THE HEART RATE RESPONSE TO NONSIGNAL AUDITORY STIMULATION WITH RESPIRATION CONTROLLED

By

GERALD W. RITTER

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Hanover College

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Thesis Approved:

Arthur E. Harrison
Thesis Adviser

Julia L. McGuth

Donald K. Fromme

D. J. Burham
Dean of the Graduate College

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

The purpose of this study is to determine the nature of the heart rate (HR) component of the orienting reflex (OR). The OR is a generalized response which is elicited by novel stimuli and facilitates the reception and evaluation of these stimuli. Since Pavlov (1927) first described the OR, considerable research has been conducted to determine the significance and principle characteristics of the OR. Research has determined that the OR is of primary importance in the processing and evaluation of stimuli in the environment. This fact has led many researchers to believe that the OR may be of central importance in the understanding of many abnormalities of behavior (Maltzman and Raskin, 1965; Lynn, 1963).

In recent years the widespread use of electro-physiological measures in behavioral research has given impetus to research on the OR. Unfortunately, this research has been hampered by the frequent use of inadequate or invalid physiological measures. Many studies have utilized single, superficial physiological measures of the OR, or have employed more complex measures which were later determined to be of questionable validity (e.g. GSR, vasomotor measures). Therefore, before any meaningful research at the physiological level on the OR can be conducted, the precise physiological components of the OR must be further explicated. For example, the HR component of the OR has been

the object of considerable research recently, but an examination of the literature reveals that present knowledge of the cardiac component of the OR is both confused and contradictory.

Many different procedures of measuring HR have been used and this has contributed to the confusion that exists concerning the HR component of the OR. The ideal HR measure permits an examination of the pre-stimulus base level and all components of the HR response following the stimulus. This can be accomplished by recording HR on a beat-by-beat basis or a second-by-second basis. When the beat-by-beat method is used HR is estimated by measuring the time elapsing between each beat. The reciprocal of length of the inter-beat-interval (IBI) gives the HR in beats per minute (bpm). The second-by-second means of measuring HR simply consists of computing the mean HR for each second (sec) preceding and following the stimulus. Other procedures such as computing a mean HR for a pre-stimulus period and a post stimulus period do not permit an examination of the many possible fluctuations of HR above and below the pre-stimulus level following the stimulus.

For the purpose of this study deceleration of HR was defined as any decrease of HR below the level of the preceding IBI or SEC. A deceleratory trend or phase was defined as any deceleration of HR which persisted for two or more successive IBIs or SECs. An acceleratory trend or phase was defined as any acceleration of HR which persisted for two or more successive IBIs or SECs. Trends of acceleration or deceleration which rose above the prestimulus level or fell below the pre-stimulus level, respectively, were so designated.

Heart rate response curves of various types have been observed in the literature. Some studies have reported a diphasic response

of initial acceleration above the pre-stimulus level followed by a phase of deceleration below the pre-stimulus level (Davis, Buchwald, and Frankmann, 1955; Lang and Hnatiow, 1962; Uno and Grings, 1965; Smith and Strawbridge, 1968). (See Fig. 1)

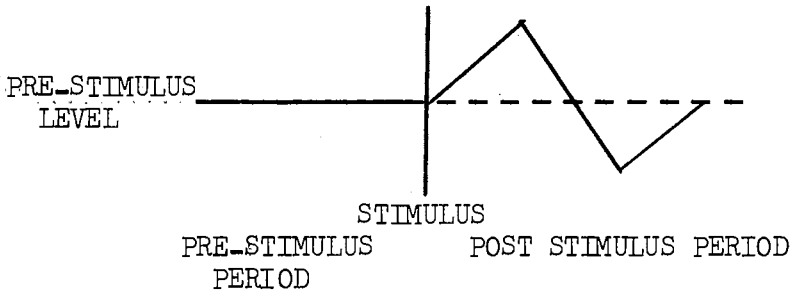

Figure 1. Approximate form of the HR response curve for studies reporting initial acceleration of HR followed by secondary deceleration.

Other studies have observed a multiphasic response of initial deceleration below the pre-stimulus level followed by a longer latency acceleration above the pre-stimulus level, and finally, a deceleratory phase below the pre-stimulus level (Meyers and Gullickson, 1967; Meyers, 1969; Raskin, Kotes and Bever, 1969; Smith and Strawbridge, 1969). (See Fig. 2)

Monophasic decelerative HR responses below the pre-stimulus level have been reported by Chase and Graham (1967), Kanfer (1958), Wilson (1964), Zeaman, Deane, and Wenger (1954), and Davis and Buchwald (1957). (See Fig. 3)

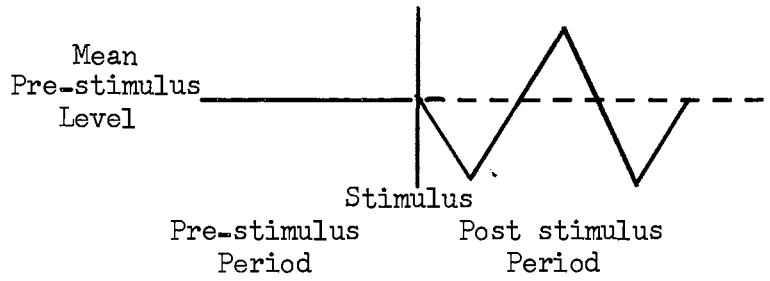

Figure 2. Approximate form of the HR response curve in studies reporting initial deceleration below the pre-stimulus level, secondary acceleration above the pre-stimulus level, and deceleration below the pre-stimulus level.

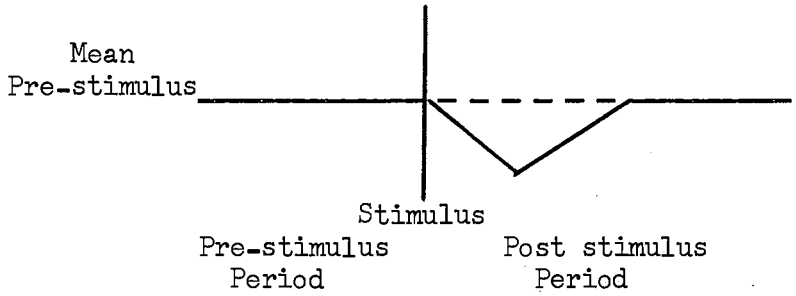

Figure 3. Approximate form of the HR response curve in studies reporting deceleration below the pre-stimulus level.

Acceleration of HR above the pre-stimulus level has been reported by Dykman, Reese, Galbrecht, and Thomasson (1959), and Germana and Klein (1968). (See Fig. 4)

Numerous attempts have been made to explain these inconsistent results. At present, most researchers have concluded that deceleration best meets the criteria of an OR (occurs to stimuli of moderate intensity and habituates). (Graham and Clifton, 1966; Lacey and Lacey, 1958; Smith and Strawbridge, 1968, 1969; Raskin, Kotes and

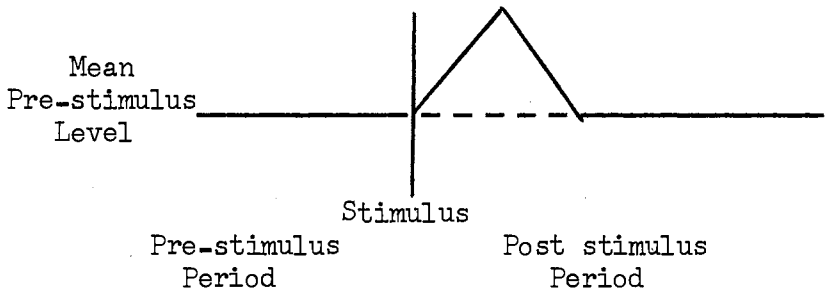

Figure 4. Approximate form of the HR response curve in studies reporting acceleration above the pre-stimulus level.

Bever, 1969). The acceleratory phase of the response curve that has been frequently observed has been explained as a phasic component of the OR (Graham and Clifton, 1966); a specific adaptive reflex (Soltysik, Jaworska, Kowalski, and Radom, 1961; Meyers and Gullickson, 1967), a startle reflex (Graham and Clifton, 1966; Smith and Strawbridge, 1968), a defensive reflex (DR) (Graham and Clifton, 1966); or as the result of respiratory artifacts (Smith and Strawbridge, 1969). Research conducted since Graham and Clifton's review tends to eliminate all of these except respiration from serious consideration. The reflexive effect of respiration upon HR (sinus arrythmia) has long been known (de Cyon and Ludwig, 1847; see Scher, 1965b), but few studies have examined the possible effect of this reflexive mechanism upon the HR response curve. Therefore, the purpose of this study was to examine the role of respiration in producing the accelerative phase of the HR response to non-signal, novel stimuli.

Review of the Literature

Psychological and Physiological Characteristics of the OR

The OR is one of three responses that may occur to simple stimuli. The OR, the adaptive response (AR), and the defensive reflex (DR) represent three distinct and important responses to simple stimuli. The OR acts to increase the effects of stimulation by eliciting generalized approaching behaviors and cortical activation (Sokolov, 1960). The AR serves a localized homeostatic function which is specific to the sense organ stimulated. For example, when the eye is exposed to a change in the level of stimulation (light or darkness) it responds by constricting or contracting. The AR persists with repetition of the stimulus, but the OR habituates as the stimulus is repeated. The DR evokes generalized "fight or flight" patterns of aggression, retreat, or freezing. When the stimulus is repeated the DR habituates very slowly if at all.

Three stimulus parameters determine whether an OR or a DR occurs to a stimulus. These are as follows: (1) the intensity of the stimulus; (2) the number of repetitions of the stimulus; and (3) the signal value of the stimulus (i.e. its capacity to serve as a conditioned stimulus). Intense stimuli produce immediate ORs and DRs on later presentations of the stimulus. If a stimulus acquires signal value, it becomes more likely to elicit ORs at both higher and lower intensities.

Sokolov (1960, 1963a) has proposed a theory of the operation of the OR. Sokolov postulates the existence of a "neuronal model" consisting of an organization of neural cells of the cortex which preserve

information about the intensity, quality, duration, and patterning of stimuli. If the parameters of a given stimulus do not parallel existing neuronal models, an OR is initiated by excitatory processes. When the parameters of a stimulus parallel those represented in an existing neuronal model, negative feedback to the excitatory system is initiated which results in an inhibition of the OR. With repeated presentations of the stimulus, the model becomes more completely developed and the magnitude of the OR gradually decreases until it disappears completely.

Although the basic propositions of Sokolov's model have been confirmed (Zimny and Schwabe, 1966; Zimny and Kienstra, 1967), research has failed to adequately define the physiological components of the OR. Lynn (1966) and Berlyne, (1960) have described the physiological changes of the OR as follows: (1) an increase in sense organ sensitivity; (2) activation of skeletal muscles that direct sense organs; (3) muscular activity (rise in general muscle tonus and electromyographic activity); (4) vegetative changes (GSR, respiration changes, variable HR, and vasodilation in the head and vasoconstriction in peripheral areas); and (5) changes in the central nervous system (e.g. disruption of alpha or slower waves). Thus, various physiological changes have been observed in association with the OR. A closer examination of the literature, however, reveals that some of these changes are not clearly defined. In particular, research on the HR response to simple stimuli of moderate intensity has failed to arrive at a precise determination of the HR component of the OR.

In the normal functioning of the heart the sino-artrial (S-A) node is responsible for the HR. Excitatory impulses generated in the S-A node initiate a wave of depolarization which results in the heart beat.

