

Pittsburg State University

Pittsburg State University Digital Commons

Electronic Theses & Dissertations

12-5-1986

Prestartle Stimulus and Interstimulus Interval Effects Upon Startle Response Modification in Man

Humphrey Minx

Pittsburg State University

Follow this and additional works at: <https://digitalcommons.pittstate.edu/etd>

Recommended Citation

Minx, Humphrey, "Prestartle Stimulus and Interstimulus Interval Effects Upon Startle Response Modification in Man" (1986). *Electronic Theses & Dissertations*. 56.
<https://digitalcommons.pittstate.edu/etd/56>

This Thesis is brought to you for free and open access by Pittsburg State University Digital Commons. It has been accepted for inclusion in Electronic Theses & Dissertations by an authorized administrator of Pittsburg State University Digital Commons. For more information, please contact digitalcommons@pittstate.edu.

PRESTARTLE STIMULUS AND INTERSTIMULUS INTERVAL EFFECTS
UPON STARTLE RESPONSE MODIFICATION IN MAN

A Thesis Submitted to the Graduate Division in Partial
Fulfillment of the Requirements for the
Degree of Master of Science

By
Humphrey Minx

PITTSBURG STATE UNIVERSITY

Pittsburg, Kansas

December 5, 1986

ACKNOWLEDGEMENTS

I would like to express my appreciation to the Department of Music and the Instructional Media Department for the use of their equipment, and to Dr. R. Harvard Ritches for the use of his computer, upon which this project depended so much. I would also like to thank Dr. Dean Bishop for his assistance in the statistical analysis of the results of this project; Dr. R. Leon Dinkins and Dr. T. Bruce Daniel for their assistance and advice in the design of the project; and the rest of the Biology faculty for their assistance in recruiting volunteers for this investigation.

A special expression of gratitude is extended to Dr. Leland E. Keller for his insight and interest in my research and career plans. By allowing me to make and correct my own mistakes, Dr. Keller provided me with the self-confidence and experience that I lacked when I first became his student.

Finally, I would like to thank my fiancée, Ms. Jennifer L. Base, for her assistance, understanding and inner strength as she patiently listened to my "ranting and raving" as this project progressed; and especially to my parents, Mr. and Mrs. George J.A. Minx, for instilling, nurturing and supporting my interest in science.

TABLE OF CONTENTS

CHAPTER		PAGE
I.	INTRODUCTION.....	1
II.	LITERATURE REVIEW.....	4
	Startle Response Stimuli.....	4
	Startle Response Modification.....	7
	Muscular Response.....	10
	Cardiovascular Response.....	11
	Neural Pathway.....	15
III.	MATERIALS AND METHODS.....	17
	Acoustic Stimuli.....	17
	Electromyograph.....	21
	Pulse Rate.....	21
	Statistical Analysis.....	24
IV.	RESULTS.....	30
	Electromyograph.....	30
	Pulse Rate.....	31
V.	DISCUSSION.....	34
VI.	CONCLUSION.....	39
	LITERATURE CITED.....	42

ABSTRACT

An investigation was conducted to study the effect of interstimulus interval upon the startle response of eighteen male subjects. Four treatments were used, each consisting of a pre-startle sound and a startle eliciting sound, with an interstimulus interval of 0, 40, 120, or 1000 milliseconds. A treatment consisting of only the startle eliciting stimulus was used as a control.

Electromyography and pulse rate were monitored to record the response to the stimuli. Each subject was tested twice over a two week period, with a week separating the two tests.

Analysis of the electromyogram indicates that a significant difference ($F= 21.82$) does exist between the muscle responses elicited by the treatments and the control response. While not significantly different from the other treatment responses, the treatment with an interstimulus interval of 120 milliseconds appeared to have exhibited the greatest amount of response inhibition. Analysis of pulse rate ($F= 0.41$) indicates that none of the treatments used elicited a significant change in pulse rate.

