12.9	28.6	10.7	11.2	10.8	10.7

Table 6

ANOVA: Prestartle Heartrate

Source	Sum of Squares	Degrees of Freedom	F	P	Greenhouse Geisser P
Groups	55.66	1	.02	.87	
Error	54883.04	24			
Seconds	359.64	8	1.28	.25	.29
Seconds X Groups	169.03	8	.60	.77	.61
Error	6722.16	192			
Seconds (lin)	.97	1	.01	.91	
Error	1747.08	24			
Seconds (quad)	39.71	1	.84	.37	
Error	1140.12	24			
Seconds (cub)	99.96	1	3.39	.08	
Error	708.71	24			
Seconds (quart)	143.31	1	5.50	.028	
Error	625.05	24			
Periods	163.48	1	.53	.47	
Periods X Groups	14.44	1	.05	.83	
Error	7381.57	24			
Seconds X Periods	128.28	8	.50	.85	.64
Seconds X Periods X Groups	103.18	8	.41	.92	.70
Error	6097.77	192			

Note: Because the majority of the interactions of the Periods and Groups factors with the Seconds trends did not approach the designated .025 alpha level, they have been ommitted from this and the following tables except where otherwise indicated.

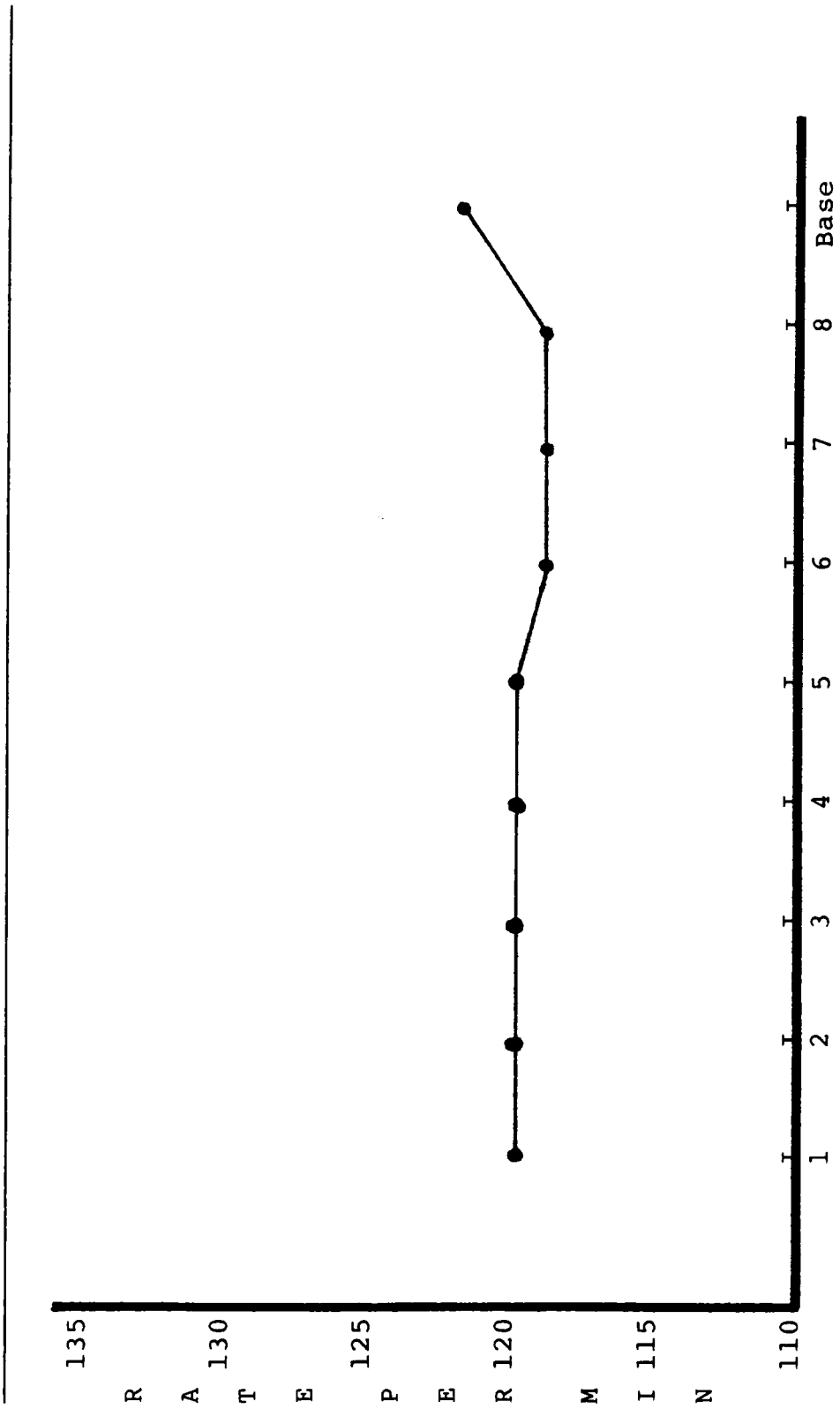

Figure 1. Prestartle Heart Rate, Averaged Across Periods and Groups

Table 7

Means and Standard Deviations of Poststartle Heart Rate

	Seconds								
	Base	1	2	3	4	5	6	7	8
Intervention									
Period 1									
Mean	121.7	125.9	129.1	130.3	126.6	124.6	123.4	122.5	124.1
(SD)	8.9	8.2	7.4	9.1	13.2	13.0	15.6	13.2	12.9
Period 2									
Mean	123.4	129.2	131.4	132.2	132.8	132.3	132.0	132.8	133.3
(SD)	12.3	8.6	8.5	9.4	10.8	8.3	11.7	10.8	9.9
Nonintervention									
Period 1									
Mean	119.7	124.5	129.6	129.6	126.5	125.0	124.7	123.6	124.9
(SD)	10.1	11.3	10.2	10.3	10.6	10.5	11.8	11.6	10.5
Period 2									
Mean	121.6	129.0	132.3	132.3	127.7	126.9	126.0	125.7	127.4
(SD)	10.7	10.9	12.0	11.0	13.1	12.7	13.9	11.8	12.3

Table 8

ANOVA: Poststartle Heart Rate

Source	Sum of Squares	Degrees of Freedom	F	P	Greenhouse Geisser P
Groups	333.21	1	.21	.65	
Error	37884.24	24			
Seconds	3175.76	8	11.21	.000	.000
Seconds X Groups	168.62	8	.59	.79	.61
Error	6909.12	192			
Seconds (lin)	59.94	1	.53	.47	
Error	2680.05	24			
Seconds (quad)	1369.09	1	20.07	.0002	
Error	1637.44	24			
Seconds (cub)	1643.24	1	31.71	.000	
Error	1243.88	24			
Seconds (quart)	17.70	1	.82	.37	
Error	520.26	24			
Periods	1894.06	1	8.48	.008	
Periods X Groups	333.89	1	1.49	.23	
Error	5360.49	24			
Seconds (lin)* X Periods X Groups	327.2	1	4.58	.043	
Error	1716.35	24			
Seconds X Periods	262.26	8	1.62	.12	.19
Seconds X Periods X Groups	400.00	8	2.47	.014	.069
Error	3884.85	192			

Note: The Seconds(linear) X Periods X Groups interaction (*) is included in this table because it approaches the designated .025 alpha level.

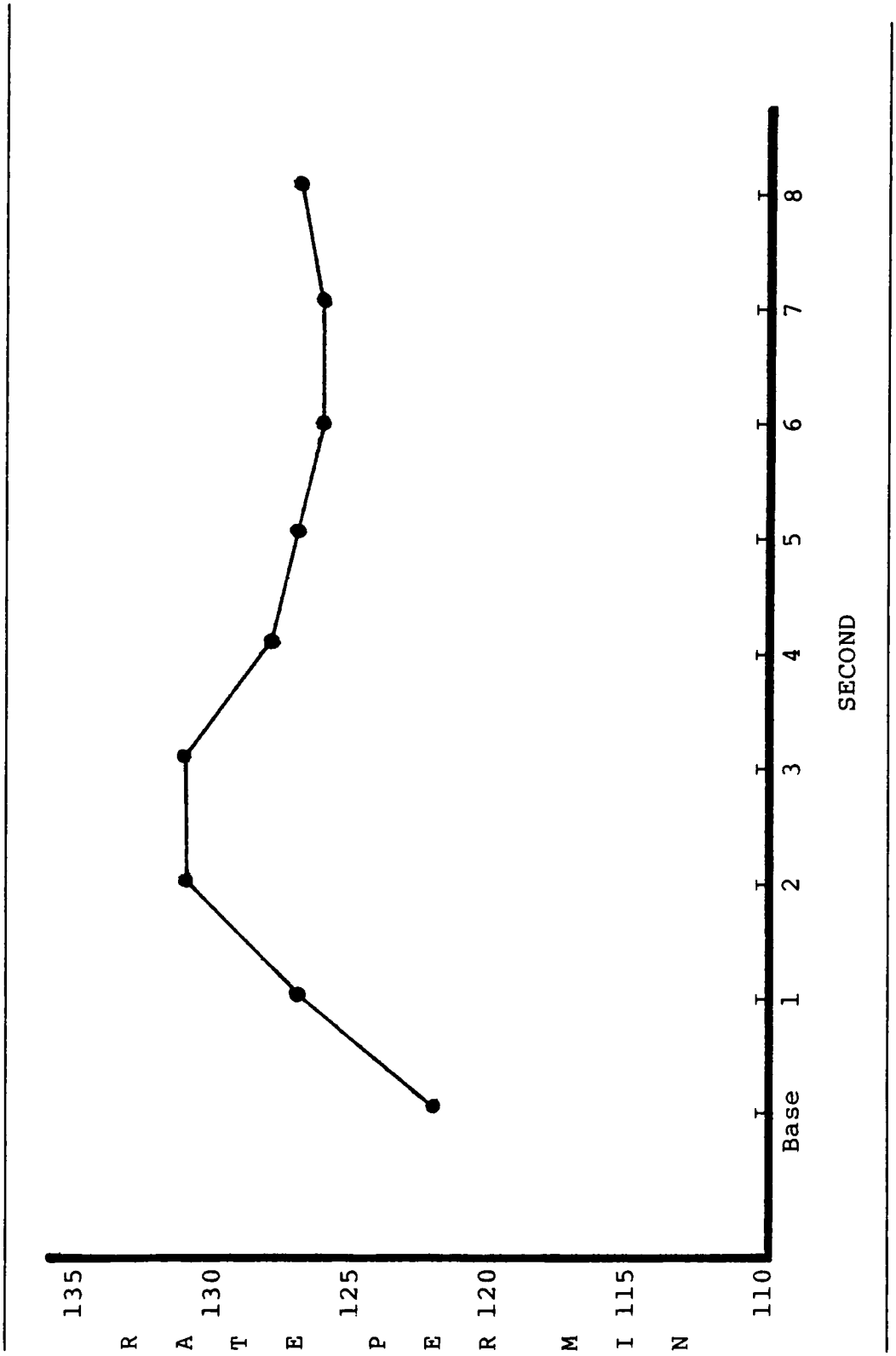

Figure 2. Poststartle Heart Rate, Averaged Across Periods and Groups

significance, $F(1,24)=4.58$, $p=.043$. While the traditional test of the Seconds x Periods x Group interaction indicates that it is reliable, $F(8,192)=2.47$, $p < .025$, the test employing the Greenhouse-Geisser correction, $p > .05$, indicates that the effects of this factor are not reliable given the variance-covariance differences in this sample. A test of the power of this analysis indicated a power of .99.