(Scher, 1965a). Changes in this pattern of excitation and, therefore, changes in HR can be produced in numerous ways. Heart activity is regulated by the cardio-vascular regulatory center located in the medulla. (Rushmer, 1965; Brener, 1967) This center is composed of three sub-centers which mediate specific changes in cardio-vascular activity. The cardiac acceleratory center acts through the sympathetic cardiac nerves to produce an increase in HR. Decreases in HR are produced by the cardio-inhibitory center through the action of the parasympathetic vagus nerve. The sympathetic vascoconstrictor center produces vaso-constriction of the peripheral arterial tree and a rise in arterial pressure.

Information on blood composition is conveyed to the medulla cardiovascular regulatory center by means of the chemoreceptors of the aortic arch and the carotid sinus. Thus, increases in CO_2 or decreases in O_2 in the blood lead to stimulation of the cardio-acceleratory center and, therefore, an increase in HR. Information on blood pressure is transmitted to the cardio-vascular regulatory center by the baro-receptors of the blood vessels, aortic arch, and carotid sinus. An increase in pressure activates the cardio-inhibitory center through the baroceptors which produces a decrease in both HR and blood pressure.

Although the cardio-regulatory center of the medulla is sufficient for homeostatic adjustments of heart activity, other areas of the brain are responsible for further changes in cardiac activity. The hypothalamus and the cerebral cortex for example, may be responsible for changes in cardiac activity by influencing the cardio-vascular regulatory center. The changes of HR associated with the OR probably originate in the cortex or other centers of the brain. Impulses from

these centers act on the cardiac-regulatory center of the medulla, thereby influencing the activity of the heart. Thus, deceleration as a component of the OR is produced by the cardio-inhibitory center through the action of the parasympathetic vagus nerve. The accelera-tory component of the OR results from stimulation of the sympathetic cardiac nerves by the cardio-acceleratory center.

Heart Rate Component of the OR

Although Sokolov (1960, 1963a, 1963b) is the leading theorist and researcher on the OR, he has not specified the HR component of the OR. A study cited by Sokolov (Soltysik et al., 1961) reported an increase in the HR of dogs in response to the onset of a sound and a decrease in HR to the offset of the sound. Sokolov (1963b) interpreted the deceleration of HR that occurred at the offset of the sound as a specific accoustico-cardiac reflex dependent upon the intensity of the sound. Responses such as this which are confined to a single sense organ cannot be a portion of the OR due to the fact that the OR represents a generalized response rather than a specific adaptive response. The initial acceleration was interpreted to be the result of a summation of the acoustico-cardiac reflex and the OR. Thus, Sokolov seems to designate acceleration as the HR component of the OR.

Lacey and Lacey (1958), however, have postulated that deceleration characterized the HR component of the OR. This conclusion is based upon the observation that increases in HR and blood pressure are associated with inhibition of cortical activity (Bonvallett, Dell, and Hiebel, 1954; Nakao, Ballim, and Gellhorn, 1956). Since the OR acts to facilitate the reception and evaluation of stimuli it should be associ-

ated with cortical activation. Lacey and Lacey, therefore, propose that deceleration accompanies stimuli or tasks which require receptivity to the external environment and acceleration occurs to stimulus patterns or tasks which necessitate or evoke rejection of the external environment.

Lacey, Kagan, Lacey, and Moss (1962) tested this hypothesis by exposing subjects (Ss) to stimulus situations designed to elicit rejection or acceptance of the external environment. It was hypothesized that the tasks which required sustained attention to the incoming signals would elicit HR deceleration, but tasks which required selective rejection of the external environment would result in HR acceleration. Two groups of Ss (N=94 and N=30) were given both of these types of tasks and the mean of the twelve fastest IBIs were determined for: (1) a one minute period preceding the presentation of an alert concerning the nature of the coming task (Base Level); (2) a one minute period following the alerting announcement (Alert); and (3) the one to two minute period during the stimulus situation (Stress).

The tasks selected which were defined as requiring rejection of the external environment involved internal manipulation of symbols and retrieval of stored information. These were: mental arithmetic (simple addition and multiplication problems), reverse spelling (identification of words verbally spelled in reverse order), and make-up sentences (Ss asked to make up 5 word sentences beginning each word with a letter announced by E). As predicted, all of these tasks resulted in acceleration of HR.

Tasks that required attention to the incoming stimulus were the following: (1) photostimulation (Ss were asked to note the varying

colors and patterns of light in the stimulus) (Flash); (2) a tape recorded recitation by a dying man with which Ss were instructed to empathize (Drama). The pattern of HR response for these tasks consisted of acceleration from Base to Alert and deceleration for the Stress period, as was predicted.

Lacey, et al. (1962) interpreted these results to mean that tasks which demand that incoming signals be noted throughout the stimulation period result in deceleration. The tasks that demanded concentration and mental work resulted in acceleration. Lacey's research, however, has involved complex stimulus situations which are not comparable to the simple non-signal stimuli used in OR research. Graham and Clifton (1966) note that such complex situations may not involve simple acceptance-rejection alone. Furthermore, Lacey's HR measure does not allow for an examination of the exact form of the HR response being elicited.

The results of the study by Lacey, et al. (1962) provides a demonstration of a decelerative HR response to moderate intensity stimulation; however, other studies which have used more intensive analyses and more relevant stimuli have failed to obtain unequivocal results. Graham and Clifton (1966) reviewed numerous studies of the HR component of the OR and found little agreement among them. Few of the studies reviewed reported a purely decelerative response to moderate intensity stimulation. Despite the confusing and contradictory nature of the literature, Graham and Clifton were able to conclude that deceleration represents the HR component of the OR. This conclusion was based on two observations: (1) the initial HR response to stimulation was virtually always deceleration when the method of data analysis permitted such an observation; and (2) this initial decelera-

tion usually habituated with repetition of the stimulus; whereas, the accelerative component tended to resist habituation. For instance, Davis, Buchwald, and Frankmann (1957) reported that by the tenth trial the decelerative component was only 20% of its initial magnitude. Also, Lang and Hnatiow (1962) reported a persistent accelerative component and a habituating deceleration component. The accelerative component best met the criterion of a DR (occurred to strong stimuli, resisted habituation, and frequently increased in amplitude with repetition of the stimulus).

Graham and Clifton (1966) discuss several alternative explanations for the frequently observed accelerative component of the HR response. They note that the accelerative component does not appear to be an essential component of the OR since it fails to occur in some appropriate stimulus situations and often resists habituation. A second explanation of the acceleratory phase is that it represents a partially inhibited DR. This is consistent with Lacey's interpretation of HR acceleration, and studies which have reported acceleration have often used stimuli in the prepain zone of intensity (Davis, Buchwald, and Frankmann, 1955; Lang and Hnatiow, 1962), whereas, studies which have reported a predominance of deceleration have frequently used stimuli within the mild or moderate range of intensity (Kanfer, 1958; Wilson, 1964; Zeaman, Deane and Wenger, 1954; Chase and Graham, 1967).

A further possibility mentioned by Graham and Clifton is that the accelerative phase represents a specific adaptive response to acoustic stimuli. They note that most of the studies reviewed used auditory stimuli and those using nonauditory stimuli have not observed acceleration as frequently (Davis, et al., 1957; Geer, 1964). Finally, Graham

and Clifton propose that the accelerative phase could be the result of a startle response. Fleshler (1965) found that the effective acoustic stimulus parameter for eliciting startle in the rat was the peak intensity reached during the first 12 milliseconds of stimulation. Thus, it seems possible that startle could be produced by a relatively low intensity tone if it reached its peak intensity quickly.

A further possibility that Graham and Clifton fail to consider is that the acceleratory phase is the result of respiratory activity. The reflexive effect of respiration on HR (sinus arrythmia) can result in both deceleration and acceleration. Therefore, the acceleratory phase could be the result of a reflexive, compensatory or homeostatic response to the deceleration produced by the OR. It is surprising that more attention has not been given to the role of respiration in producing the acceleratory component.

Recent Research

Several equally plausable explanations for the acceleratory phase exist. An examination of research published since Graham and Clifton's review may provide more insight into which of these explanations is correct. Uno and Grings (1965) recorded HR responses along with several other physiological variables from twelve Ss. The stimuli were two sec bursts of white noise presented at five intensity levels (60, 70, 80, 90, and 100 db). Each of five interstimulus intervals (30, 40, 45, 50, and 60 sec) appeared once in each of five trial blocks. According to Uno and Grings the HR response to the 70, 80, and 90 db noise was characterized by a predominance of acceleration above the pre-stimulus level. The 60 and 100 db response curves, on the other hand, exhibited

a predominance of deceleration below the pre-stimulus level.

Repetition of the 100 db stimulus changed the HR response from deceleration to acceleration above the pre-stimulus level, although deceleration continued to occur for the later post-stimulus IBIs. All other intensities continued to result in acceleration with repetition. The fact that the observed deceleratory trend tended to habituate while the acceleratory trend persisted indicates that the deceleratory trend best meets the primary criterion of an OR (systematic habituation). The failure of deceleration to occur for the other intensities may have been the result of an artifact such as respiration superimposed upon the HR component of the OR. Also, the failure for the observed acceleratory trends to exhibit ordering of amplitude according to intensity of the stimulus indicates that the acceleration may not be due to a startle reflex. Since acceleration occurred to stimuli which varied from moderate to painful in intensity, the acceleratory phase does not seem to be attributable to a DR. Similarly, if a startle reflex was responsible for the acceleratory phase, the effective stimulus parameter which elicited the startle would have to be some variable other than the intensity of the stimulus. The rise time of the stimulus or the time required for the stimulus to attain its peak intensity might constitute such a parameter.

Chase and Graham (1967) presented 10 continuous, 18 sec, 71 db tones to 10 Ss. Half of the Ss received a series of identical tones first and then received a series of "different" tones. The "different" tones were equally spaced from 300 to 2000 Hz on a logarithmic scale and ranged from 60 to 87 db. The other half of the Ss received the tones in reverse order. Deceleration occurred to both the onset and

offset of the tones. The decelerative component habituated with repeated presentations until the onset and offset responses were not apparent during the last three trials. The rate of habituation was not effected by the difference in the two series of tones. Each series habituated at approximately the same rate. Chase and Graham concluded that variable nature of the tones did not introduce sufficient novelty.

Chase and Graham's failure to observe an acceleratory trend is the most interesting aspect of this study. An examination of the response curves for the data of trial blocks #1 and #3, however, may reveal the reason for this failure. Acceleration appears to have occurred for both of these trial blocks, but it was delayed for the first trial block until 2.5 sec. after the onset of the stimulus and only $\frac{1}{2}$ sec. for the third trial block. Thus, if this trend is characteristic of the rest of the data, an overall response curve might appear to maintain a constant level throughout stimulation when in fact acceleration occurs on each trial block in different portions of the response curve. This apparent acceleration did not habituate with repetition as did the deceleration phase.

Meyers and Gullickson (1967) presented 40 stimuli of $\frac{1}{2}$ sec. duration (71 db) at a rate of 1 every 10 sec. to 48 Ss. Each stimulus was composed of 2 successive tones of different frequencies. The first 20 stimuli comprised a familiarization phase, and the second 20 stimuli made up the test phase of the experiment. During the familiarization phase, half of the Ss heard them in a low-high frequency sequence. The high frequency tone was 700 Hz, and the low frequency tone was 300 Hz. The only change in the test phase was to reverse the frequency sequence heard by each S.