LIST OF FIGURES

FIGURE	PAGE
1. VIEW OF EXPERIMENTAL APPARATUS.....	26
2. VIEW OF PHYSIOGRAPH TRACING.....	28
3. AVERAGE AMPLITUDE OF EMG RESPONSE TO CONTROL AND TREATMENTS.....	32
4. AVERAGE PULSE RATES RECORDED AT 2 SEC., 18 SEC., AND 50 SEC. DURING EACH TREATMENT.....	33

CHAPTER I

INTRODUCTION

The startle response is defined as an immediate, involuntary response to a sudden or intense stimulus (Jones and Kennedy 1951). As the environment is constantly filled with startle stimuli in the form of sudden or intense sounds, research into the effect of startle upon the physiological systems of the body is of great importance. Investigation of the physiological response to and the modification of the startle reaction may provide some insight into the prevention of startle related accidents.

The most apparent aspect of the response is a rapid tensing of the muscles, usually characterized as a generalized flexion of the head, trunk and limbs (Rossignol 1975). Early investigations of the muscular response were performed using high speed photography and motion pictures. More recent studies have used the Accelerometer to study the response in small animals, and the Electromyograph to observe the muscular response in large animals (such as human subjects) or to record the response in specific muscles (Bierman and Yamshon 1948; Hoffman and Flesher 1964; Basmajian 1967).

Other physiological responses to startle stimuli have been studied. Sudden and intense stimuli have been observed

to cause marked changes in galvanic skin resistance, respiration rate, peripheral vasoconstriction and hormonal secretion (Kryter 1970).

Heart rate response has been reported to be either an acceleration or deceleration in the rate. Eves and Gruzelier (1984) observed individual cardiac differences to high intensity acoustical stimuli and reported that there are two cardiac responses elicited by loud sounds. The primary response consists of an acceleration in heart rate that begins 10 beats after stimulus onset. The secondary response is either an increase or a decrease in heart rate and is dependent upon the rise time of the stimulus (Graham and Clifton 1966).

Modification of the startle response has been elicited by the use of a weak, or neutral stimulus that precedes the startle eliciting stimulus. Changes in the intensity or duration of the pre-startle stimulus will either facilitate or inhibit the response (Ison and Krauter 1975; Hoffman and Ison 1980). The length of the interval between stimuli has also been observed to have some effect upon the startle response. Interstimulus intervals of approximately 100 milliseconds will cause inhibition, while intervals of less than 5 milliseconds will facilitate the response (Ison and Hammond 1971; Stitt et al. 1973).

This investigation was conducted to study the effect of pre-startle stimulus and interstimulus interval upon the

modification of the startle response in eighteen male subjects. Treatments consisted of a pre-startle and a startle eliciting stimulus, with an interstimulus interval of either 0, 40, 120, or 1000 milliseconds. Exposure to only the startle eliciting stimulus was used as a control. The response elicited was monitored using electromyography and by recording the pulse rate.

CHAPTER II

LITERATURE REVIEW

The startle response is defined as an immediate , involuntary response to a sudden or intense stimulus (Jones and Kennedy 1951). The startle response system, far from producing a mere "reflection" of the eliciting stimulus, yields a reaction which is lawfully modulated by diverse shifts in the environmental surroundings (Ison and Hammond 1971).

Startle Response Stimuli

Various types of stimuli have been used to elicit the response. Krauter et.al. (1973) used puffs of air to elicit the startle response in the eye blink reflex of human subjects. Electrical impulses were used upon the skin as a tactile stimulus (Bohlin et.al. 1981) and in direct stimulation of the areas in the brainstem that contain the neural centers which control the response (Hoffman and Ison 1980; Davies 1982; Leaton and Supple 1986).

Light has also been used as a stimulus for eliciting and modifying the startle response. Light flashes of various intensities and durations have been used to elicit the response in both rats (Ison and Hammond 1971) and man (Bohlin et.al. 1981). Sensitization of the startle response

in rats has been reported to have been caused by repetition of photic stimulations (Russo and Ison 1979).

By far the majority of the previous studies have dealt with the use of acoustical stimulation. The acoustical stimuli used have ranged from simple sounds to complex combinations of noise. Prosser and Hunter (1936) studied the effect of sound stimuli upon the startle response and spinal reflexes in white rats. The stimulus used in their investigations consisted of a "click" produced by a telegraph key. The effect of a very sudden, very intense sound upon the physiological systems of the human body was studied using gunshots. Blank cartridges were fired in an enclosed room at irregular intervals while the investigators observed the responses of volunteer subjects (Jones and Kennedy 1951; Sternbach 1960; Pretorius and Van der Walt 1967).