As shown in Figure 2, the significant Seconds main effect and trends are attributable to a 10 beat per minute rise in heart rate immediately following a startle, followed by an approximately 5 beat per minute decrease after 3 seconds with a final small but significant increase at 6 seconds. The reliable Periods effect is attributable to a generally higher poststartle heart rate during the second half of the observation period (Mean poststartle heart rate period 1 = 126.2, SD = 9.97; Mean poststartle heart rate period 2 = 130.4, SD = 10.22). Figure 3 represents the pre- and poststartle heart rate activity together.

The means and standard deviations of the prestartle respiratory activity are represented in Table 9 and the results of the analysis of the this activity are represented in Table 10. This analysis indicates a reliable Seconds effect, $F(8,192)=4.64$, $p < .001$ (Greenhouse-Geisser $p < .005$). This Seconds effect is associated with significant Seconds(linear), $F(1,24)= 8.73$, $p < .01$, and

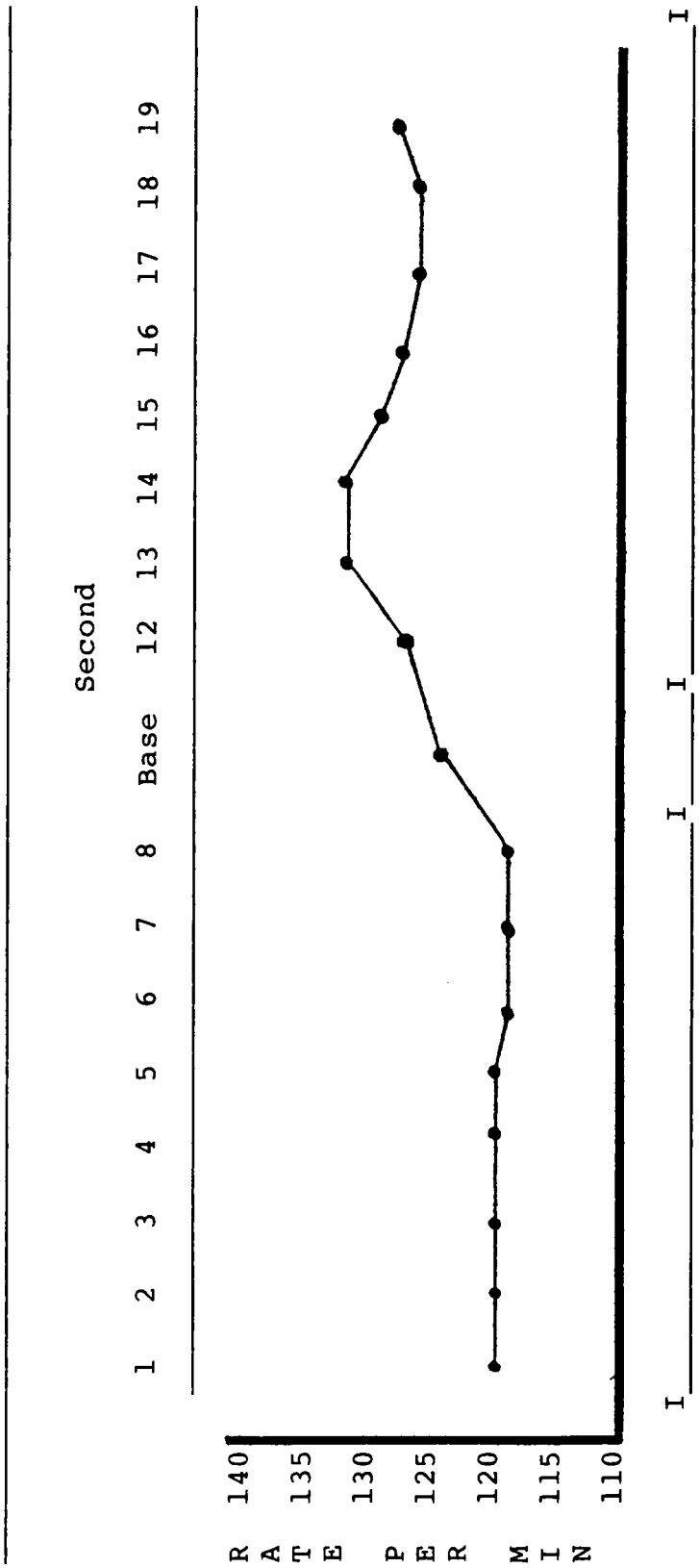

Figure 3. Pre- and Poststartle Heart Rate Averaged Across Period and Group

Table 9

Means and Standard Deviations of Prestartle Respiratory Rate

	Seconds								
	1	2	3	4	5	6	7	8	Base
Intervention									
Period 1									
Mean	44.6	44.3	43.9	44.0	43.6	43.1	41.3	40.6	39.2
(SD)	8.6	9.4	8.9	8.3	9.8	10.2	9.3	7.6	7.8
Period 2									
Mean	38.0	37.2	41.4	42.5	41.7	41.4	39.3	36.8	30.8
(SD)	13.2	13.6	13.7	13.5	16.3	16.0	14.2	10.2	11.6
Nonintervention									
Period 1									
Mean	35.8	36.5	35.5	36.0	35.8	33.6	32.6	33.7	33.3
(SD)	10.4	12.2	11.4	11.3	9.3	9.8	13.44	13.4	13.7
Period 2									
Mean	36.6	38.6	37.2	37.1	37.9	35.5	35.0	34.7	33.3
(SD)	10.4	9.4	7.6	8.2	9.3	7.7	8.8	9.9	9.9

Table 10

ANOVA: Prestartle Respiration Rate

Source	Sum of Squares	Degrees of Freedom	F	P	Greenhouse Geisser P
Groups	3243.74	1	2.55	.12	
Error	30531.89	24			
Seconds	1502.21	8	4.64	.000	.003
Seconds X Groups	376.93	8	1.16	.32	
Error	7769.13	92			
Seconds (lin)	936.22	1	8.73	.007	
Error	2572.88	24			
Seconds (quad)	526.83	1	6.61	.016	
Error	1914.09	24			
Seconds (cub)	.01	1	.00	.98	
Error	681.23	24			
Seconds (quart)	3.46	1	.07	.79	
Error	1204.70	24			
Periods	180.82	1	.91	.34	
Periods X Groups	851.04	1	4.30	.049	
Error	4753.35	24			
Seconds X Periods	265.73	8	.66	.73	.57
Seconds X Periods X Groups	144.80	8	.36	.94	.77
Error	9716.99	192			

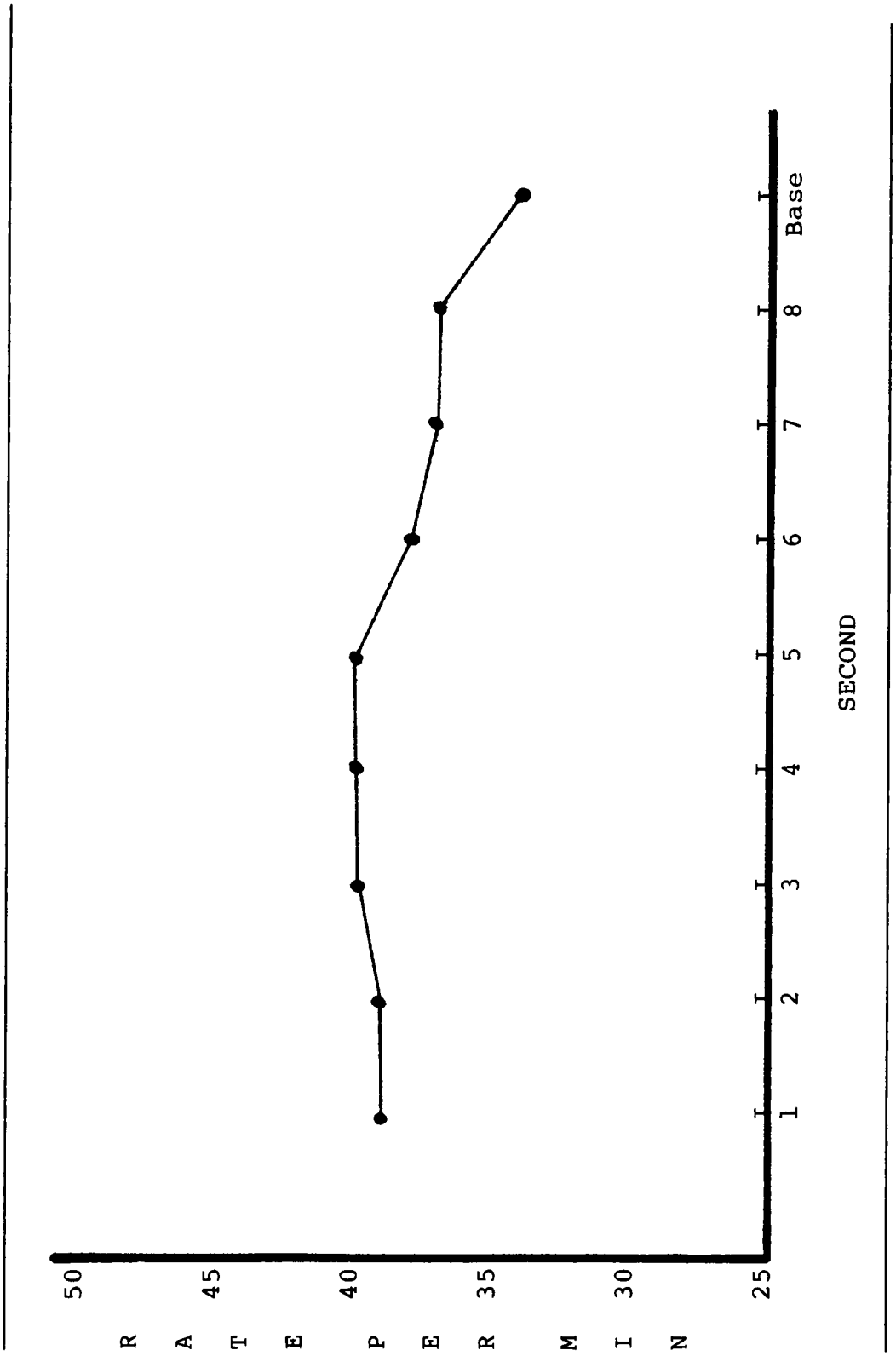

Figure 4. Prestartle Respiratory Rate, Averaged Across Periods and Groups

Seconds(quadratic), $F(1,24)=6.61$, $p < .025$, trends. As represented in Figure 4, the linear trend is attributable to a decrease of approximately 5 inspirations per minute between the first of the eight prestartle seconds and the baseline. The significant quadratic trend appears to be attributable to the small increase in the rate at which followed by the decrease between the 6th prestartle second and the baseline level. There were no reliable main effects for the Periods or Groups factors, and these factors did not interact reliably with the Seconds factor or trends.