The response curve for both the first and second 20 trials was diphasic in nature. Slight initial deceleration was followed by pronounced acceleration. The only discernable difference between the two sets of trials was an increase in initial deceleration for the second set of trials. The HR deceleration observed on trial #1 had completely disappeared by trial #2, indicating habituation effects which are characteristic of the OR. The deceleration was replaced by acceleration which Meyers and Gullickson suggest might be an adaptation response, since it was judged unlikely that a DR would occur to a stimulus of moderate intensity.

Smith and Strawbridge (1968) presented 16 stimuli at 40 db and 16 stimuli at 85 db (4000 cps) at durations of 1 sec. and 15 sec. to 48 male Ss. The interstimulus interval was constant at 30 sec. The intensity of the stimulus did not prove a significant factor, but duration of the stimuli seemed to be responsible for a marked change in the form of the response after 5-7 post-stimulus IBIs. At this point, the response to the 15 sec. tone fell below the pre-stimulus level and remained there for a period of 10 to 12 IBIs. The HR response for the 1 sec. tone remained largely above pre-stimulus levels during this period. With repetition, the response curve of the 1 sec. tone did not change appreciably, but the response curve to the 15 sec. tone changed to acceleration similar to that of the 1 sec. tone.

Smith and Strawbridge concluded that deceleration is the HR component of the OR based on the rapid habituation of the deceleratory phase. Since the acceleratory phase persisted, they concluded that it could not be a part of the OR. Likewise, it could not be a part of the DR because it occurred to stimuli of both high and low intensity.

Smith and Strawbridge attributed the acceleration to a startle response caused by the rise time of the tones, or to a specific response to acoustic stimuli.

Germana and Klein (1968) presented five tones (1000 Hz of one sec. duration) at intensities of 50, 70 and 90 db to 6 Ss. The inter-stimulus interval was constant at 10 sec. Measures of HR indicated that the overall response was acceleration above the pre-stimulus level with multiphasic responses superimposed upon it. Trials #1 and #3 consisted of initial acceleration above the pre-stimulus level followed by deceleration (but not below the pre-stimulus level), and another late accelerative response above the pre-stimulus level. The accelerative phase habituated with repetition, but the decelerative phase did not. Furthermore, the accelerative phase was greater for the higher intensity tones (70 and 90 db). Germana and Klein concluded that the criterion of an OR was best met by the accelerative phase (systematic habituation and ordering of response magnitude according to intensity).

There are several weak points in this study which may limit the validity of these findings, however. The small number of Ss (N=6) may limit the validity of the findings and, possibly, expose the study to the individual differences which have been observed in OR functioning (Maltzman and Raskin, 1965; Lynn, 1963). Secondly, there was little apparent difference in the response to the 70 db tones and the 90 db tones. According to Sokolov (1963), a 90 db tone lies in the prepain zone of intensity, while a 70 db lies within the moderate range of intensity. Therefore, the 90 db tone should elicit a DR while the 70 db tone should elicit an OR. The fact that there was little discernable difference between the obtained responses to these tones makes

the validity of these findings questionable. Thirdly, Germana and Klein seemed to ignore the fact that the 50 db tone did produce a substantial deceleratory phase (below the pre-stimulus level) which habituated. A slight deceleration reappeared on trial #5, but the spontaneous recovery of a habituated OR is not impossible. The important point is that both the acceleratory phase and deceleratory phase exhibited habituation to some extent. This indicates that although it cannot be determined which represents an OR, neither can conclusively be classified as a DR or a startle response. Both the DR and startle response do not permit the rapid habituation observed in the response curves. Finally, the short inter-stimulus interval of 10 sec. hardly allows for a full development of the HR response to a stimulus before the next one is presented. Such a short inter-stimulus interval may have caused a compounding of the HR responses. Germana and Klein were apparently aware of some of these difficulties for they conclude by pointing out that precise HR measures of the OR are dependent upon the elimination of artifactual components such as those resulting from respiration.

Smith and Strawbridge (1969) conducted a study in which both HR and respiratory responses to nonsignal stimuli were examined. Using 18 male Ss, they presented 10 (54 db, 700 Hz), 3/4 sec. tones and similar visual stimuli (8.0 ml.). At inter-stimulus intervals of (in order of presentation) 50, 60, 45, 55, 40, 45, 40, 55, and 60 sec. The tones elicited a HR response of predominately acceleration above the pre-stimulus level with repetition of the stimulus. Both of the response curves (auditory and visual) exhibited an initial response (beats #0-1) of deceleration which rapidly changed to acceleration.

The initial deceleration habituated for both the light and the tone with repetition of the stimuli. Thus, the two stimuli although of two diffferent modalities, elicited roughly identical response curves.

Significant changes in respiratory rate and amplitude followed the auditory and visual stimuli. These changes did not habituate with repetition of the stimuli. The mean respiratory rate for both stimuli increased over pre-stimulus levels, although this increase was significant only for the visual stimulus. Amplitude of respiration, on the other hand, was significantly greater for the tone, but not for the light flash. The magnitude of these responses did not habituate over the 10 presentations of the stimuli. Attempts to correlate pre- and post-stimulus respiration measures with the corresponding HR measures at points of maximum HR acceleration and HR deceleration (post-stimulus IBIs #1, 3, 4 and 10), yielded low and insignificant correlations, generally. In the case of the light flash, however, the amount of HR acceleration was directly related to the amount of respiration amplitude increase for several HR IBIs (HR IBI #3, rho= .46, $p < .05$; HR IBI #4, rho=.37, $p < .10$). Thus, the observed HR acceleration phase appears to be accompanied by persistent respiratory changes. The fact that the deceleratory phase habituated in the presence of persistent respiratory changes was noted by Smith and Strawbridge as evidence that the deceleration was not influenced by respiration. Smith and Strawbridge conclude that the HR response to simple auditory and visual stimuli consists of a habituating deceleratory phase of short latency and long duration, with a shorter duration and longer latency acceleratory phase superimposed upon it. Also, the

acceleratory phase is resistent to habituation and related to corresponding respiratory changes.

Raskin, Kotes, and Bever (1969) attempted to differentiate between ORs and DRs using numerous physiological indices (forehead-skin pulse amplitude, forehead-skin blood content and HR). Thirty $\frac{1}{2}$ sec. bursts of white noise, presented at inter-stimulus intervals of 30, 45, and 60 sec., made up the stimuli. The stimuli were presented at two intensities (80 and 120 db) to 30 male Ss. The 80 db noise resulted in an overall response curve that was characterized by a slight initial deceleration, a secondary acceleration, and a later deceleration. The 120 db noise produced a pronounced initial acceleration with a slight later deceleratory trend (about 8-10 IBIs following the stimulus). Repetition of the stimuli resulted in greater habituation of the acceleratory component for the 120 db noise than for the 80 db noise. Raskin, et al. concluded that the OR may be discriminated from the DR on the basis of the short latency deceleration for the 80 db noise and the predominance of acceleration for the 120 db noise. However, the 80 db noise may not have elicited an OR at all. A stimulus of 80 db lies in the pre-pain zone of intensity and therefore, the observed HR response very likely reflects DRs as well as ORs. This fact makes it difficult to conclude that ORs may be discriminated by the presence of the initial deceleratory trend. Had this study used a stimulus clearly falling within the moderate range of intensity such a conclusion might have been warranted.

Meyers (1969) examined the HR response of forty adolescents (11-13 years old) to a moderate (70 db) or a loud (95 db) stimulus pattern. The stimulus pattern was identical to that employed by Meyers

and Gullickson (1967) which was described earlier. Meyers reported that the only significant deviation from the pre-stimulus HR level for the first presentation of the 70 db stimulus pattern was an initial decelleratory trend. The 95 db stimulus pattern exhibited a significant initial deceleration below the pre-stimulus level and a later decelleratory trend below the pre-stimulus level (5-10 sec following the stimulus). Thus, both intensities elicited an initial deceleratory trend below pre-stimulus levels, but the 95 db stimulus pattern also exhibited a secondary deceleratory trend.

Repetition of the 70 db stimulus pattern changed the HR response from deceleration to acceleration above the pre-stimulus level. However, this acceleration habituated after ten trials. Repeated presentations of the 95 db stimulus pattern resulted in deceleration below the pre-stimulus level which continued to occur through the final trial block.

The results of recent research do not appear to support the explanations for the acceleratory phase proposed by Graham and Clifton (1966). The fact that the acceleratory trend occurred without regard to the intensity of the stimulus (Uno and Grings, 1965; Smith and Strawbridge, 1968, 1969; Germana and Klein, 1969; Raskin, Kotes, and Bever, 1969; and Meyers, 1969) indicates that this trend can neither be an essential component of the OR or a partially inhibited DR. Presumably, if either of these alternatives were correct, the accelleratory trend would exhibit some tendency to occur to stimuli of a particular range of intensities. This is due to the fact that the OR is elicited by moderate intensity stimuli and the DR occurs to intense stimuli. For much the same reasons the acceleratory trend does not

appear to be the result of a startle reflex unless the rise time of stimulus is the effective stimulus parameter eliciting startle. The proposal that acceleration represents a specific adaptive response to acoustic stimuli was eliminated when Smith and Strawbridge (1969) failed to observe a difference in the HR response curves for auditory and visual stimuli.

The only proposal that has not been discredited is the respiratory explanation for the acceleratory trend. The reflexive effect of respiration upon HR (sinus arrhythmia), has long been known (de Cyon and Ludwig, 1847, see Scher, 1965b). This phenomenon consists of reflexive acceleration of HR in conjunction with inspiration and reflexive deceleration of HR in conjunction with expiration. Although there appears to be some controversy over the exact physiological mechanisms involved in sinus arrhythmia (Manzotti, 1958; Scher, 1965; Clynes, 1960), the effect of respiration upon HR has been clearly documented.

Westcott and Huttenlocher (1961) conducted a study on the effects of various levels of respiratory activity upon HR. Ss were trained to respirate at various rates (6, 10, and 20 cycles/min) and depths (shallow, moderate, and deep). Westcott and Huttenlocher examined the amount of variability of HR introduced by these procedures. Deep breathing at 6 cycles/min produced HR changes (initial acceleration above the pre-test levels followed by deceleration below the pre-test levels) on the order of 30 bpm; at 10 cycles/min produced HR changes of 14 bpm; and at 20 cycles/min. resulted in a rapid steady HR instead of the orderly fluctuating HR observed for the other rates.

Medium respiratory depth resulted in HR changes of 15, 15, and 8 bpm for the three rates respectively. Shallow breathing resulted in dependable HR changes at 6 cycles/min and 10 cycles/min only (8 and 5 bpm respectively). The HR change for rapid shallow respiration (20 cycles/min) was small and irregular. Isolated gasps of respiratory activity resulted in consistent HR changes. A single sharp inspiration, for example, produced an acceleration above the pre-test level followed by an equally sharp deceleration below the pre-test level before returning to the base level.

Although OR studies have largely neglected to give adequate consideration to the role of respiration in producing acceleration, numerous HR conditioning studies have attempted to study the conditioned cardiac response independent of respiratory activity (Wood and Obrist, 1964; Riege and Peacock, 1967; Zeaman and Smith, 1965; Westcott and Huttenlocher, 1961). Because these studies dealth with complex conditioning contingencies and frequently employed intense shock as the CS, the results are not applicable to OR research. Therefore, only the methods used to eliminate or minimize respiratory artifacts will be reviewed here.

Three principle procedures have been employed in these studies to control respiratory effects. Sustained inhalation and exhalation have been successfully used in several studies (Riege and Peacock, 1967; Smith, 1965; Zeaman and Smith, 1965). These procedures require that Ss extend the inspiration or expiration portion of their respiratory cycle when a signal is presented. Thus, when a stimulus is due to be presented, some signal must be given to the S for him to initiate the appropriate respiratory pattern. These methods, obviously, cannot

be used for prolonged periods, and seem inappropriate for OR research due to the stimulus confounding that is likely to occur.