With the advance of the technological age came an increase in the level of noise to which the average individual was exposed. Noise has been observed to have a similar effect upon the physiological systems of the body as startling sound (Kryter 1970). Early investigations dealt with the level of noise in factories and other industrial complexes (Bartlett 1934), but an increase in automobile and aircraft traffic prompted an expansion of the investigations to include residential areas (Lipscomb 1974; May 1978). The effect of intense noise upon productivity, learning, stress,

sleep, and fetal development was the subject of several investigations (Semotan, Semotanova and Oldman 1969,; Kryter 1970; Rosen 1970). The findings of these studies resulted in the formation of an Environmental Protection Agency committee to investigate the problem of noise abatement and control (U.S. Environmental Protection Agency 1971). Nixon (1970), along with several civilian and military investigators, studied the physiological effect of sonic booms, which, with the advent of the Supersonic Transport (S.S.T.), had become a greater threat to the population living in large cities (U.S. Environmental Protection Agency 1971).

Most of the studies investigating the startle effect of sound have used sounds manufactured by sound generators, which have the ability to produce sounds of various frequencies and intensity levels. The intensity level of a sound is considered to be the most important factor in eliciting a startle response (May 1978). Investigations using different intensities of a startle eliciting sound report that marked changes in the response were observed as the intensity of the sound increased (Kryter 1970; Davies 1982; Eves and Gruzelier 1984). It has been reported that while the level of intensity at which the startle response begins to occur varies according to the physiological function measured, there have been few changes of any significance observed to have been induced by sounds with an

intensity level less than 70 decibels (May 1978). Ison and Hammond (1971) observed that, in man, reaction speeds are an increasing function of the intensity of the eliciting stimulus. The frequency of a sound also determines its startle response potential. High frequency sounds have been observed to be more successful in eliciting a startle response than sounds with a low frequency (May 1978).

Startle Response Modification

Modification of the muscular response to a startle stimulus has been observed and demonstrated by several investigators. It has been shown that for a variety of species and reflexes that reflex expression is inhibited if its eliciting stimulus is preceded by a weak or neutral stimulus (Ison and Krauter 1975). A weak acoustical stimulus has been described as a sound with an intensity level just above the threshold of hearing and which, by itself, would not elicit a startle response (Ison and Hammond 1971). The inhibitory effect has been observed to occur relatively immediately after the onset of the pre-startle stimulus and the amount of inhibition elicited is reported to be dependent upon the intensity of the pre-startle stimulus (Ison and Hammond 1971; Hoffman and Ison 1980).

In addition to the intensity of the pre-startle stimulus, the time interval between the two stimuli has also been demonstrated to modify the response. Davies (1970),

studying the startle response of Guinea pigs to the sound of a closing mouse trap, observed that if the same stimulus was heard three seconds before the startle eliciting stimulus, the magnitude of the response was reduced and that the magnitude was reduced even more if the time interval between the two sounds was decreased. Using very sensitive and accurate timing devices, investigations have shown that if the pre-startle stimulus precedes the startle stimulus by 10-1000 milliseconds, the magnitude of the response was reduced and the latency of the response tended to increase (Davies 1974). Intervals of less than 10 milliseconds reduced response latency but the response inhibition was not as profound.

In man, maximum inhibition of the startle response has been recorded when the interstimulus interval is in the range of 100-120 milliseconds (Braff et al. 1978; Hoffman and Ison 1980). In comparison, using rats, Ison and Hammond (1971) reported that inhibition of the response was generally more profound and maximal with an interstimulus interval of 40 milliseconds.

Based on the results of previous investigations of the effect of interstimulus interval upon the startle response, it has been concluded that even with the most intense startle eliciting stimulus, given an appropriate lead interval and a moderately intense pre-startle stimulus, the amplitude of the inhibited response will be no greater than

60% of the amplitude of the uninhibited control response (Hoffman and Ison 1980).

Facilitation of the response was observed when the pre-startle stimulus originated 5.0 milliseconds before and overlapped the startle stimulus (Hoffman and Wible 1969; Stitt et al. 1973).