Table 11 represents the means and standard deviations of the poststartle respiratory data and the results of the analysis of these data are represented in Table 12. This analysis indicates a significant Seconds effect, $F(8,192)= 7.63$, $p <.001$ (Greenhouse-Geisser $p < .001$), which is associated with a significant Seconds(linear) effect, $F(1,24)=18.8$, $p <.001$. As represented in Figure 5, the significant linear trend is attributable to a decrease of approximately 10 inspirations per minute between the baseline respiratory rate and that of the last poststartle second. Again, there were no reliable main effects or interactions for the Periods and Groups factors.

Figure 6 represents the pre- and poststartle respiratory activity together. Because there was a significant linear decrease in both the pre- and poststartle

Table 11

Means and Standard Deviations of Poststartle Respiratory Rate

	Seconds								
	Base	1	2	3	4	5	6	7	8
Intervention									
Period 1									
Mean	39.2	37.0	34.2	33.5	29.0	29.4	29.9	31.1	31.3
(SD)	7.9	9.8	9.3	15.5	12.6	14.5	15.3	13.6	13.9
Period 2									
Mean	30.8	30.5	32.6	30.7	27.6	30.5	30.7	26.7	21.8
(SD)	11.6	16.2	13.8	15.8	9.8	12.4	15.4	16.3	16.1
Nonintervention									
Period 1									
Mean	33.3	29.6	29.0	26.8	23.8	23.8	26.0	24.3	23.6
(SD)	13.7	13.1	14.0	11.3	8.9	11.4	11.9	10.1	9.3
Period 2									
Mean	33.3	32.3	29.8	24.8	22.9	22.6	20.1	18.57	19.1
(SD)	10.0	13.9	15.6	16.8	15.8	15.8	15.2	14.8	15.0

Table 12

ANOVA: Poststartle Respiratory Rate

Source	Sum of Squares	Degrees of Freedom	F	P	Greenhouse Geisser P
Groups	3108.80	1	2.61	.12	
Error	28622.09	24			
Seconds	5275.35	8	7.63	.000	.000
Seconds X Groups	425.73	8	.62	.76	.62
Error	16583.60	192			
Seconds (lin)	4787.89	1	18.8	.0002	
Error	6111.52	24			
Seconds (quad)	200.92	1	1.65	.21	
Error	2922.76	24			
Seconds (cub)	5.51	1	.05	.83	
Error	2740.05	24			
Seconds (quart)	121.51	1	1.43	.24	
Error	2038.31	24			
Periods	893.36	1	1.08	.31	
Periods X Groups	92.98	1	.11	.74	
Error	19920.95	24			
Seconds X Periods	535.86	8	.96	.47	.44
Seconds X Periods X Groups	686.60	8	1.23	.28	.30
Error	13399.45	192			

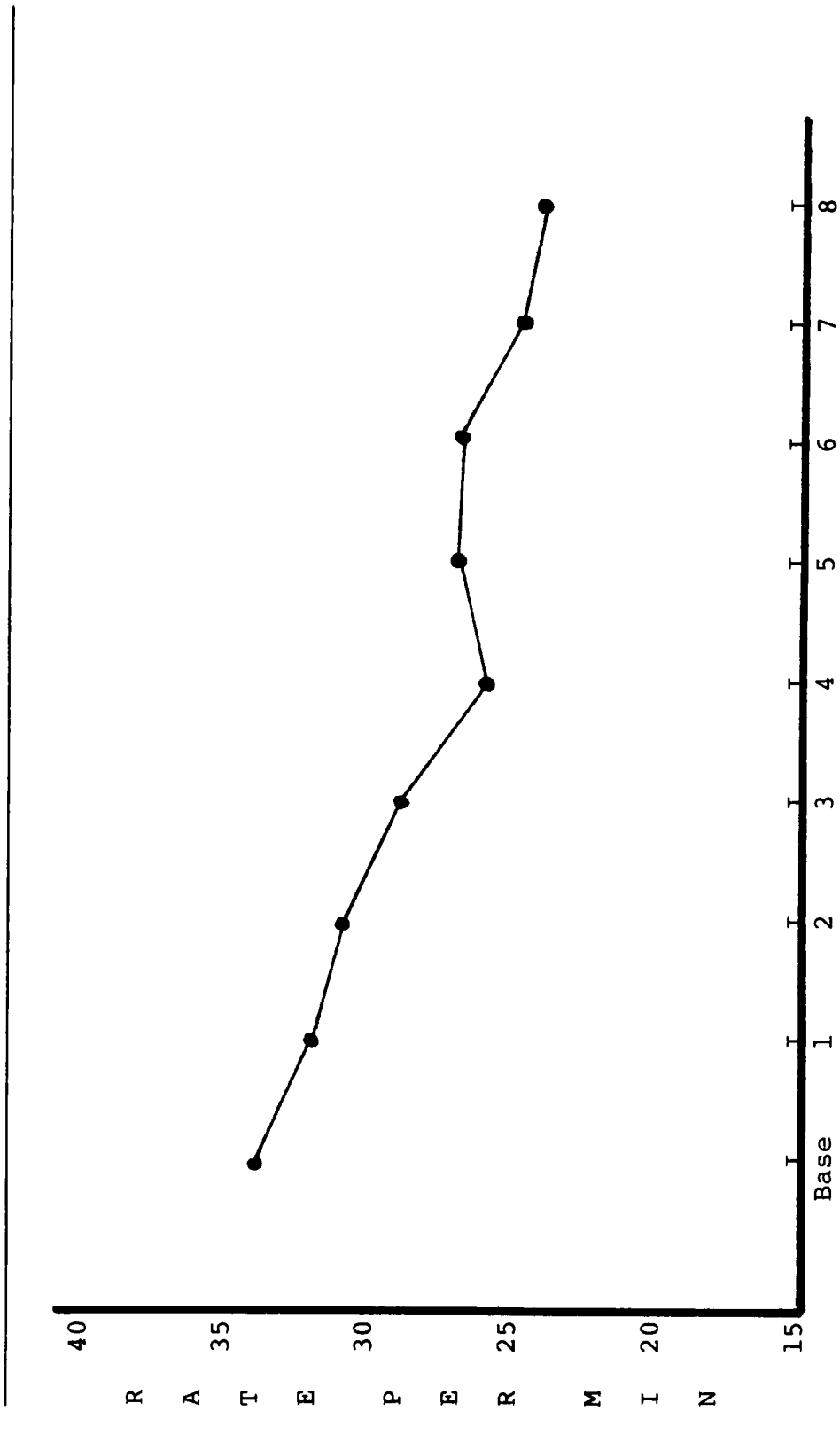

Figure 5. Poststartle Respiratory Rate, Averaged Across Periods and Groups

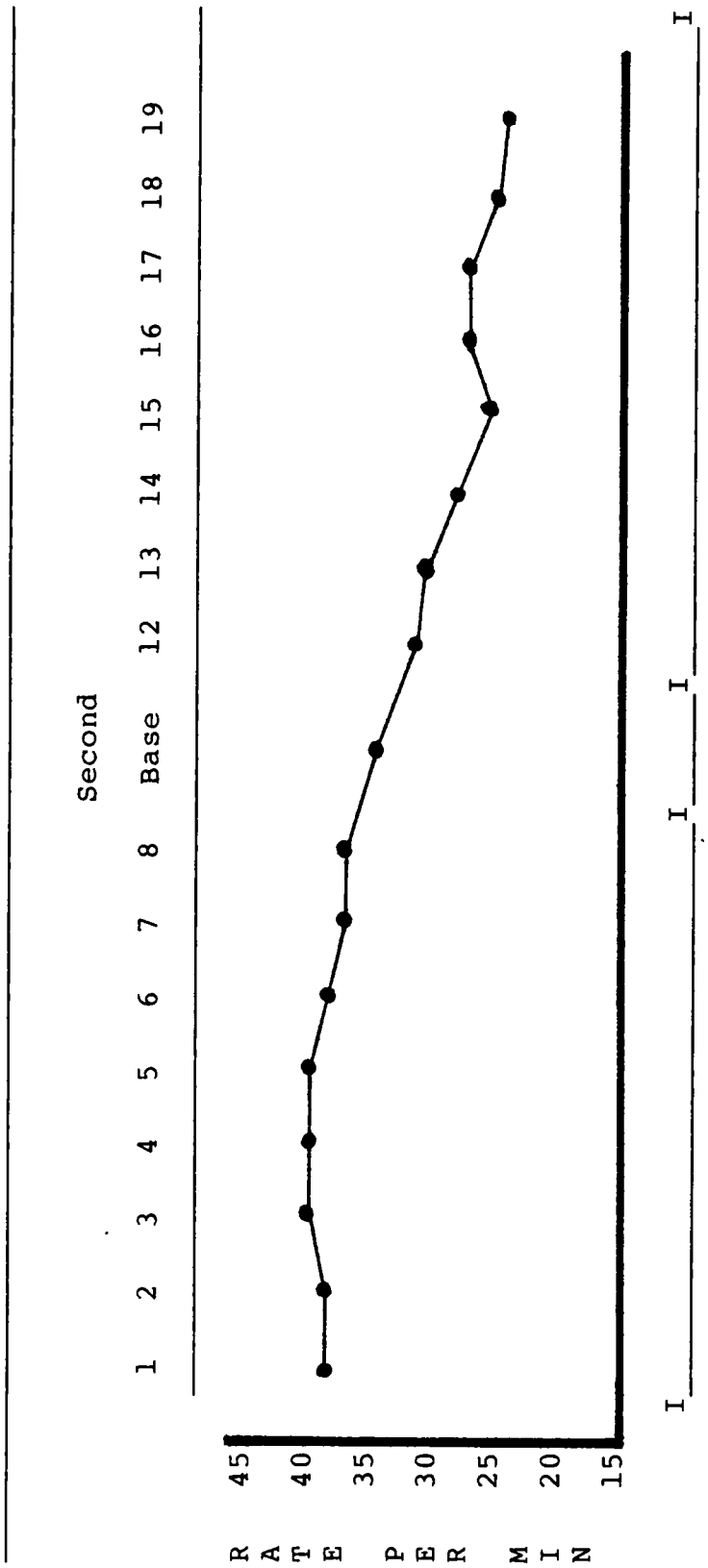

Figure 6. Pre- and Poststartle Respiratory Rate Averaged Across Periods and Groups

activity, the rate of decrease preceding the startle was compared to that following a startle via a 2 Groups X 2 Slopes (Prestartle and Poststartle) ANOVA. The results of this analysis indicated that the rate of change in the poststartle epoch ($\underline{X}$ = 1.14, SD = 1.41) was marginally greater than the rate in the prestartle epoch ($\underline{X}$ = .51, SD = 1.17), $\underline{F}(1,24) = 5.16$, $\underline{p} = .032$. There were no reliable main effects or interactions for the Groups factor.