Several studies have used training procedures in which Ss were to maintain a pace set by a stimulus which was either adjusted to correspond to the Ss normal respiratory pattern or at a predetermined rate. In these studies, training was usually continued until each S exhibited adequate control of respiratory activity and then the pacing stimulus was removed. With this method Ss can maintain the desired pattern for prolonged periods without creating an abnormal physiological state.

Thus, pacing methods seem best suited for OR research. The use of these methods is not likely to eliminate respiratory artifacts from HR altogether, but two effects may be achieved: (1) the effects of respiration will be minimized or held constant; and (2) isolated gasps and irregularities which seem to be capable of producing profound changes in HR will be eliminated.

With the use of such a method, caution must be taken that the emphasis placed by the training procedures on bodily activity does not produce HR changes independent of the changes produced by the training procedures. It has been reported that attending to bodily processes alone results in HR deceleration (Miller and Caul, 1969). Therefore, the use of respiratory control procedures should be accompanied by the inclusion of a group of Ss who are merely instructed to attend to their normal respiratory pattern without attempting to control it.

Statement of the Problem

The review of the literature has demonstrated that studies on the HR component of the OR have yielded confusing and contradictory results.

While deceleration appears to be the HR component of the OR, an acceleratory component has been frequently observed which has not been adequately explained. Graham and Clifton (1966) proposed four alternative explanations for this acceleratory trend, but research published since their review tends to eliminate all but one of their proposals. The only remaining proposal is that the acceleratory phase represents a startle response dependent upon the rise time of the stimulus as the effective stimulus parameter for eliciting startle. While this proposal merits study and investigation, a more reasonable explanation is that the acceleratory phase is the result of artifactual respiratory activity.

This study, therefore, attempted to examine the role of respiration in producing the acceleratory component of the HR response to nonsignal auditory stimuli of moderate intensity. HR responses were observed under two different conditions of uncontrolled respiration and under one controlled respiration condition. In the Uncontrolled I condition, Ss received no instructions concerning respiratory control or any physiological function. Ss in the Uncontrolled II condition received instructions only to attend to their normal respiratory pattern and to detect any changes in it during the experimental period. In the Controlled condition, Ss received extensive training in maintaining a constant rate and amplitude of respiration prior to the presentation of the experimental stimuli (a series of 10 moderate intensity tones presented at variable inter-stimulus intervals).

It was hypothesized that: (1) control of respiration would result in a HR response curve which contained a significantly diminished acceleratory trend than for the uncontrolled conditions; (2) there

would be no significant difference between the response curves for the two uncontrolled respiration conditions.

CHAPTER II

METHOD

Subjects

Fifty-four male volunteers drawn from undergraduate psychology classes were used as Ss in the experiment. The Ss ranged in age from 18 to 23 years and were free from any cardiac or auditory abnormalities. A further requirement for participation was that the Ss not be under medication of any kind. Ss were randomly assigned to one of the three conditions of the experiment upon entering the experimental room.

Apparatus

The stimuli (tones) were presented on magnetic tape via a Sony stereo tape recorder (model 124 CS) through a set of Sony stereo headphones (model DR 3A). The tones which served as the stimuli were produced by an audio generator (model 377, Electronic Instrument Co.). The tones were $\frac{1}{2}$ sec in duration and were presented at an intensity of 60 db (1000 Hz). A stimulus of 60 db was selected because this intensity falls well within the moderate range and was considered likely to elicit ORs which were not confounded by other responses such as DRs or startle reflexes. Two Hunter timers were wired so that they would recycle sequentially. The lights signifying the end of the timing interval were covered so that only the light indicating the duration of the timing interval was visible on each timer. The timers were

placed within easy reach of the S.

Physiological measures were recorded on a six-channel Physiograph (E and M Instrument Co.). Heart rate and respiration measures were derived from the electrocardiogram (EKG). The EKG was recorded through two surface electrodes placed on either side of the S's chest at approximately the level of the fifth rib, and a third surface electrode placed over the S's heart. During the recording of the physiological measures, each S was seated in a normal sitting position in the experimental room with the temperature maintained within the limits of normal comfort.

Procedure

Upon entering the experimental room, each S was asked to remove his shirt and to be seated. The electrodes were then attached to his chest. Following the attachment of the electrodes, each S was given the instructions appropriate to his assigned condition (see Appendix A).

In the Controlled Respiration Condition each S was trained to maintain a normal rate and amplitude of respiration. The S was instructed to adjust two timers to correspond to his normal respiratory pattern. When the timers had been adjusted, the S was instructed to pace his respiration with the timers. Eventually, the timers were turned off and the S was asked to continue to maintain a constant rate and amplitude without the aid of the timers. During the period of training without the timers, E gave verbal feedback to the S on how well he was doing. The feedback consisted of reinforcement such as, "good," "fine," or "keep it up, you're doing fine," when the observed respiratory pattern reflected adequate control. The criterion of

control was that each cycle not deviate from the previous cycle by more than 20% in amplitude. As long as such a record was maintained, only positive reinforcement was given. When a record did not conform to this standard, E gave such feedback as, "try to maintain a constant rate and amplitude," or "concentrate on maintaining a constant rate and amplitude." More specific instructions were given if the standard instructions failed to induce adequate control, however, these instances were rare. If the S failed to achieve adequate control, he was dropped from the experiment. This decision was made prior to the presentation of the stimuli. (See Appendix B for the exact times allotted for each phase of the experiment.)

In the Uncontrolled Respiration Condition I, each S received no training. The S simply sat in the experimental chair for 20 minutes prior to the presentation of the stimuli to keep the time in the experiment constant across all conditions and to permit the equipment to be calibrated and stabilized. (See Appendix B).

In the Uncontrolled Respiration Condition II, each S was initially instructed to relax while the physiograph was calibrated and stabilized. Ten minutes into the experiment, however, each S was instructed to attend to his normal respiratory pattern. The S was told that he would listen to a tape later in the experiment and would be asked to report any changes in his respiratory pattern that occurred during the tape. Therefore, the S was instructed to take a few minutes prior to the presentation of the tape to become familiar with his normal respiratory pattern. The stimuli were presented at the conclusion of this ten minute period (see Appendix B).

After this initial twenty minute period, each S listened to a tape which consisted of a series of 10 tones. The first two minutes of the tape were blank, but during the remaining six minutes of the tape, the tones were presented at random intervals of 30, 40, or 50 seconds. The inter-stimulus intervals were randomized so that each interval appeared three times during each tone series. Each of the 18 Ss in each condition received one of six tapes with the inter-stimulus intervals randomly distributed on each.

Data Analysis

Heart rate measures were derived from the EKG by a cardiotachometer which recorded the time that elapsed between each heart beat. The reciprocal of the elapsed times for each IBI yielded HR expressed in beats per minute. Data was collected for five pre-stimulus IBIs and fifteen post-stimulus IBIs. Thus, the data collected from each S consisted of 20 HR values for each of the 10 stimuli. The data was grouped into trial blocks each of which included two of the ten successive stimuli making a total of five trial blocks. The experimental design was a three factor experiment with reported measures on two of the factors (Winer, 1962; Wilson, 1967). The three factors were Conditions, Trial Blocks, and Intervals (IBIs). Repeated measures were made on Trial Blocks and Intervals.

CHAPTER III

RESULTS

Statistical Design

The experimental procedure used in this study conforms to a three-factor, factorial design with repeated measures on two factors. All three factors were considered fixed. The first factor (A) was Conditions. Factor A was composed of three levels (Controlled Respiration, Uncontrolled Respiration I, and Uncontrolled Respiration II). The second factor (B) was Trial Blocks which had five levels each representing one trial block. Each Trial Block was composed of data from two successive stimuli. The third factor (C) was Intervals. Factor C was composed of twenty levels. Repeated measures were made on factors B (Trial Blocks) and C (Intervals).

This design corresponds to that recommended by Wilson (1967) for physiological research of this type and also appears in Winer (1962, pp. 319-337) and Bruning and Kintz (1968, pp. 72-83). The usual analysis of variance assumptions apply to this design plus an additional assumption of homogeneity and equality of variance-covariance matrices. To correct for his observation that HR data fail to meet this assumption, Wilson (1967) recommends that the degrees of freedom for Intervals be reduced by $\frac{1}{2}$ when F-tests are made.

Respiratory Control

The training procedure outlined in the previous chapter for the Controlled Respiration Condition proved to be adequate to elicit a constant rate and amplitude of respiration in most Ss. Only one S had to be dropped from the experiment because adequate control was not achieved.

Results

The summary table for the main analysis reported in Table I shows that the main effects for Trial Blocks ($p < .025$) and Intervals ($p < .005$) were significant. In addition, the interactions of Conditions X Intervals ($p < .025$) and Conditions X Trial Blocks ($p < .025$) were statistically significant. Figure 5 presents the mean overall HR response curve for each condition and Figures 6, 7, 8, 9, and 10 present the response curves for each trial block.

An orthogonal trend analysis was performed on the data (Appendix C) to determine the type of function that best fit the data points. The main effects for Trial Blocks were best approximated by a linear function ($p < .01$), and the Intervals main effects were approximated by a quadratic function ($p < .01$). The Conditions X Intervals interaction was approximated by both a significant linear function ($p < .025$) and a significant cubic function ($p < .01$).

As Wilson (1967) recommends, a separate analysis was performed on each condition to facilitate interpretation of the results. Tables II, III, and IV report the results of the analysis for each condition. The main effects for Intervals were significant for each condition ($p < .005$ for each condition). The main effects for Intervals for the

TABLE I

AOV: MAIN ANALYSIS

Source	df	SS	MS	F	P
Total	5399	629,650.6805	26,054.0359	1.2909	n.s.
Between Ss	53	540,721.6515			
Conditions (A)	2	26,054.0718	26,054.0359	1.2909	n.s.
Error (Between)	51	514,667.5797	10,091.5212		
Within Ss	5346	88,929.0290			
Trial Blocks (B)	4	942.3271	235.5818	2.8391	.025
Intervals (C)	19	1,586.5705	97.7142	5.6706	.005*
AB	8	1,610.5916	201.3240	2.4262	.025
AC	38	818.3782	21.5363	1.7654	.025*
BC	76	1,148.4729	15.1115	1.2384	.25 *
ABC	152	1,643.7700	10.8143		n.s.
Error 1	204	16,927.4303	82.9776		
Error 2	969	16,697.6003	17.2318		
Error 3	3876	47,283.8881	12.1991		

*Degrees of freedom reduced by 1/2 according to Wilson's (1967) recommendation.