The preceding analyses examined separately the prestartle and poststartle cardiac and respiratory activity. Another way to examine the relation of this activity to startles is to test the differences between the prestartle activity averaged across the eight seconds, the poststartle activity averaged across the eight seconds, and the activity averaged over the full half hour observation period. The measure of the activity for the observation period was the heart rate observed every thirty seconds averaged across each half hour of the observation. Cardiac and respiratory activity were examined in separate 3 Recording Epoch (prestartle, poststartle, full period) x 2 Group (auditory intervention- nonintervention) x 2 Period (1st half of observation - 2nd half of observation) ANOVAs.

Table 13 represents the means and standard deviations of heart rate by recording epoch, group and period. Table 14 represents the results of the analysis of these data. The

Table 13

Means and Standard Deviations of Heart Rate by Recording Epoch, Observation Period, and Group

	Group			
	Auditory Intervention		No Auditory Intervention	
	Observation Period		Observation Period	
Recording Epoch	1	2	1	2
Prestartle				
Mean	119.2	120.8	119.2	119.1
(SD)	11.2	14.5	10.5	11.3
Poststartle				
Mean	126.1	132.1	126.3	128.6
(SD)	11.0	9.0	9.3	11.4
Period				
Mean	125.7	129.7	128.0	130.3
(SD)	10.2	8.6	10.2	12.5

Table 14

ANOVA: Heart Rate By Recording Epoch, Observation Period, and Group

Source	Sum of Squares	Degrees of Freedom	F	p	Greenhouse Geisser p
Group	5.77	1	.01	.92	
Error	13476.63	24			
Epoch	2670.28	2	38.06	.000	.000
Epoch X Group	70.10	2	1.00	.38	.37
Error	1683.73	48			
Period	282.69	1	5.48	.028	
Period X Group	55.44	1	1.07	.31	
Error	1238.51	24			
Epoch X Period	77.80	2	2.39	.10	.10
Epoch X Period X Group	8.83	2	.27	.76	.76
Error	781.63	48			

only clearly reliable effect emerging from this analysis is the significant difference for the Recording Epoch factor, $F(2,48)=38.06$, $p < .001$ (Greenhouse-Geisser $p < .001$). A priori comparisons indicate that this significant difference is attributable to the mean prestartle heart rate being lower than those of the poststartle, $F(1,24)=76.6$, $p < .001$, or full period epochs, $F(1,24)=54.9$, $p < .001$, while the latter two do not differ, $F(1,24)=.008$. $p=.93$.

In addition to the significant Recording Epoch effect, the Period effect, $F(1,24)=5.48$, $p =.027$, closely approaches the designated significance level of .025. This effect indicates that there is a clear (but statistically nonsignificant) trend towards higher heart rate activity in the second half hour of the observation period than in the first. This trend supports the significant Period effect in the analysis of poststartle heart rate, but fails to support the lack of a Period effect in prestartle heart rate. Table 13, however, illustrates that this trend is likely due to the larger differences between Period 1 and Period 2 poststartle heart rate. Although the Period difference is greater for the poststartle heart rate than for the prestartle heart rate, this difference is not large enough to cause a reliable Recording Epoch x Period interaction. There were no significant main effects or interactions for the Groups factor, which is consistent with the results of

the analyses of the ongoing pre- and poststartle heart rate activity.

The means and standard deviations of respiratory rate by recording epoch, period, and group are represented in Table 15. Table 16 represents the results of analysis of these data. As was found in the analysis of heart rate activity, the only clearly significant effect which emerges from this analysis is the Recording Epoch effect, $F(2,48)=62.04$, $p < .001$ (Greenhouse-Geisser $p < .001$). A priori comparisons indicate that this effect is attributable to the prestartle respiratory activity being significantly lower than the average full-period respiratory rate, $F(1,24)=9.1$, $p < .01$, and the poststartle rate being lower that either the prestartle rate, $F(1,24)=61.4$, $p < .001$ or the average rate across the observation period, $F(1,24)=124.1$, $p < .001$. There were no reliable main effects or interactions for the Period and Group factors, although the Group effect approached the designated .025 alpha level.

Results of analyses of the means and variabilities of heart rate, respiration rate, and number of startles

The multivariate analysis of variance (MANOVA) was employed to examine the means and variabilities of heart rate and respiration rate, along with the number of startles emited by each infant. MANOVA techniques were chosen for these analyses for two reasons. First, MANOVA is the

Table 15

Means and Standard Deviations of Respiration Rate by Recording Epoch, Observation Period, and Group

	Group			
	Auditory Intervention		No Auditory Intervention	
	Observation Period		Observation Period	
Recording Epoch	1	2	1	2
Prestartle				
Mean	43.2	39.8	34.1	36.7
(SD)	8.1	9.5	10.8	8.1
Poststartle				
Mean	31.9	29.4	25.8	19.5
(SD)	11.0	8.1	9.5	9.9
Period				
Mean	42.2	43.3	39.6	38.3
(SD)	6.4	7.1	6.7	6.2

Table 16

ANOVA: Respiration Rate By Recording Epoch, Observation Period, and Group

Source	Sum of Squares	Degrees of Freedom	F	p	Greenhouse Geisser p
Group	1382.48	1	4.98	.04	
Error	6666.83	24			
Epoch	6017.08	2	76.44	.000	.000
Epoch X Group	115.07	2	1.46	.24	.24
Error	1891.53	48			
Period	107.08	1	3.55	.07	
Period X Group	.17	1	.01	.94	
Error	724.38	24			
Epoch X Period	150.70	2	2.62	.08	.10
Epoch X Period X Group	173.64	2	3.02	.06	.07
Error	1380.64	48			

analysis of choice when a set of dependent variables can be conceptualized as a system of interrelated parts. Second, the same logic that applies to the use of the ANOVA model rather than multiple t-tests applies to the use of MANOVA rather than multiple ANOVA tests (Hertzog & Rovine, 1985). Performing multiple ANOVAs inflates the probabilty of spuriosly significant results in the same way that performing multiple t-tests does. Reducing the overall number of tests via MANOVA reduces this probability. Both of these recommendations apply to the present analyses.

The first reason for using MANOVA is of particular importance in this case. One of the primary hypotheses of this study was that changing the pattern of cardiac and respiratory activity would affect the number of startles which an infant would emit. This hypothesis implies that spontaneous startles are related to cardiac and respiratory activity in a systematic way and that the auditory intervention will affect the whole system, thus making MANOVA the appropriate analysis technique. Thus, these analyses are important for testing both the effectiveness of the auditory intervention in changing the ongoing cardiac and respiratory activity of the infant, and more importantly, the relation of these changes to the occurrence of spontaneous startles.

The means and variabilities of cardiac and respiratory

activity are related to each other, but not necessarily affected by the auditory intervention in the same way. In order to ensure that a lack of effects on either rate or variability would not obscure possible effects on the other, two separate MANOVAs were performed. The mean heart rate, respiratory rate, and number of startles were analyzed together in a 2 Period (1st half of observation period - 2nd half of observation period) x 2 Group (auditory intervention - no intervention) x 3 Dependent Variables MANOVA. The variabilities of heart rate and respiratory rate were analysed in a separate 2 Period x 2 Group x 2 Independent Variables analysis. The Period factor was a repeated measures factor on both analyses.

The means and standard deviations of heart rate, respiratory rate, and number of startles are represented in Table 17. Table 18 represents the results of thes analyses, and the data are visually represented in Figure 7. The significance of the Box's M test ($p < .01$) indicates that the data violate the assumption of equality of variance-covariance matrices. Bartlett's tests of sphericity for Groups ($p = .34$) and Periods ($p = .20$) indicate that the data do not violate the assumption of sphericity. Violation of either of these assumptions biases the univariate tests of those effects that include that repeated measures factor

Table 17

Means and standard deviations of heart rate, respiration rate, and number of startles by Observation Period, and Group

Measure	Group: Auditory Intervention		Group: No Auditory Intervention	
	Observation Period		Observation Period	
	1	2	1	2
Heart Rate				
Mean	125.7	129.7	128.0	130.3
(SD)	10.2	8.6	10.3	12.5
Range	33.5	28.6	34.8	45.0
Respiration Rate				
Mean	42.2	43.3	39.6	38.3
(SD)	6.4	7.1	6.7	6.2
Range	23.2	24.8	22.0	20.4
Number of Startles				
Mean	11.2	4.5	7.4	8.6
(SD)	7.8	3.6	3.6	7.4
Range	30.0	12.0	14.0	26.0

Table 18

MANOVA: Heart rate, respiration rate, and number of startles by observation period, and group

Multivariate Analyses:

Box's M = 73.9, p = .000
Bartlett's Test of Sphericity for Groups = 1.74, p=.62
Bartlett's Test of Sphericity for Periods = 3.82, p=.28

Source	Wilks Value	Approx. F	p
Group	.890	.88	.46
Period	.739	2.58	.08
Group X Period	.635	4.20	.017

Univariate Analyses:

Source	Sum of Squares(df)	Error Sum of Squares(df)	F	p
Group				
Heart Rate	27.18(1)	4676.53(24)	.14	.71
Respiration Rate	185.82(1)	1919.68(24)	2.32	.14
Number of Startles	4.48(1)	1305.54(24)	.009	.93
Period				
Heart Rate	128.68(1)	598.71(24)	5.16	.03
Respiration Rate	.07(1)	190.05(24)	.009	.93
Number of Startles	96.94(1)	387.54(24)	6.00	.022
Group X Period				
Heart Rate	9.65(1)	598.7(24)	.39	.54
Respiration Rate	19.08(1)	190.05(24)	2.40	.13
Number of Startles	204.02(1)	387.54(24)	12.63	.002

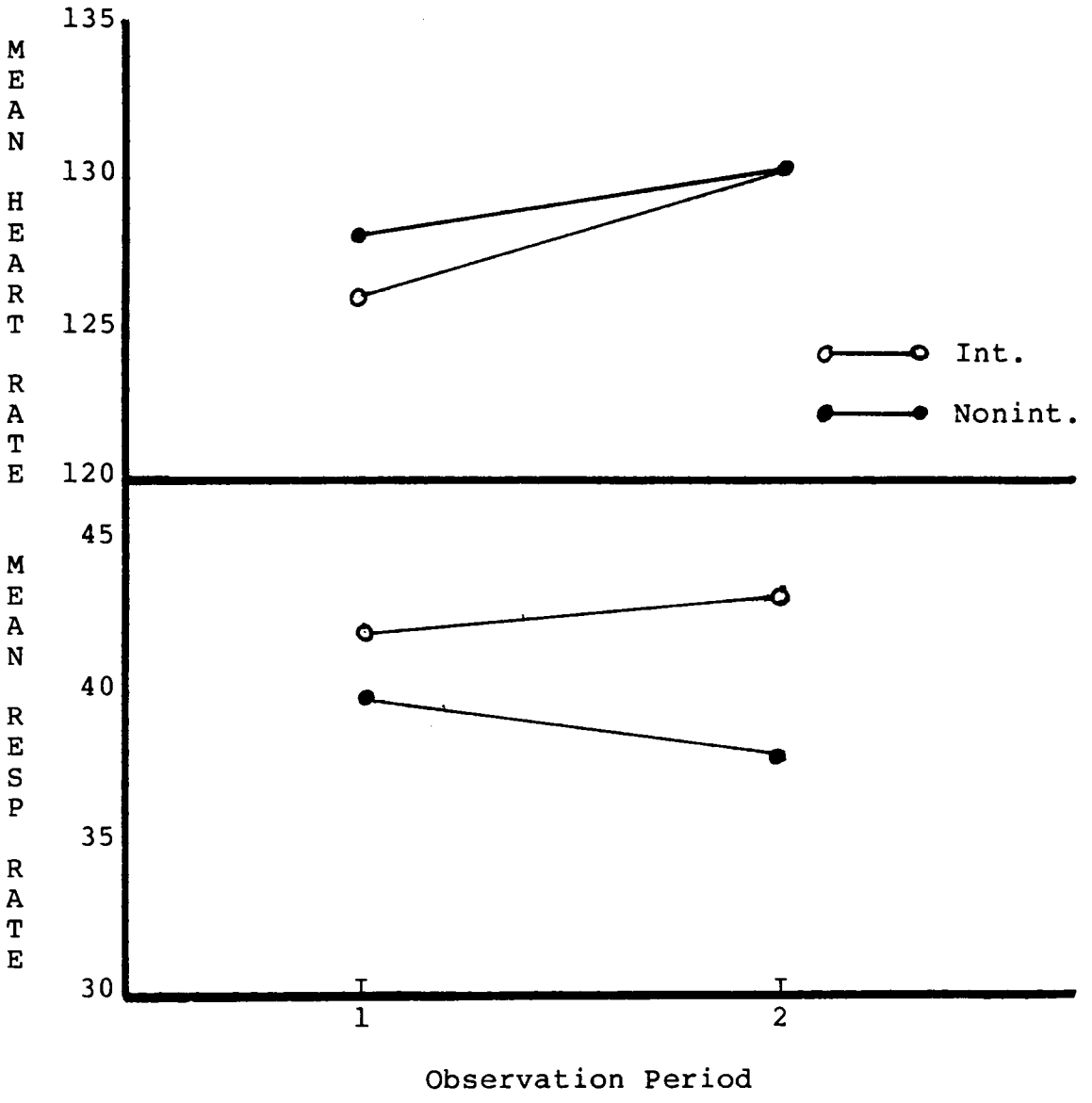

Figure 7. Mean Heart Rate, Respiratory Rate, and Number of Startles by by Periods and Groups

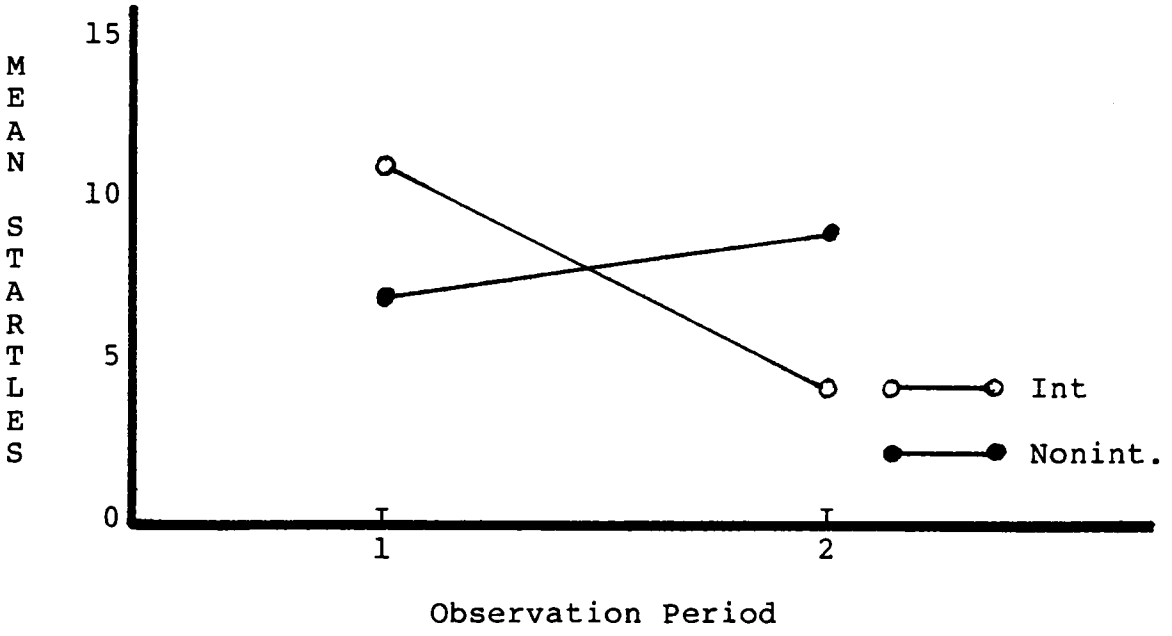

Figure 7 (continued). Mean Heart Rate, Respiratory Rate, and Number of Startles by by Periods and Groups

toward spuriously high rates of rejection of the null hypothesis (Herzog & Rovine, 1985).

The results of the multivariate analyses indicate that there was a reliable effect for the Groups by Periods interaction, $F(3,22)= 4.2$, $p=.015$. Examination of the univariate tests for this effect indicate that the reliable effect is attributable to a significant Groups by Periods interaction for the number of startles, $F(1,24)=12.62$ $p=.001$. Post hoc Neuman Kuels tests indicate that this effect is attributable to the fact that the number of startles emitted by the intervention group during period one was higher than their number of startles during period two, as well as being higher than the number of startles emmitted by the nonintervention group during the first period. The number of startles emitted by the intervention group in the second period was lower than the number emitted by the nonintervetion group in either period one or period two. Finally, the number of startles emitted by the nonintervention group did not differ across the periods. There were no significant effects at the multivariate level for the Period or Group factors. Because of the bias caused by the violation of the homogeneity of variance assumption, any conclusions drawn from these results should be supported by converging evidence.

Table 19 represents the means and standard deviations of the variabilities of heart rate and respiratory rate. These data are visually represented in Figure 9. The results of this analysis are presented in Table 20. The nonsignificance of Box's M (p =.21) combined with the nonsignifigance of Bartlett's test of sphericity for either the Group (p = .42) or Period (p = .48) effects indicates that these data do not violate the homogeneity and sphericity assumptions assumptions of the ANOVA model. Thus, given significant multivariate test results, the examination of the univariate test results is appropriate. As indicated, however, the multivariate test results detected no reliable effects for either the Group or Period factors.

Supplementary and supporting analyses

A possible reason for the lack of reliable effects in the analysis of the means and variability of heart rate, respiratory rate is the relatively large variability within each of these variables. For example, the auditory intervention group in observation period 1 had a mean heart rate of 125.7. The range of values for this variable was 35.2, with a minimum 106.5 and a maximum of 141.7. This large range suggests wide individual differences in this measure (see Table 17).

The major hypothesis which the above MANOVAs tested was that, to the extent that spontaneous startles are related to

Table 19

Means and standard deviations of heart rate variability and respiration rate variability by Observation Period, and Group.

Measure	Group: Auditory Intervention		Group: No Auditory Intervention	
	Observation Period		Observation Period	
	1	2	1	2
Heart Rate Variability				
Mean	6.2	7.4	7.8	8.5
(SD)	3.7	3.6	2.5	4.9
Range	14.4	9.5	14.7	19.2
Respiratory Variability				
Mean	8.4	10.3	6.4	6.9
(SD)	4.0	4.2	1.6	2.4
Range	13.8	4.9	14.4	9.1

Table 20

MANOVA: Heart rate and respiratory rate variability by observation period, and group

Multivariate Analyses:

Box's M = 16.05, p = .18
Bartlett's Test of Sphericity for Groups = .009, p = .923
Bartlett's Test of Sphericity for Periods = 1.03, p = .31

Source	Wilks Value	Approx. F	p
Group	.790	3.06	.07
Period	.812	2.65	.09
Group X Period	.939	.75	.48

Univariate Analyses:

Source	Sum of Squares(df)	Error Sum of Squares(df)	F	p
Group				
Heart Rate Variability	23.31(1)	423.20(24)	1.32	.26
Respiratory Variability	105.91(1)	406.17(24)	6.32	.02
Period				
Heart Rate Variability	13.64(1)	262.24(24)	1.24	.28
Respiratory Variability	17.36(1)	91.40(24)	3.81	.04
Group X Period				
Heart Rate Variability	.89(1)	262.24(24)	.08	.78
Respiratory Variability	6.46(1)	91.48(24)	1.70	.20