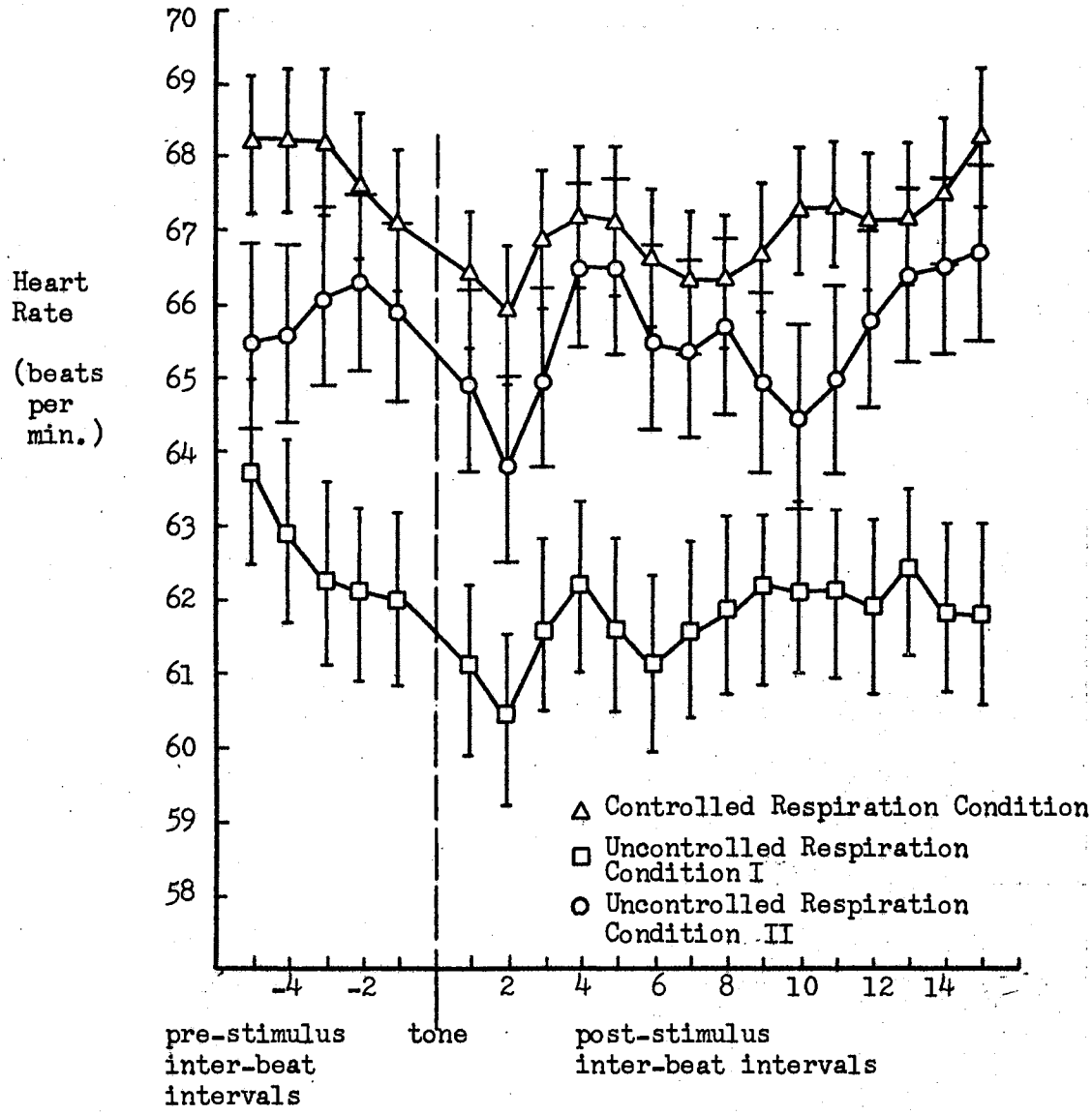

Figure 5. Mean HR response for each condition averaged over all Trial Blocks. Standard errors indicated by bands surrounding each point.

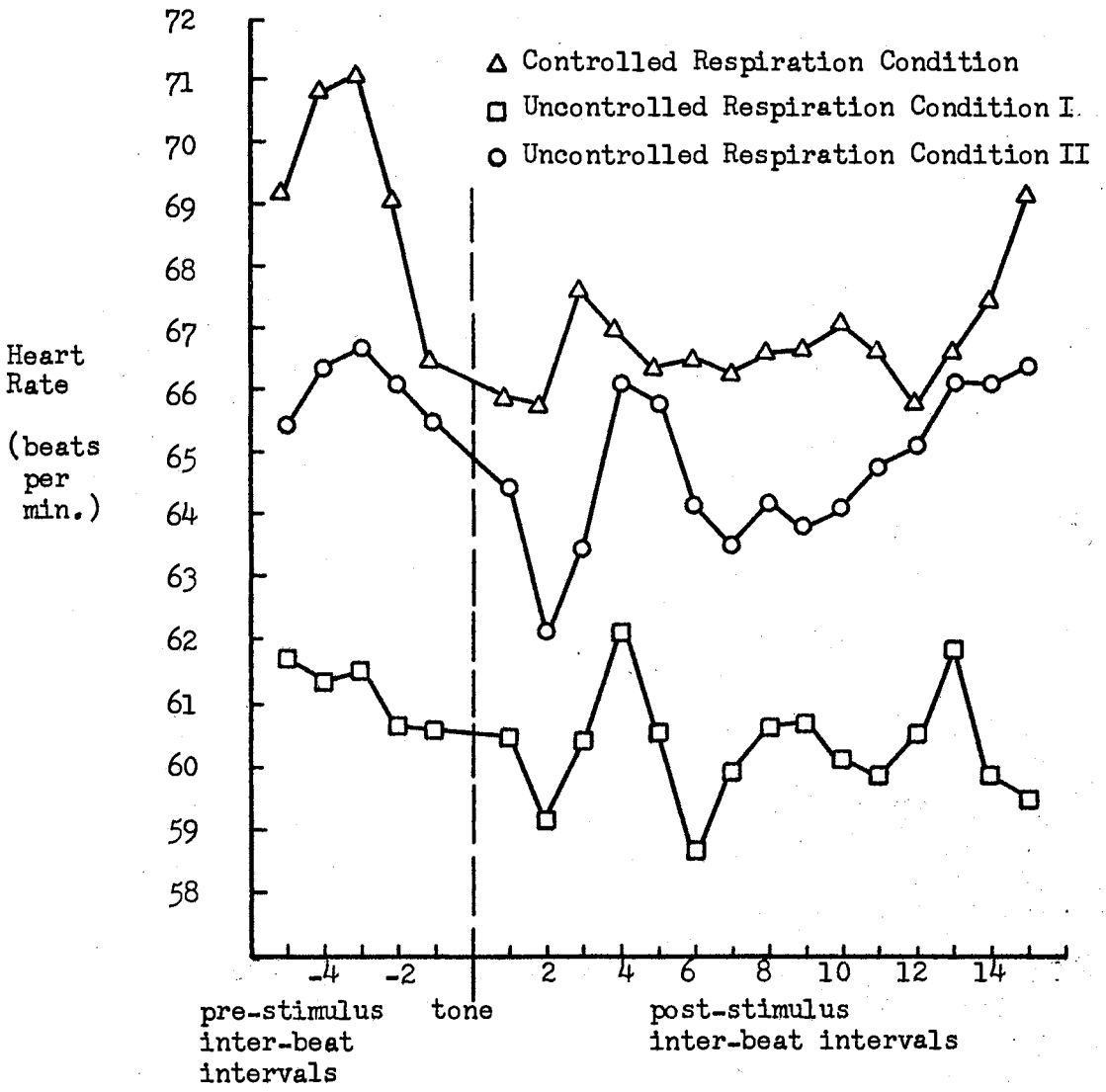

Figure 6. Mean HR response for each condition on Trial Block #1.

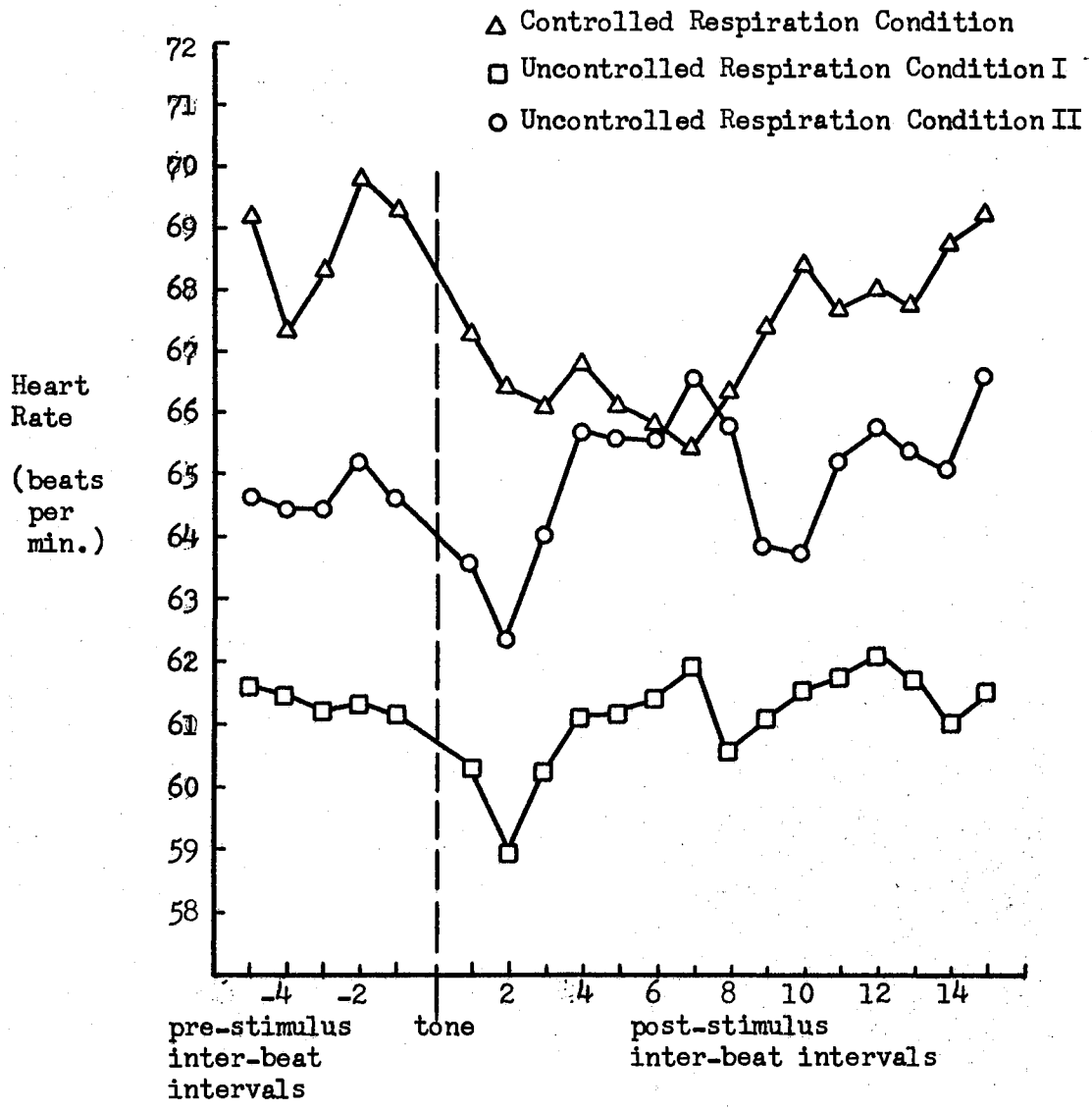

Figure 7. Mean HR response for each condition on Trial Bock #2.