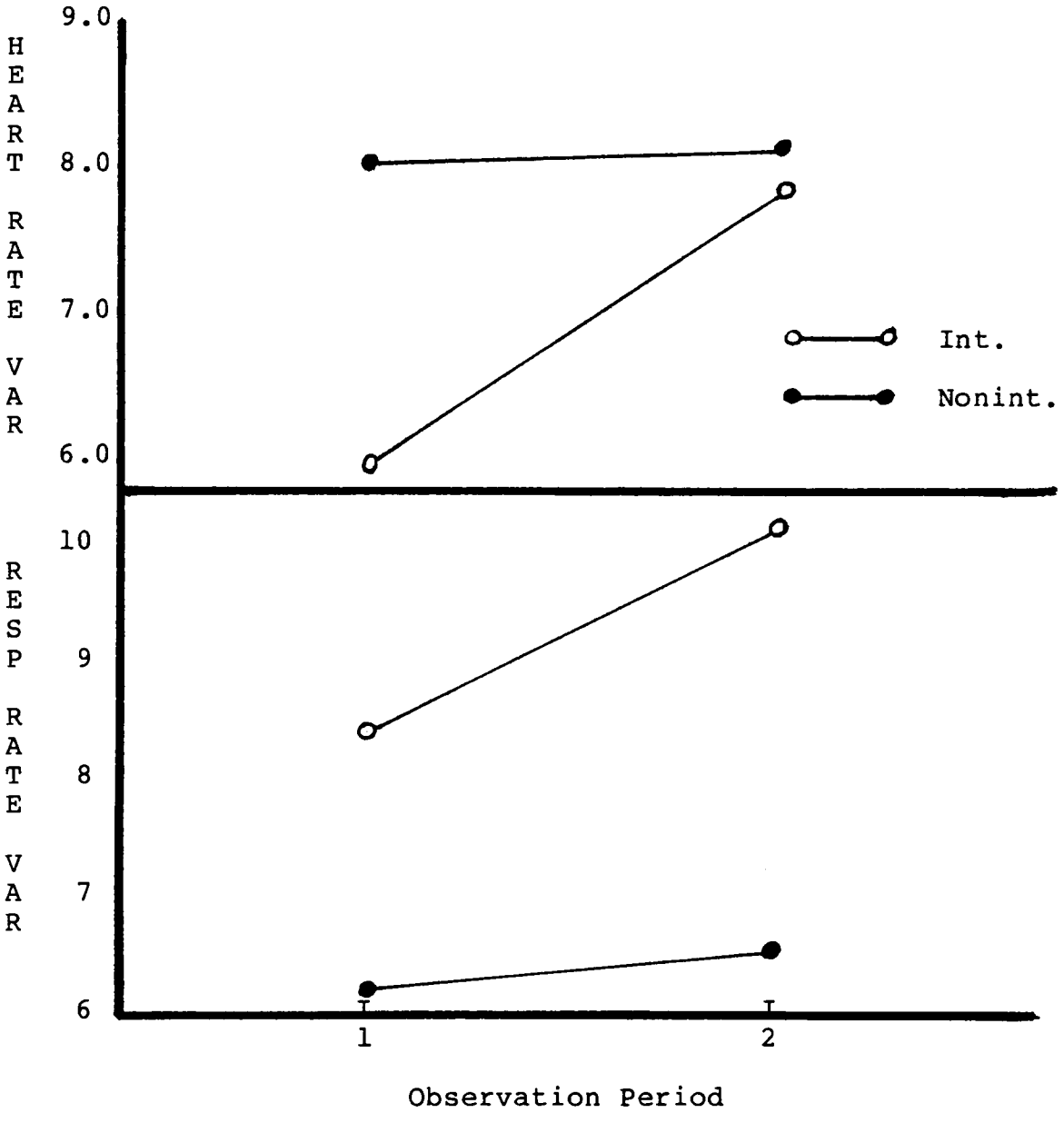

Figure 8. Heart Rate and Respiratory Rate Variability by Periods and Groups

the ongoing cardiac and respiratory activity, altering this activity by the application of an auditory stimulus would also affect the occurrence of spontaneous startles. Directionally, the expectation was that a decrease in heart rate and/or respiration rate would be associated with an increase in the number of startles. The tests of differences in these variables showed reliable effects on the rate of startles, but did not show any effects on heart rate or respiratory rate, probably in part because of the wide range of individual differences on these variables

Another way to test the effects of the auditory intervention is to examine the infants' heart rate, respiratory rate, and number of startles in the first half of the observation period relative to their rates in the second half. The hypothesis that the auditory stimulation would affect heart rate, respiratory rate, and number of startles leads to the prediction that the intervention group should show lower heart rates and respiratory rates and higher numbers of startles in the first half of the observation period than in the second. On the other hand, the nonintervention group should not show a differential distribution. For these analyses, infants were classiffied on the basis of whether they had lower heart rate, lower respiratory rate, and higher number of startles in period 1 or period 2. The distribution of lowest heart rate,

respiratory rate, and higher number of startles were analyzed in separate chi-square analyses for goodness-of-fit. If there was no relation between the auditory intervention and these variables, both the intervention and nonintervention groups should have been evenly distributed across the halves of the observation period.

Table 21 represents the results of the chi-square analyses of lower heart rate, respiratory rate, and higher number of startles within Groups by Periods. For the intervention group, the distribution of lower heart rate and higher number of startles significantly different from that expected if the auditory intervention did not have an effect ($\underline{X}^2(1)= 8.34$, $\underline{p} < .01$ for both heart rate and number of startles), indicating that more infants in the auditory intervention group showed a lower heart rate and higher number of startles in the first half of the observation period. The nonintervention group did not show differential distributions, ($\underline{X}^2(1)=.34$, $\underline{p} > .10$ for both heart rate and number of startles). For respiratory rate, neither group was differentially distributed, ($\underline{X}^2(1)=3.0$, $\underline{p} > .10$ for both groups).

One important consideration in the above analyses is whether the difference in the rate of startles between the first and second half of the observation period is large

Table 21

Chi-square Analyses for Goodness-of-Fit: Higher Number of Startles, Lower Heart Rate, and Lower Respiratory Rate

Higher Number of Startles				
Intervention Group	O	E	O-E	$(O-E)_2/E$
Higher Number of Startles, Period 1	11	6	5	4.17
Higher Number of Startles, Period 2	1	6	-5	4.17
			$\underline{X}^2(1)=$	8.34 $\underline{p} < .01$
Nonintervention Group				
Higher Number of Startles, Period 1	5	6	-1	.17
Higher Number of Startles, Period 2	7	6	1	.17
			$\underline{X}^2(1)=$	.34 $\underline{p} > .10$
Lower Heart Rate				
Intervention Group	O	E	O-E	$(O-E)_2/E$
Lower Heart Rate, Period 1	11	6	5	4.17
Lower Heart Rate, Period 2	1	6	-5	4.17
			$\underline{X}^2(1)=$	8.34 $\underline{p} < .01$
Nonintervention Group				
Lower Heart Rate, Period 1	7	6	-1	.17
Lower Heart Rate, Period 1	5	6	1	.17
			$\underline{X}^2(1)=$	.34 $\underline{p} > .10$

Table 21 (continued)

Chi-square of Goodness-of-Fit: Higher Number of Startles, Lower Heart Rate, and Lower Respiratory Rate

Lower Respiratory Rate				
Intervention Group	O	E	O-E	$(O-E)_2/E$
Lower Respiratory Rate, Period 1	3	6	-3	1.5
Lower Respiratory Rate Rate, Period 2	9	6	3	1.5
			$\underline{X}^2(1)=$	3.0 $\underline{p} > .10$
Nonintervention Group				
Lower Respiratory Rate Period 1	3	6	-3	1.5
Lower Respiratory Rate Period 2	9	6	3	1.5
			$\underline{X}^2(1)=$	3.0 $\underline{p} > .10$

enough to be meaningful. The MANOVA analysis of heart rate, respiratory rate, and number of startles tentatively demonstrated that the intervention group showed significantly more startles than the nonintervention group during the first period. Another way to examine these differences is to test the proportion of startles shown in each observation period. If the increase in the number of startles shown by the auditory intervention group during the intervention period is meaningful, the proportion of the number of startles shown in this period to the overall number of startles should be significantly higher than 50%, which would be expected by chance.

Table 22 represents the results of the analyses of the proportions of startles shown by each group in each period. These results indicate that the proportion of the intervention group's total number of spontaneous startles that occurred during the auditory intervention was significantly greater than the 50% that would be expected by chance and the proportion of their startles that occurred in the second half of the observation period was significantly lower. The nonintervention group did not show a differential distribution of proportion of startles.

Another way to examine the relation between cardiac and respiratory activity and spontaneous startles which derives directly from the above chi-square analyses is to examine

Table 22

Proportions of Startles

Proportion of startles for each Group and Period compared to an expected proportion of .50.

	Proportion	z	p
Intervention Group, Period 1	.72	2.20	< .025
Nonintervention Group, Period 1	.46	.40	> .10
Intervention Group, Period 2	.27	-2.30	< .025
Nonintervention Group, Period 2	.54	.40	> .10

the concordance of these variables. Concordance was determined in the following way. For the chi-square analyses each infant was classified as having a higher number of startles, lower heart rate, and lower respiration rate in the first or second half of the observation period. For the analysis of concordance, infants were classified as concordant for heart rate if they had their lower heart rate in the same period as their higher number of startles. Likewise, infants were classified as concordant for respiration if they had their lower respiration in the same period as their higher number of startles.

The analysis of concordance is important because it allows a more detailed examination of the relation of heart rate and respiratory rate to spontaneous startles than do the chi-square analyses. For example, the chi-square analyses indicate that more infants in the intervention group showed both lower heart rate and higher rates of startles during the intervention period, while the nonintervention infants were distributed evenly between the two observation periods. These analyses do not examine whether, for a particular infant regardless of group, the highest rate of startles occurred during the same period as either the lower heart rate or respiration rate. This is the question at which the analysis of the rate of concordance is directed.

Table 23 demonstrates the concordance between high number of startles, low heart rate, and low respiratory. For heart rate, 75% of the infants were concordant, while for respiration, 50% were concordant. Thus, lower heart rate was associated with a higher number of startles in more infants than was lower respiration rate. If the number of children who were concordant on either heart rate or respiration rate is examined, however, this figure rises to 96%. Thus, all but one infant had either a lower heart rate or a lower respiration in the same period as they had their higher number of startles.

Because the infants can be viewed as being concordant or discordant, these concordance rates were analyzed via a binomial test of the proportions showing concordance. Table 23 represents the results of these analyses. The concordance rate for lower respiratory rate and higher number of startles was not different from that which would be expected by chance, $z=0$, $p > .05$. The concordance rate for lower heart rate and higher number of startles was reliably different from that expected by chance, $z=2.5$, $p < .01$, as was that for concordance of either heart rate or respiratory rate, $z=4.6$, $p < .01$. These results indicate that, beyond the relation indicated by the chi-square results, spontaneous startles are associated with lower heart rates, regardless of the group that the infant was in. While

Table 23

Binomial Test of the Concordance of Number of Startles, Low Heart Rate, and Low Respiratory Rate

Concordance	Proportion	*z*	*p*
High Startles, Low Heart Rate	.75	2.5	< .01
High Startles, Low Respiration	.50	0.0	> .10
High Startles, Low Heart Rate *or* Low Respiration	.96	4.6	< .01

examination of the concordance of respiraton and startles showed no significant relation, if the concordance of respiration or concordance of heart rate with spontaneous startles is examined, all but one infant was concordant.

The pattern of state behavior is also useful for examining the effects of the auditory intervention. State was not analysed parametrically because it is at best an ordinal variable. Active sleep is the dominant state of newborn infants (Petre-Quadens, 1967). Although any infant might show more occurrences of quiet sleep than active sleep in a half hour period, one would expect these infants to be randomly distributed between the two groups and across the halves of the observation period. Further, one would expect that in both groups in both periods most infants should show more observations of active than quiet sleep.

For this analysis infants were classified on the basis of whether they spent more than half of each half of the observation period in quiet sleep or active sleep. Chi-square analyses indicated that the intervention group had equal numbers of infants showing more observations of quiet sleep (6 infants) and of active sleep (7 infants), while the nonintervention group had unequal numbers of infants showing more quiet sleep (2 infants) and more active sleep (11). Table 24 represents these analyses. Chi-square analyses of the data for the second half of the observation

Table 24

Chi-square Analysis of Goodness-of-Fit for Infants Showing More Quiet or Active Sleep by Periods and Groups

Intervention Group	O	E	O-E	$(O-E)^2/E$
Period 1				
More Quiet Sleep	6	6.5	-.5	.04
More Active Sleep	7	6.5	.5	.04
			$\underline{X}^2(1)=$	.08 $\underline{p} > .10$
Nonintervention Group				
More Quiet Sleep	2	6.5	-4.5	3.11
More Active Sleep	11	6.5	4.5	3.11
			$\underline{X}^2(1)=$	6.22 $\underline{p} < .025$
Period 2				
Intervention Group	O	E	O-E	$(O-E):2/E$
More Quiet Sleep	0	6.5	-6.5	6.5
More Active Sleep	13	6.5	6.5	6.5
			$\underline{X}^2(1)=$	13.0 $\underline{p} < .01$
Nonintervention Group				
More Quiet Sleep	1	6.5	-5.5	4.65
More Active Sleep	12	6.5	5.5	4.65
			$\underline{X}^2(1)=$	9.30 $\underline{p} < .01$

period indicated that both groups had more infants showing more observations in quiet sleep than in active sleep. Thus, only when the infants were exposed to the auditory stimulation was there an equal occurrence of infants displaying quiet and active sleep.