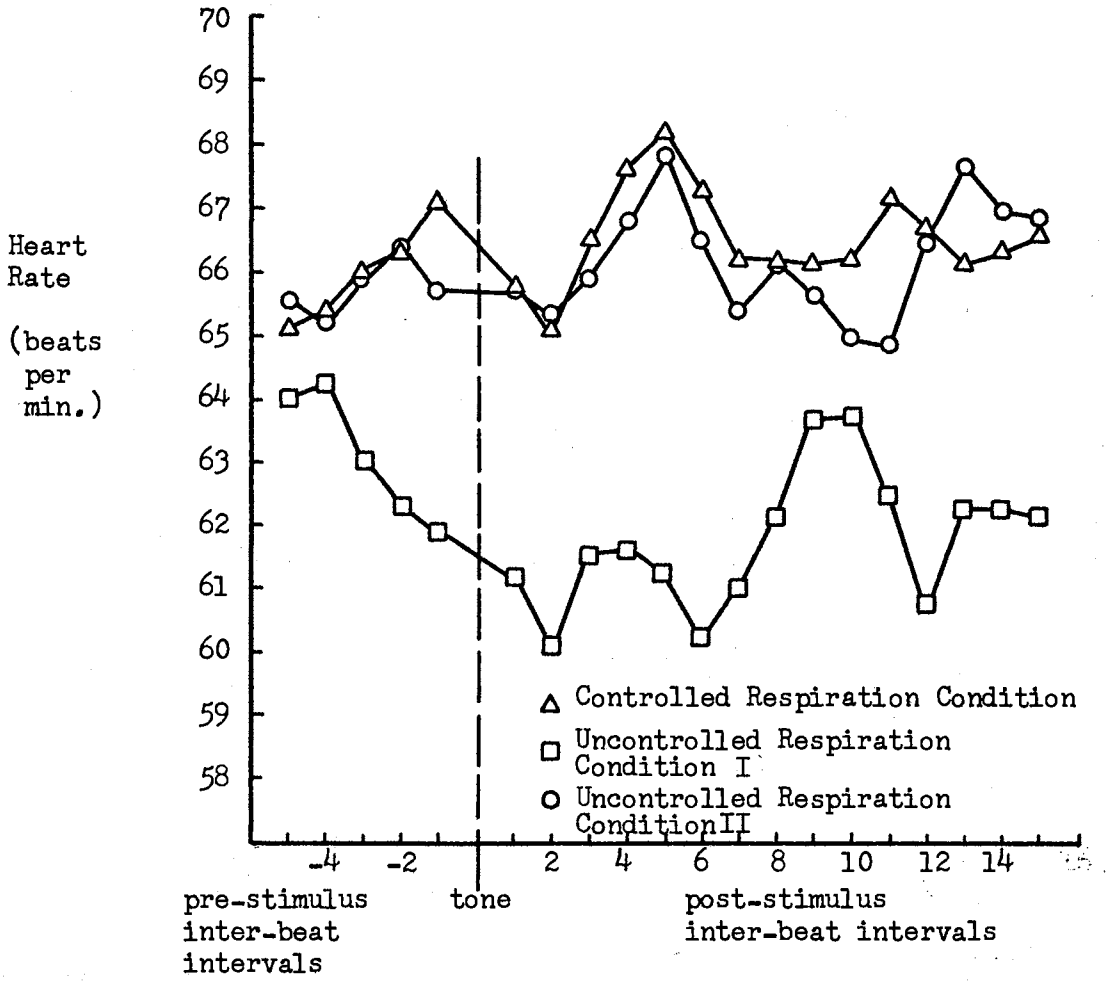

Figure 8. Mean HR response for each condition on Trial Block #3.

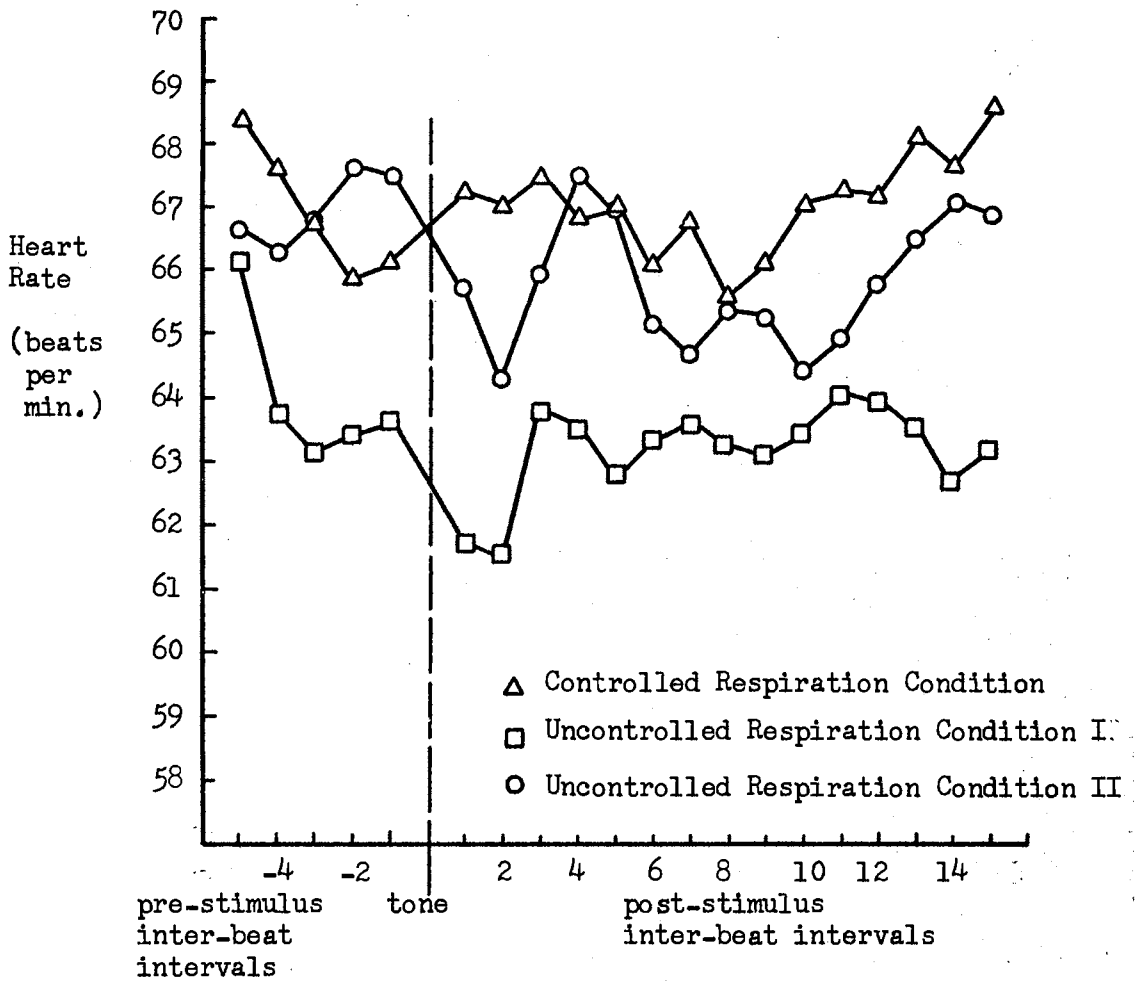

Figure 9. Mean HR response for each condition on Trial Block #4.

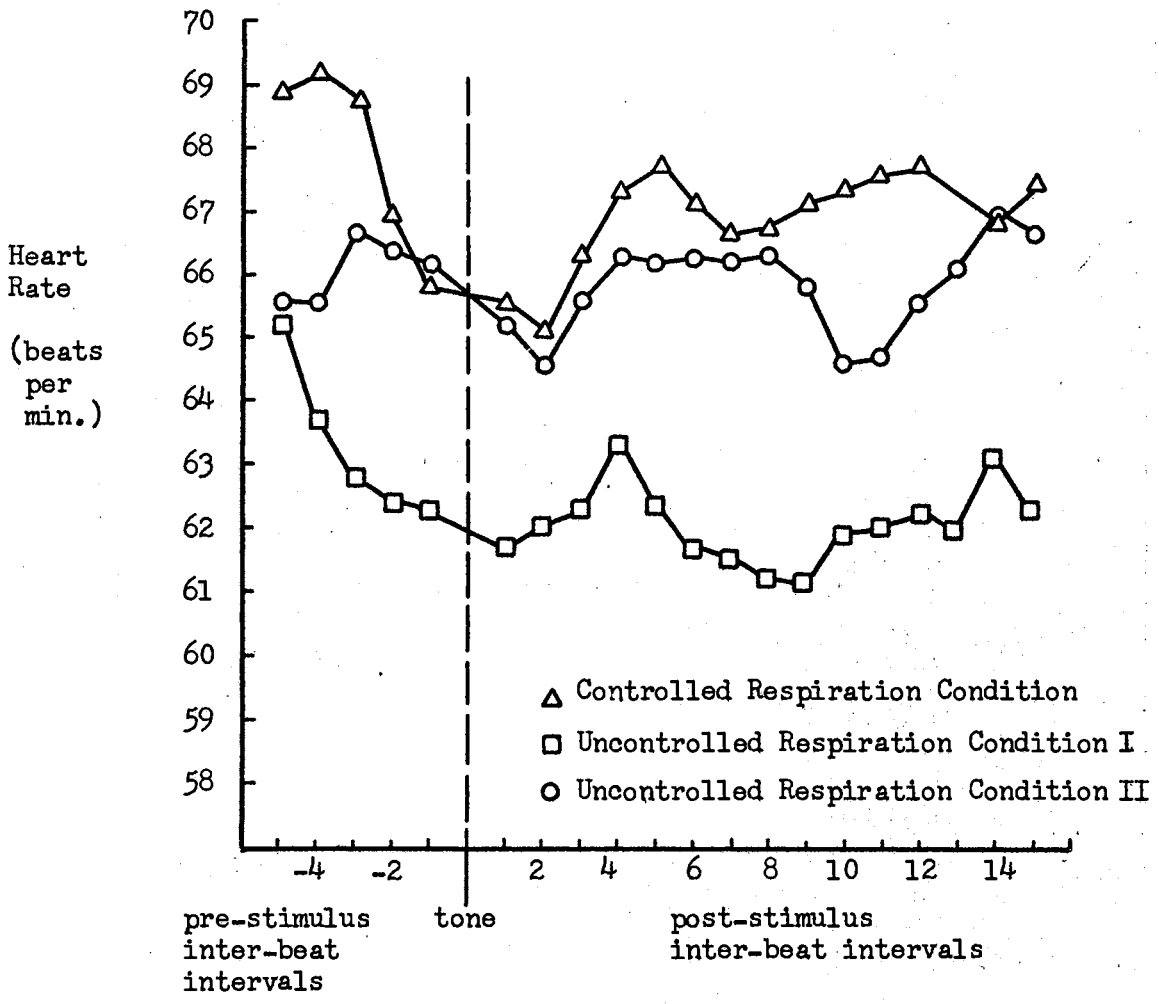

Figure 10. Mean HR response for each condition for Trial Block #5.

TABLE II

ANALYSIS OF VARIANCE OF TRIAL BLOCKS AND INTERVALS
FOR UNCONTROLLED RESPIRATION CONDITION I

Source	df	SS	MS	F	P
Total	1799	226,335.59			
Subjects	17	195,548.8740			
Within Ss	1782	30,786.7160			
Trial Blocks (A)	4	1,784.1680	446.042	4.569	.005
Linear	1	1,255.04	1,255.04	12.856	.005
Quadratic	1	277.77	277.77	2.845	.10
Cubic	1	180.99	180.99	1.854	.25
Residual	1	70.368	70.368		
Intervals (B)	19	805.7270	42.406	2.725	.005*
Linear	1	41.27	41.27	2.652	.25
Quadratic	1	298.64	298.64	19.189	.005
Cubic	1	260.55	260.55	16.742	.005
Residual	16	205.267	12.829		
A X B	76	723.1220	9.514	< 1	
Ss X A	68	6,638.5020	97.625		
Ss X B	323	5,027.0470	15.563		
Ss X A X B	1292	15,808.1500	12.235		

*Degrees of freedom reduced by 1/2 according to Wilson's (1967) recommendation.

TABLE III

ANALYSIS OF VARIANCE FOR TRIAL BLOCKS AND INTERVALS FOR UNCONTROLLED RESPIRATION CONDITION II

Source	df	SS	MS	F	P
Total	1799	235,960.29			
Subjects	17	210,443.63			
Within Ss	1782	25,516.660			
Trial Blocks (A)	4	452.655	113.164	1.422	.25
Linear	1	270.71	270.701	3.401	.10
Quadratic	1	55.18	55.18		
Cubic	1	67.89			
Residual	1	58.88			
Intervals (B)	19	1,059.053	55.740	3.594	.005*
Linear	1	34.24	34.24	2.208	.25
Quadratic	1	161.81	161.81	10.434	.005
Cubic	1	70.96	70.96	4.576	.05
Residual	16	792.04	49.503	3.192	.005
AB	76	616.998	8.118	<1	
Ss X A	68	5,413.128	79.605		
Ss X B	323	5,009.122	15.508		
Ss X A X B	1292	12,965.686	10.035		

*Degrees of freedom reduced by 1/2 according to Wilson's (1967) recommendation.

TABLE IV

ANALYSIS OF VARIANCE FOR TRIAL BLOCKS AND INTERVALS FOR CONTROLLED RESPIRATION CONDITION

Source	df	SS	MS	F	P
Total	1799	141,300.73			
Subjects	17	108,675.07			
Within Ss	1782	32,625.66			
Trial Blocks (A)	4	316.186	79.047	1.102	
Linear	1	38.19	38.19		
Audratic	1	71.52	71.52		
Cubic	1	21.87	21.87		
Residual	1	184.61	184.61	2.575	.05
Intervals (B)	19	810.247	42.645	2.068	.025*
Linear	1	30.02	30.02	1.456	
Quadratic	1	534.08	534.08	25.897	.005
Cubic	1	3.35	3.35		
Residual	16	242.80	15.175		
AB	76	1,452.126	19.107	1.334	.25*
Ss X A	68	4,875.76	71.702		
Ss X B	323	6,661.357	20.623		
Ss X A X B	1292	18,510.036	14.327		

*Degrees of freedom reduced by 1/2 according to Wilson's (1967) recommendation.

two uncontrolled conditions were approximated by significant quadratic ($p < .005$ for both conditions) and cubic ($p < .005$ for Uncontrolled Respiration I and $p < .05$ for Uncontrolled Respiration II) trends. The main effects for Intervals for the Controlled Respiration Condition was approximated by significant quadratic trend ($p < .005$). In addition, the main effects for Trial Blocks were significant for Uncontrolled Respiration Condition I ($p < .005$), but not for the other conditions.

Although the main analysis (Table I) did not show the main effects for Conditions to be statistically significant, the differences between the conditions were consistent and, seemingly, large. Therefore, the Newman-Keuls test of differences among means was performed on the means of the conditions (Appendix D). The results verified that none of the condition means differed significantly from one another. The Newman-Keuls procedure was also performed on the means of the main effects for Trial Blocks and Intervals (Appendix D). The only comparison that was significant for the Trial Blocks main effects was the difference between the means of Trial Block 1 and Trial Block 4. All other comparisons were not significant. The comparisons for the Intervals main effects revealed several significant differences. The mean of post-stimulus Interval 2 was significantly lower than the means of all other Intervals. The mean of post-stimulus Interval 1 was significantly different from the means of pre-stimulus Intervals -5, -4, -3, and -2, and post-stimulus Intervals 13, 14 and 15. In addition, the mean of pre-stimulus Interval -5 differed significantly from the means of post-stimulus Intervals 3, 6, 7, 8, 9, and 10.

CHAPTER IV

DISCUSSION

The results of this study confirm that the HR component of the OR involved deceleration below the pre-stimulus level. The HR response to the stimuli consisted of initial deceleration lasting for two post-stimulus IBIs, followed by an acceleratory trend which varied in complexity and magnitude across the conditions. The significant main effects for Intervals indicates that the changes in HR produced by the stimuli were significant. The Newman-Keuls test on the IBI means revealed that the point of maximum deceleration (post-stimulus IBI 2) was significantly below all the remaining IBI means. The acceleratory trend which followed the initial deceleratory phase reached its maximum point of acceleration at IBI 4. The point of maximum acceleration at IBI 4 represented a significant increase of HR over the point of maximum deceleration at IBI 2. The acceleratory trend was followed by a slight deceleratory trend at post-stimulus IBIs 5 through 7, but this phase did not represent a significant decrease from the point of maximum acceleration at IBI 4. Furthermore, the second deceleratory trend remained significantly above the point of maximum deceleration at IBI 2.

Thus, the mean HR response curve consisted of initial deceleration below the pre-stimulus level, followed by an acceleratory trend. To make certain that means represented in the HR response curves were not

obscuring idiosyncratic response patterns among Ss the individual response curves of Ss on Trial Block 1 were subjected to a visual examination. Trial Block 1 was selected for this examination because the most reliable changes of HR would be expected on the initial presentations of the stimuli. On later presentations of the stimuli the habituation effects of the OR would be expected to increasingly reduce the consistency of the response pattern across Ss. Of the 18 Ss in each condition, the HR response curve of initial deceleration followed by an acceleratory trend was shared by 13 Ss in both Uncontrolled Respiration Condition II, and the Controlled Respiration Condition, and by 12 in Uncontrolled Respiration Condition I. The analysis of individual response patterns, therefore, confirms that the mean HR response curves reflect reliable and consistent responses across all Ss.

The initial deceleratory phase habituated in all three conditions, although the rates differed. The deceleratory phase disappeared on Trial Block 3 for the Controlled Respiration Condition and Uncontrolled Respiration Condition II, but it did not disappear until Trial Block 5 for the Uncontrolled Respiration Condition II. The acceleratory component of the response continued to occur after the deceleratory response had habituated. Therefore, the principle criterion of an OR, systematic habituation, was met by the deceleratory phase of the observed response.

An interesting tendency for the deceleratory response to reappear on Trial Block 4 for Uncontrolled Respiration Condition II was observed. The habituation observed on Trial Block 3 may not have been genuine, or the reappearance of the deceleratory trend may have been due to some artifact. The latter of these explanations is probably

correct, since fatigue and other subject variables (e.g. boredom and restlessness) became increasingly apparent as the experiment progressed.

The significant interaction of Conditions X Intervals provided evidence for a significant difference in the response curves across conditions. The data of both uncontrolled conditions were approximated by significant quadratic and cubic trends. The Controlled Respiration Condition data, however, was approximated by a quadratic function. The absence of the significant cubic trend for the Controlled Respiration Condition was interpreted as indicating that the acceleratory component contributed to the cubic effect for the uncontrolled conditions and not for the Controlled Respiration Condition. Therefore, the failure of the Controlled Respiration Condition to exhibit a significant cubic trend was attributed to a diminished acceleratory trend in this condition.

The findings of this study have provided added insight into the nature of the HR component of the OR. The study has successfully demonstrated that the acceleratory component can be significantly decreased by controlling respiration. This finding is especially significant in view of the method used to control respiration. The training procedure used to elicit controlled respiration did not alter the respiratory cycle as does sustained inhalation or exhalation. The success in diminishing the acceleratory phase achieved by limiting respiration to regular, constant cycles indicates that the exaggerated acceleration observed in the uncontrolled conditions may have been the result of isolated gasps or sharp deviations from normal respiratory activity. Normal, regular respiration of constant rate and amplitude does not seem to have significantly contributed to the acceleratory

phase. Therefore, either the acceleratory trend that has been observed represents a respiratory component of the OR, or random variations in respiratory activity unrelated to any quality of the stimulus. The latter of these alternatives does not seem tenable in view of the consistency with which the acceleratory component has been observed. Therefore, the acceleratory trend may well be related to the respiratory component of the OR.

Verbal reports from Ss in Uncontrolled Respiration Condition II provide some insight into the nature of the respiratory response to simple stimuli. Ss frequently reported that the effect of the stimulus was to interrupt their respiration. Furthermore, several Ss noted that if the stimulus came during a portion of their respiratory cycle (inhalation or exhalation) the interruption was greater than if it came between these portions of the cycle. Though these observations have not been objectively substantiated, it is probably safe to speculate that such interruptions cause gasps or compensatory intakes of air which would produce an acceleration of HR. Petelina (1958) reported similar observations on the respiratory component of the OR. Based upon observations of the responses of dogs to tones, Petelina described a "compression reaction" during which breathing was momentarily interrupted. The interruption of respiratory activity was associated with slower, shallower respiration and deceleration of HR. Petelina's observations confirm that the effect of novel stimuli upon respiration may include interruption of ongoing activity. If the exaggerated acceleratory phase were dependent upon the interruption of respiration created by the OR, however, the acceleratory component would be expected to disappear as the deceleratory component (OR) habituated to

the stimulus. This could account for the results of some studies which have reported habituation of the acceleratory phase of the HR response (Germana and Klein, 1968; Raskin, Kotes, and Bever, 1969).

Cardiac activity and respiratory activity are so intimately related that it seems meaningless to attempt to utilize the HR response for the identification of the OR without considering the influence of respiration upon HR. Thus, while research is still needed to specify the separate contributions of the cardiovascular and respiratory systems to the OR, studies which use physiological measures merely for the identification of ORs might be advised to devise a cardio-respiratory measure of the OR. Such a measure would dispense with the current practice of looking at the two as relatively independent and recognize the inseparable functioning of the two systems.

At present, too little is known about the relationship of these two systems as they relate to the OR. It would be interesting to conduct a study in which stimuli were presented at various portions of the respiratory cycle. A comparison of the HR response curves with the stimuli so presented might further clarify the relationship of respiratory and HR components of the OR.

One possible criticism of this study grows out of an attempt to be overly rigorous. In order to be certain that the uncontrolled conditions, which received no respiratory training, spent as much time in the experiment as the condition that did receive training, Ss in Uncontrolled Respiration Condition I were required to remain seated for two ten minute periods prior to the presentation of the stimuli. This empty period seems to have produced a reduced HR in this condition

(although the difference in HR across conditions was not significant). Future research should make certain that all groups have more comparable pre-test experiences.

CHAPTER V

SUMMARY

This study was performed to determine the HR component of the orienting reflex (OR) and, more specifically, to determine if the acceleratory phase of the HR response to simple stimuli could be attributed to respiratory artifacts. Grham and Clifton (1966) reviewed the literature and concluded that the HR component of the OR was deceleration. Four possible explanations for the secondary acceleratory phase were proposed by Graham and Clifton (that it represents an essential component of the OR, a partially inhibited defensive reflex, a startle response, or a specific adaptive response to acoustic stimuli). Of these, only startle was not eliminated by a review of research published since Graham and Clifton's review. A more logical explanation for the acceleratory phase (respiratory artifact) was not examined by Graham and Clifton. Although the relationship between respiration and cardiac activity is well known, few researchers have closely examined the role of respiration in producing the acceleratory trend.

The experiment design was a three factor factorial with repeated measures on two factors (Trial Blocks and Intervals). Fifty-four male Ss were randomly assigned to three experimental conditions. The Controlled Respiration Condition received training to maintain a constant rate and amplitude of respiration. Two other conditions received no

such training. Uncontrolled Respiration Condition I was merely instructed to listen to a tape while seated and Uncontrolled Respiration Condition II was "trained" to attend to their respiratory patterns and to detect any changes that might occur during the presentation of the stimuli. Ten simple stimuli (1000 Hz, 60 db tones) were presented to each S at random intervals of 30, 40 and 50 sec.