Discussion

The results of this study support the hypotheses that 1) the system that mediates spontaneous startles is related to the system that mediates evoked startle respones, 2) the occurrence of spontaneous startles is related to the ongoing homeostatic activity of the infant, especially to periods of low arousal, and 3) affecting the ongoing activity via an auditory intervention affects the occurrence of startles. The results do not support the hypothesis that spontaneous startles will be related to the homeostasis of the infant via an increase in the poststartle respiratory rate of the infant. The results will be discussed as they pertain to these hypotheses.

The hypothesis that the system which mediates spontaneous startles is related to the system that mediates the evoked startle response is supported by the analyses of the poststartle cardiac and respiratory activity. In relation to heart rate, there was a reliable poststartle HR increase of approximately nine beats per minute following a startle, with heart rate returning before the end of the eight seconds to a level that did not differ from the base level preceding the startle. While the significant Period effect on this analysis indicates a general rise in the poststartle heart rate during the second half of the observation period, the lack of an interaction of the Period factor with any of the Seconds trends indicates that the

auditory intervention did not affect the trends in the poststartle heart rate.

The Seconds by Periods by Groups interaction on the poststartle heart rate analysis bears discussion. This was the only effect on which the results of the traditional significance test and the Greenhouse-Geisser corrected test differed. Herzog and Rovine (1985) indicate that in the case of a dissagreement between the results of the traditional tests and the corrected tests, the "true" probability of the effect actually lies somewhere in between the two results. In this case, fortunately, the Seconds (linear) by Periods by Groups interaction provides support for the conclusion of the corrected test, that the Seconds by Periods by Groups interaction indicates a strong, but statistically nonsignificant, tendency of the intervention group to show a delayed return of heart rate to the baseline level following a startle. If this result was reliable, it could indicate a rebound effect in the period after the removal of the auditory stimulation. There is no evidence from the analysis of prestartle HR activity that the intervention group showed greater depression of HR during the auditory intervention, providing little support for the suggestion of a rebound effect.

The poststartle respiratory activity also reflects the occurrence of a spontaneous startle. There is a reliable decrease of approximately 9 inspirations per minute

following the startle. This rate of decrease is marginally greater than the rate of decrease in the prestartle respiratory rate, as indicated by the comparison of the pre- and poststartle slopes. Also, the average poststartle respiratory activity was lower than either the average prestartle activity, or the average activity across the observation period. The lack of reliable effects of the Periods and Groups factors, or interactions of these factors with the Seconds factors or trends indicates that these poststartle respiratory changes were not affected by either the passage of time or the auditory intervention.

The poststartle changes in both cardiac and respiratory activity parallel those reported in studies of evoked startle responses. Evoked startles in infants are reliably accompanied by rapid heart rate acceleration (Bridger & Reiser, 1959; Campos & Brackbill, 1973; Clifton, & Nelson; Graham et al., 1968; Lipton & Steinschneider, 1964), and initial slowing of respiration (Steinschneider, 1968). These results, combined with the results of previous studies which reported that spontaneous startles disappear in the second month of life (Wolff, 1966), relate the spontaneous startle to both the evoked startle and the Moro reflex and support the hypothesis that the mechanism mediating spontaneous startles is related to the midbrain and brainstem mechanisms that mediate the evoked startle reponse (Davis et al., 1982). To the extent that spontaneous startles are mediated

by a midbrain and brainstem mechanism, this finding supports the hypothesis that the spontaneous startle mechanism is related to other systems involved in homeostatic control in the newborn.

Clearly, this one result is only suggestive, and not compelling, evidence that spontaneous and evoked startles are mediated by the same system. One counter-arguement is that the cardiac and respiratory changes are an artifactual byproduct of the activity changes, however, mediation of the physiological changes by a separate, parallel, central mechanism is supported by the findings of Freychuss (1970), that the intention to grasp is accompanied by heart rate changes in subjects whose arms are immobilized by neuromuscular blockade. Future research might examine the neural mechanism of spontaneous startles more thoroughly through the use of an animal model such as that employed by Davis and his colleagues to examine the neural mechanisms of the evoked startle response.

The second major hypothesis of this study, that spontaneous startles are related to periods of low arousal in the ongoing homeostatic activity of the infant is supported by the analyses of the prestartle levels of, and trends in, heart rate and respiratory rate activity. First, there was a reliable decrease in respiratory rate across the eight second epoch directly preceding a startle. Second, the average respiratory rate during this epoch was significantly

lower than the average rate across the full observation period. Third, while there were no reliable trends in heart rate across these eight seconds, the average heart rate during this epoch was significantly lower than the average heart rate across the observation period. These results indicate that the occurence of a spontaneous startle is related to a period of low or declining activity.

Supporting the results of the analyses of the poststartle activity reported previously, the auditory intervention did not affect the level of heart rate or respiratory rate at which startles occurred, nor did it affect the trends in cardiac or respiratory rate preceding a startle. This conclusion is indicated by the lack of reliable Group effects or interactions in these analyses. In other words, when a startle occurred it was preceded by a period of lowered heart rate and declining respiration that was not different across the intervention and nonintervention groups. This is an important result in that it suggests that there is a basic homeostatic level that is related to the occurrence of startles.

The conclusion that there is a basic homeostatic level at which startles occur is further supported by the finding that the passage of time, as tested by the Period factor, did not affect the relation of the ongoing cardiac or respiratory activity to startles. In relation to the epoch preceding a startle, the Period factor showed a trend which

approached significance in the test of the differences in the prestartle, poststartle, and average heart rates. This result indicates a general rise in the heart rate level across the two halves of the observation period, but inspection of Table indicates that this trend was predominantly related to period differences in the poststartle heart rates across the periods, which is supported by the reliable Period effect in the analysis of the poststartle heart rate changes. Thus, while there was a trend toward higher heart rates across the halves of the observation period, this trend did not affect either the level of, or the trends in heart rate or respiration rate preceding a startle.

The results of the chi-square analyses also support the conclusion that a period of lowered heart rate is related to the occurrence of spontaneous startles. First, those infants in the intervention group showed both a lower average heart rate and a higher number of startles during the period in which they were exposed to the auditory stimulus. Second, regardless of group status, a significant proportion of the infants showed concordance between the period in which they had their lower heart rate and the period in which they had their higher number of startles. Neither of these results would be expected if the level of heart rate was unrelated to the occurrence of spontaneous startles.

The results of the chi-square analyses of the relation

of respiratory rate to spontaneous startles lead to less clear conclusions. There was no evidence of a differential distribution of lower respiratory rate related to groups as there was in the analysis of heart rate. Further, there was no indication of concordance between low respiratory rate and higher number of startles. When concordance of the period of higher startles with either heart rate or respiratory rate was considered, however, all but one infant's period of higher startles was accounted for.

The chi-square analysis of the infants' state behavior also provides support for the hypothesis that spontaneous startles are related to periods of low arousal. This analysis demonstrated that the group that was exposed to auditory stimulation had a larger number of infants who were observed more in quiet than in active sleep. This group also had more infants who had their higher rate of startles during the auditory intervention sleep. These results support the results of the chi-square analyses of lower heart rate and higher number of startles.

The results of the above reported analyses of prestartle cardiac and respiratory activity combined with the supporting chi-square analyses indicate that the occurrence of spontaneous startles is related to periods of low autonomic arousal in the infant. Spontaneous startles appear to be most strongly related to cardiac activity, evidenced by the period of low heart rate preceding a

startle and by the concordance of lower heart rate and increased number of startles. Startles also appear to be related to respiration. Respiratory rate showed a reliable decline during the eight second epoch preceding a startle, with the mean respiratory rate during this epoch being lower than the average respiratory rate across the observation period. The concordance results do not indicate a strong relation between respiration and startles, however some degree of relation is suggested by the finding that the addition of those infants who were concordant for respiration and number of startles to those who were concordant for heart rate and number of startles accounts for the higher startle periods of all but one infant. Finally, more infants in the intervention group showed more observations of quiet sleep in the same period as this group showed their higher rate of startles.

The MANOVA analyses of the means and variabilities of heart rate and respiratory rate only partially support the hypothesis that changing the ongoing activity through an auditory intervention would affect the occurrence of spontaneous startles. At the multivariate analysis level there was a reliable Periods by Groups interaction. The univariate analyses indicate that the rate of startles of the intervention group was increased during the auditory stimulation and decreased after the stimulation was removed, but there were not concurrent changes in the infants' heart

rates and respiratory rates, as tested by the MANOVAs. Likewise, there was no evidence that heart rate variability or respiratory rate variability were affected by the auditory stimulation. The violation of the assumption of homogeneity of the variance-covariance matrices indicates that any conclusions drawn from the the univariate analyses should be tentative, and should require converging support from other analyses.

The chi-square analyses provide converging evidence that the auditory intervention affected the occurrence of spontaneous startles. As reported above, the auditory intervention group showed a differential distribution of startles, with more of the infants having their higher number of startles in the first period. The nonintervention group did not show a differential distribution of startles, suggesting that the differential distribution was related to the auditory intervention and not to the passage of time. Further support comes from the finding that the proportion of startles shown by the intervention group infants during the auditory stimulation was reliably greater than that shown after the removal of stimulation.

While the MANOVAs did not detect significant group differences in the heart rate or respiratory rate, the results of the chi-square analyses also bare on this question. In the intervention group, 11 of the 12 infants showed lower heart rates while exposed to the auditory

stimulation and 10 of 12 infants were concordant for lower heart rate and higher number of startles during the auditory intervention. The nonintervention group did not show a differential distribution of heart rate and startles in relation to the periods. Thus, while the MANOVAs did not detect reliable heart rate differences, the results of the chi-square analyses indicate that higher rates of startles were related to the auditory intervention and, importantly, lower heart rates were associated with the auditory intervention and occurred concurrently with the higher rates of spontaneous startles.

Finally, the chi-square analysis of state behavior also supports the conclusion that changing the ongoing behavior of the infant affected the occurrence of spontaneous startles. More infants showed a higher number of observations in quiet sleep during the auditory intervention. In the nonintervention group, and in the intervention group after the auditory stimulation period, most infants showed predominantly active sleep.