The results of the study confirmed that the HR component of the OR was deceleration, and that control of respiration produced a diminished acceleratory phase. The main effects for Intervals were significant which indicated that the stimuli resulted in a significant change in HR. The significant Conditions X Intervals interaction, indicated that a difference in the response curves existed across the conditions. A trend analysis performed on the data of each condition revealed that the response curves for the uncontrolled conditions were approximated by significant quadratic and cubic functions. The Controlled Respiration Condition, however, was approximated by a significant quadratic effect alone. This was interpreted as indicating that the acceleratory component had been significantly diminished by the respiratory control training.

The acceleratory phase of the response curve was attributed to artifactual respiratory activity. It was proposed that future research examine HR and respiratory activity in conjunction rather than treating them as separate components of the OR.

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APPENDIX A

Instructions

INSTRUCTIONS FOR UNCONTROLLED RESPIRATION CONDITION I

(following the attachment of the electrodes) Be seated and just relax for a few minutes.

(at the beginning of the third adaptation period - 15 minutes into the experiment) Please sit up straight, keep both feet flat on the floor, your arms on the arms of the chair, and look straight ahead.

(immediately preceding the tape) Now, I am going to let you listen to a tape. Just relax and look straight ahead. You will not hear anything for a couple of minutes.

INSTRUCTIONS FOR UNCONTROLLED RESPIRATION CONDITION II

(following the attachment of the electrodes) Be seated and just relax for a few minutes.

(preceding the training period - 10 minutes into the experiment) Sit up straight, keep both feet flat on the floor, your arms on the arms of the chair, and look straight ahead. During the next few minutes I want you to observe your normal respiratory pattern. Later in the experiment, I'm going to let you listen to a tape and I want you to be able to detect any changes in your respiration that might occur during the tape. So just relax and become familiar with your normal respiratory pattern.

(immediately preceding the tape) Now I'm going to let you listen to a tape and I want you to attend to your respiratory pattern and report any changes that occur at the end of the tape. You will not hear anything for the first couple of minutes.

INSTRUCTIONS FOR CONTROLLED RESPIRATION CONDITION

(following the attachment of the electrodes) Be seated and just relax for a few minutes.

(preceding initial training period - 5 minutes into the experiment) These are two timers. What I want you to do is adjust these timers to correspond to your normal respiratory pattern. That is, when this light is on, (E pointed to the appropriate timer) I want you to be inhaling and when this light is on (E pointed to the appropriate timer) I want you to be exhaling. You adjust the timers using the top two knobs on each timer. The top knob is for seconds and the second knob is for tenths of a second. When you do this, there is a tendency for you to alter your respiratory pattern and attempt to maintain a rate and amplitude that is uncomfortable, so I would advise you to take a few minutes and observe your normal pattern before you attempt to adjust the timers. Do you understand what I want you to do?

(preceding the second training period - 15 minutes into the experiment) Now, I'm going to turn these timers off and I want you to maintain a constant rate and amplitude without the aid of the timers. I'll let you know how well you're doing. Just relax and maintain a constant rate and amplitude.

(immediately preceding the tape) Fine. Now, I'm going to let you listen to a tape and I want you to concentrate on maintaining a constant rate and amplitude throughout the tape. You won't hear anything for the first couple of minutes.

APPENDIX B

BREAKDOWN OF EXPERIMENTAL PROCEDURE FOR EACH CONDITION

BREAKDOWN OF EXPERIMENTAL PROCEDURE FOR EACH CONDITION

Time	Controlled Respiration Condition	Uncontrolled Respiration Condition I	Uncontrolled Respiration Condition II
5 min.	adaptation period	adaptation period	adaptation period
5 min.	adjusting timers	adaptation period	adaptation period
5 min.	training with the aid of the timers	adaptation period	Ss 'trained' to detect respiratory changes
5 min.	training without the aid of the timers	adaptation period	Ss 'trained' to detect respiratory changes
8 min.	stimuli presented	stimuli presented	stimuli presented

APPENDIX C

TREND COMPONENTS OF

MAIN ANALYSIS

TREND COMPONENTS FOR TRIAL BLOCKS

Source	df	SS	MS	F	P
Trial Blocks	4	942.3271	235.5818	2.8391	.025
Linear	1	696.1633	696.1633	8.3898	.01
Quadratic	1	81.5175	81.5175		
Cubic	1	96.5223	96.5223	1.1632	n.s.
Residual	1	68.1240	68.1240		
Error 1	204	16,927.4303	82.9776		

TREND COMPONENTS FOR INTERVALS

Source	df	SS	MS	F	P
Intervals	19	1,856.5705	97.7142	5.6706	.005*
Linear	1	12.2060	12.2060		
Quadratic	1	947.1751	947.1751	54.9667	.005
Cubic	1	30.4000	30.4000	1.7642	n.s.
Residual	16	866.7894	54.1743	3.1439	.005
Error 2	969	16,697.6003	17.2318		

*Degrees of freedom reduced by 1/2 according to Wilson's 1967 recommendation.

TREND COMPONENTS FOR CONDITIONS X TRIAL BLOCKS INTERACTION

Source	df	SS	MS	F	P
AB	8	1,610.5916	201.3240	2.4262	.025
Linear	2	867.7900	433.8950	5.2291	.01
Quadratic	2	322.9653	161.4827	1.9461	.25
Cubic	2	174.2387	87.1194	1.0499	n.s.
Residual	2	245.5976	122.7988	1.4799	.25
Error 1	204	16,927.4303	82.9776		

TREND COMPONENTS FOR CONDITIONS X INTERVALS INTERACTION

Source	df	SS	MS	F	P
AC	38	818.3782	21.5363	1.7654	.025*
Linear	2	93.3356	46.6678	3.8255	.025
Quadratic	2	56.3639	28.1820	2.3102	.10
Cubic	2	304.4752	152.2376	12.4794	.005
Residual	32	364.2035	11.0692		
Error 3	3876	47,283.8881	12.1991		

*Degrees of freedom reduced by 1/2 according to Wilson's 1967 recommendation.

APPENDIX D

TESTS ON MAIN EFFECTS OF CONDITIONS, TRIAL BLOCKS, AND INTERVALS USING NEWMAN-KEULS PROCEDURE

TESTS ON CONDITIONS MAIN EFFECTS
USING NEWMAN-KEULS PROCEDURE

	Uncontrolled Respiration Condition I	Uncontrolled Respiration Condition II	Controlled Respiration Condition
Means	61.926	65.622	67.160
61.926	------	3.696	5.234
65.622		-----	1.538
67.160			------

p	2	3
$q(p,n_2)$*	2.83	3.40
Wp**	6.71	8.06

*$q(p,n_2)$ = tabulated value for p No. of means and n_2 error degrees of freedom.

**$Wp = q(p,n_2)\ s_{\bar{c}}$

$$s_{\bar{c}} = \sqrt{\frac{\text{MS error between}}{npr}} = 2.37$$

TESTS ON TRIAL BLOCKS MAIN EFFECTS
USING NEWMAN-KEULS PROCEDURE

Trial Block #	1	2	3	5	4
Means	64.313	64.653	65.869	65.139	65.539
64.313	------	.340	.556	.826	1.226*
64.653		-----	.216	.486	.886
64.869			------	.270	.670
65.139				-----	.400
65.539					------

*Significant at the .05 level, $(p,\infty df)$.

p	2	3	4	5
$q(p,n_2)$*	2.77	3.31	3.63	3.86
Wp**	.757	.917	1.006	1.069

*$q(p,n_2)$ = tabulated value for p No. of means and n_2 error degrees of freedom.

**$Wp = q(p,n_2)s_{\bar{t}}$

$$s_{\bar{t}} = \sqrt{\frac{\text{MS error 1}}{npr}} = .277$$

p	2	3	4	5	6	7	8	9	10
$q(p,n_2)$*	2.77	3.31	3.63	3.86	4.03	4.17	4.29	4.39	4.47
Wp**	.688	.837	.918	.977	1.020	1.055	1.085	1.111	1.131

11	12	13	14	15	16	17	18	19	20
4.55	4.62	4.68	4.74	4.80	4.85	4.89	4.93	4.97	5.01
1.151	1.169	1.184	1.199	1.214	1.227	1.237	1.247	1.257	1.267

$$**Wp = q(p,n_2) \quad s_{\bar{B}}$$

$$s_{\bar{B}} = \sqrt{\frac{\text{MS error 2}}{npr}}$$

$*q(p,n_2)$ = Tabulated value for p No. of means and n_2 error degrees of freedom.

TESTS ON INTERVALS MAIN EFFECTS
USING NEWMAN-KEULS PROCEDURE

Beat #	-5	15	-4	-3	13	-2	14	4	5	-1
Means	65.81	65.61	65.55	65.54	65.32	65.32	65.28	65.28	65.07	65.00
65.81	----	.20	.26	.27	.49	.49	.53	.53	.74	.81
65.61		-----	.06	.07	.29	.29	.33	.33	.54	.61
65.55			----	.01	.23	.23	.27	.27	.48	.55
65.54				----	.22	.22	.26	.26	.47	.54
65.32					----	.00	.04	.04	.25	.32
65.32						----	.04	.04	.25	.32
65.28							----	.00	.21	.28
65.28								----	.21	.28
65.07									----	.07
65.00										----

Beat #	12	11	8	10	9	3	7	6	1	2
Means	64.95	64.78	64.62	64.61	64.53	64.51	64.42	64.41	64.11	63.33
65.81	.86	1.03	1.19*	1.20*	1.28*	1.30*	1.39*	1.40*	1.70*	2.48*
65.61	.66	.83	.99	1.00	1.08	1.10	1.19	1.20	1.50*	2.28*
65.55	.60	.77	.93	.94	1.02	1.04	1.13	1.14	1.44*	2.22*
65.54	.59	.76	.92	.93	1.01	1.03	1.12	1.13	1.43*	2.21*
65.32	.37	.54	.70	.71	.79	.81	.90	.91	1.21*	1.99*
65.32	.37	.54	.70	.71	.79	.81	.90	.91	1.21*	1.99*
65.28	.33	.50	.66	.67	.75	.77	.86	.87	1.17*	1.95*
65.28	.33	.50	.66	.67	.75	.77	.86	.87	1.17*	1.95*
65.07	.12	.29	.45	.46	.54	.56	.65	.66	.86	1.74*
65.00	.05	.22	.38	.39	.47	.49	.58	.59	.89	1.67*
64.95	-----	.17	.33	.34	.42	.44	.53	.54	.84	1.62*
64.78		-----	.16	.17	.25	.27	.36	.37	.67	1.45*
64.62			-----	.01	.09	.11	.20	.21	.51	1.29*
64.61				-----	.08	.10	.19	.20	.50	1.28*
64.53					-----	.02	.11	.12	.42	1.20*
64.51						-----	.09	.10	.40	1.18*
64.42							-----	.01	.31	1.09*
64.41								-----	.30	1.08*
64.11									-----	1.08*
63.33										-----

*Significant at the .05 level, (p_{∞}df)

VITA
2

Gerald William Ritter

Candidate for the Degree of

Master of Science

Thesis: THE HEART RATE RESPONSE TO NONSIGNAL AUDITORY STIMULATION WITH RESPIRATION CONTROLLED

Major Field: Psychology

Biographical:

Personal Data: Born in Bedford, Indiana, February 7, 1945, the son of Mr. and Mrs. Gerald Ritter.

Education: Graduated from Springs Valley High School, French Lick, Indiana in May, 1963. Received the Bachelor of Arts degree from Hanover College in 1967 with a major in psychology.

Professional Experience: Graduate teaching assistant, College of Arts and Sciences, Oklahoma State University, 1969-1971.