In summary, the results of the MANOVA analyses and the supporting chi-square analyses support the conclusion that changing ongoing activity of the infant through the application of an auditory intervention increased the occurrence of spontaneous startles. First, the infants in the intervention group emitted more startles during the auditory intevention. Second, there was not a differential

distribution of startles in the nonintervention group. Third, the more infants in the intervention group had there lower heart rate period during the intervention, as well as showing more observations of quiet sleep. These results would not be expected if the auditory intervention had no effect on either the ongoing behavior of the infant or the occurrence of spontaneous startles.

The results of the analyses of poststartle respiratory changes do not support the hypothesis the startles would be followed by an increase in the respiratory activity of the infant. In fact, the infants' respiratory activity decreased reliably following a startle. While this result is contrary to the hypothesized result, it does not contradict the hypothesis that spontaneous startles might be related to the ongoing homeostatic activity of the infant. One shortcoming of this study is that the method used to measure respiration cannot provide information about the depth of respiration. While the rate of respiration is important, it is not the only parameter which affects the amount of oxygen that the infant receives. The ventilation of the infant is affected by both the rate and the depth of respiration. The poststartle decrease in respiratory rate could increase the ventilation of the infant if it was accompanied by an increase in the depth of respiration. Unfortunately, it is difficult to measure the depth of respiration in infants without changing their pattern of respiration.

This point is also important in relation to the prestartle respiratory activity. In this study, it is not possible to determine whether the reliable decline in respiratory rate preceding a startle was associated with a concurrent change in the depth of respiration. A decrease in the depth of respiration, combined with slowing of respiration could have a large effect on the ventilation of the infant. One way to examine this speculation in future research is to use a more direct measure, at a different level, of the infant's homeostatic activity, such as measuring transcutaneous oxygen pressure ($TCPO_2$) or oxygen saturation. Use of one of these measures would allow the examination of both the pre- and poststartle homeostatic activity of the infant at a different level than that examined in this study. Examination of the activity at this level would provide the ability to determine whether the decrease in respiratory activity preceding the startle is related to decreasing oxygenation, and whether the decrease in respiratory rate following a startle is related to increasing oxygenation, both of which are consistent with the hypotheses of this study.

The results of this study are interesting in relation to the suggestions of previous studies about the possible functional significance of spontaneous startles. Wolff (1966) suggested that spontaneous startles occur to release energy which would otherwise wake the infant. This

suggestion implies that spontaneous startles should be preceded by periods of increasing arousal, and that the period following a startle should show signs of decreasing arousal. In this study, startles occurred during periods that were characterized by low or decreasing arousal as indexed by cardiac, respiratory, and state activity, and were followed by increases in cardiac activity. These results differ from those that would be expected based on Wolff's "energy release" model.

These results also apply to Korner's suggestion that startles might serve the homeostatic function of providing necessary stimulation for the infant's developing nervous system during those periods in which the infant is relatively inaccessible to external stimulation. The results of this study do not entirely support Korner's suggestion. The highest rate of startles occurred during the period in which the infants were directly exposed to the highest level of stimulation. This result would not be expected if the function of startles was to provide stimulation during periods of "deafferentation". If that were the case startles should occur more frequently in those infants who were unstimulated.

Interestingly, in this study those infants who were stimulated showed lower heart rates and lower state activity in association with the increase in startles. This effect might be a function of the repetitive nature of the

stimulation. As the infants habituate to the almost constant stimulation, their level of arousal decreases. If the stimulation were more variable the results might have supported Korner's suggestion by demonstrating that those infants who were less stimulated showed fewer startles.

In addition, these results provide evidence that there is a more proximal functional mechanism for the production of spontaneous startles than to provide stimulation for future development. Whether the stimulation provided by spontaneous startles also functions to aid in the infant's development is possibly an empirical question. The self-stimulation provided by other "spontaneous" behaviors has been theoretically and empirically related to later development in both animal and human models. For example, Gottlieb's (1975) research with ducklings indicates that the auditory stimulation provided by their own calling prior to hatching is necessary for the proper development of their recognition of maternal calls after hatching. Also, Thelan (1981) suggested that an infant's rhythmical behavior may provide stimulation which aids in organizing behavioral development.

Speculation

One way in which spontaneous startles might be related to later development is via the prevention of sudden infant death syndrome (SIDS). One indication that startles might be related to prevention of SIDS is the finding that startles

disappear during the second month of life, which is the period reported as the beginning of the highest risk period for SIDS (Valdes-Dapena, 1979). Rovee-Collier and Lipsitt (1982) suggested that the apparent shift to cortical control mechanisms at around two months of age might be a precipitating factor in the increase of cases of sudden infant death. During the period of inhibition associated with this change, those mechanisms which prevent the infant from succumbing to SIDS during the neonatal period would be supressed, while some infants might not have developed new mechanisms to avert these life threatening periods of low arousal. Spontaneous startles might be one behavior which helps to arouse infants from periods of dangerously low activity during the neonatal period, but which disappears later.

In order to examine further the mechanisms and possible importance of spontaneous startles, future research might focus on individual differences in the display of spontaneous startles, and the relation of these differences to infant characteristics. For example, this study and previous studies of elicited startles have primarily focused on relatively healthy infants. Examination of the characteristics of spontaneous startles, both the pattern and physiological concomitants, in infants who show higher numbers of risk characteristics might contribute to the understanding of this behavior.

One point that requires attention is the difference between the groups in one minute Apgar scores. The average score for the nonintervention group was significantly lower than that for the intervention group (see Figure 2). Could the differences found in this study be attributable to this difference in mean Apgar scores? This suggestion appears unlikely for at least two reasons. First, the difference in the Apgar scores is attributable to a very low one minute score (3) for one infant in the nonintervention group. This infant's condition had improved by five minutes (Apgar score = 9), which is reflected in the lack of group differences on the five minute Apgar scores. It is unlikely this infant was the sole cause of the differences found in this study.

The second reason that it is unlikely that the effects found in this study could be attributed to the differences in one minute Apgar scores is that most of the effects were not between group effects. For example, the analyses of heart rate and respiratory rate changes in relation to spontaneous startles showed no group differences in the trends preceding or following a startle. Where there were group differences, such as in the number of startles, these differences were clearly attributable to changes in the activity of the intervention group, and not to the activity of the nonintervention group. Thus, the conclusion that the differences found in this study are attributable to the group differences in one minute Apgar scores is not

supported.

In conclusion, the results of this study support the hypothesis that the system that mediates spontaneous startles is related to the system that mediates the evoked startle response. In addition these results of this study strongly support the suggestion that spontaneous startles serve a homeostatic function related to periods of low arousal indexed by cardiac and respiratory activity. The application of auditory stimulation provided strong support for this suggestion by demonstrating the concurrent lowering of heart rate and state activity and increasing rate of startles. Spontaneous startles appear to be more strongly related to cardiac activity than to respiratory activity, but future work should examine the relation between the oxygenation of the infant (as a more direct measure of homeostatic activity) and the occurrence of spontaneous startles. Future work should investigate the developmental course of spontaneous startles in order to elucidate developmental trends and the possible relation between this behavior and the prevention of sudden infant death syndrome.

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Appendix 1:

LETTER OF INFORMED CONSENT

Dear Parent:

As you know, crying is something that babies frequently do. For many mothers the second of their baby's cry tells them what the baby wants or needs. For example, some mothers say that they can tell if their babies are tired or hungry or if they need changing by the way the cry of their baby sounds. The cry sound may also tell something about how well the baby is after its birth. For this reason we are examining the cries and other behaviors of babies recently born so that we may better understand what the sound of the cry may tell us about the baby's health and future development. We would like you to help us by giving us permission to examine your baby.

First, we will record the important facts surrounding your baby's birth from the medical records and from questions we ask you in a short interview. Then we will conduct what is called a Brazelton Neonatal Behavioral Assessment Scale examination. For about 20 minutes we will examine how well your baby responds to different sights and sounds such as bells, rattles and peoples' faces and how much your baby likes to cuddle and be held.

Then for about 90 minutes we will let your baby rest in its crib while we watch the rate of its heart beats and breathing as determined by a monitor that your baby is attrached to. We will also watch how your baby sleep during this time.

Then, by snapping the baby's foot with a rubber band just hard enough to make the baby cry we will be able to tape record a cry in the nursery for later examination in the office. By the way, making the baby cry is a regular part of the medical examination that all babies get; using the rubber band is our way of making sure that each infant's cry is the same.

To see how your baby's cry and other behaviors change, we may examine your baby on different days during your stay in the hospital. This procedure will present no harm to you or your baby and all findings will be kept confidential. Although this examination will not directly benefit your baby, the findings of this examination may help mothers and their babies in the future. We could really use your help.

I understand that the examiners will answer any questions I have concerning the examination of my baby. I understand that I may withdraw from this examination at any time and may prevent my baby from taking part.

I AGREE TO PARTICIPATE.

_____ _____

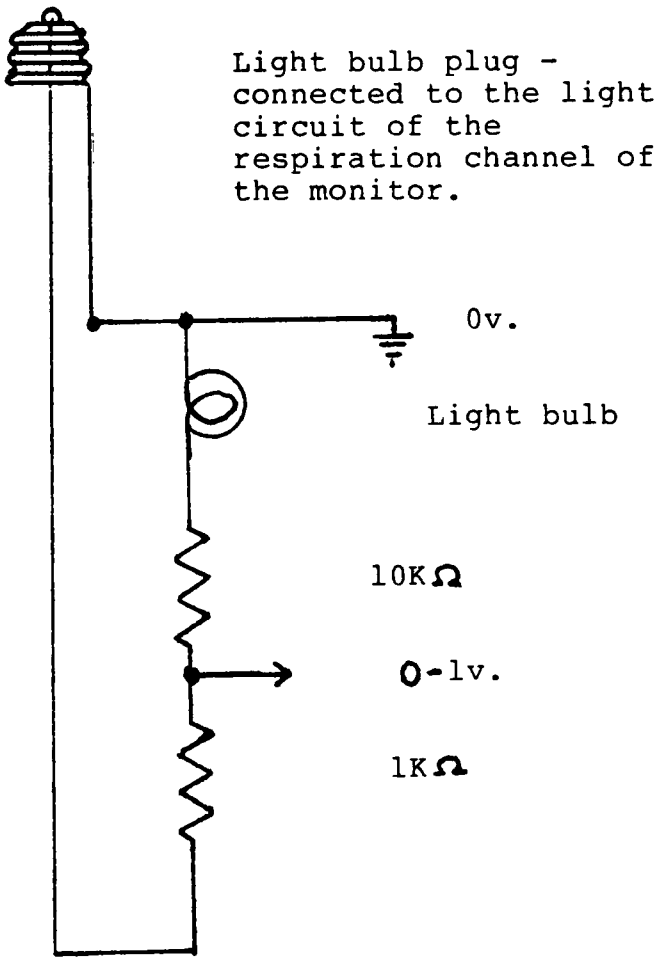
(Mother's signature) (Date)

_____ _____

Philip Sanford Zeskind, Ph.D. (Date)
Examiner

Appendix 2

Circuit designed to take signal from the light output of the monitor



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