As in inhibition, an increase in the intensity level of the pre-startle pulse will also facilitate the startle response when a pulse of long duration is used. An increase in the degree of facilitation was demonstrated as the intensity increased until the pre-startle intensity reach an optimum value of 75 decibels. Facilitation of the startle response then decreased as the intensity continued to increase (Ison and Hammond 1971).

Several investigators have studied the effect of background intensity level upon the startle response elicited by acoustic stimuli. Their results indicate that animals who were startled by a sudden, intense stimulus in a background of steady noise reacted more violently than animals who were tested in complete silence (Hoffman and Flesher 1963; Russo and Ison 1979). A marked increase in the recorded response was seen in tests conducted with a background of relatively weak, steady, random noise (Hoffman and Flesher 1963; Stitt et al. 1973). Modification of the startle response , whether facilitation or inhibition, was more noticeable when tests were conducted using a background

intensity level of 60 to 70 decibels (Ison and Hammond 1971).

Muscular Response

The earliest and most used measure of the startle response has been the observation of rapid muscle tensing caused by the sudden or intense sound (May 1978). A startle stimulus has been observed to elicit a set pattern of muscle contractions, which have been described as a generalized flexion of the head, trunk and limbs (Rossignol 1975). Jones and Kennedy (1951) report that the startle response exhibits a definite, complex pattern of flexion, which begins in the head and passes down the body.

Early investigators of the muscular response to a startle stimulus have used high speed photography and motion pictures to record the pattern of flexion. Because these methods proved to be too tedious, demanding and expensive, alternate methods were developed to record and measure the response (Jones and Kennedy 1951).

The startle response in rats or other small animals is usually recorded as a general flexion of the entire body of the animal. To monitor and record the response, a device known as an accelerometer was developed. The device is highly sensitive to the sharp, sudden movements caused by intense stimuli, but is insensitive to the slow, gross movements involved in general activity (Hoffman and Flesher 1964).

Experiments in which human subjects were used, or in which the response of specific muscles was monitored, recorded the muscle activity using an electromyograph. The electromyograph records the electrical potentials produced by the muscle when it contracts (Basmajian 1967). The muscle potential response elicited by a startle stimulus consists of a burst of discharges in the group of muscle fibers from which the potentials were recorded (Prosser and Hunter 1936). Jones and Kennedy (1951) used electromyographic techniques to examine the startle response of several muscles throughout the body. A typical example of their investigation records what appears to be a wave-like change of electrical potentials beginning from the back of the neck and passing down the back of the vertebral column. The travel time of the response recorded from the back of the neck to the small of the back was approximately 0.1 seconds (Jones and Kennedy 1951).

Cardiovascular Response

A review of the literature revealed a variety of different heart rate responses reported to be elicited by startle stimuli. In an investigation involving novel and signal situations, novel or unfamiliar stimuli were reported to produce decelerations in heart rate (Bohlin et al. 1981). Noise and sounds characterized as startle stimuli by their intensity levels and suddenness of onset were also observed

to elicit a deceleration of heart rate (Graham and Clifton 1966; Hatton et al. 1970; Davies 1974).

On the opposite end of the spectrum, Pretorius and Van der Walt (1967) reported that heart rate accelerated due to a startle stimulus. Using blank pistol shots as startle stimuli, they observed that the heart rates changed from the base line values recorded before the stimulus and that the maximum increase occurred 12-30 seconds after stimulus onset.

Eves and Gruzelier (1984) conducted a study on heart rate response to high intensity auditory stimulation based on earlier reports that there is a primary and secondary cardiovascular response to sound stimuli, and that the secondary response differs, either an increase or decrease in heart rate, from individual to individual. The primary cardiac response was observed to be an increase in heart rate that occurred 10 beats post-stimulus and was considered by Eves and Gruzelier to be a component of startle. The secondary response occurred at 17 seconds post-stimulus and returned to baseline by 50 seconds post-stimulus (Eves and Gruzelier 1984). The secondary deceleration of heart rate was thought to be vagally induced, while acceleration was thought to be caused by either a relatively complete removal depressor activity by the startle stimulus, or this factor plus the activation of the sympathetic centers which are

reciprocally related to the innervation of the parasympathetic nerves (Berg and Beebe-Center 1941).

The rise time of the startle stimulus may dictate whether the secondary cardiac response will be acceleration or deceleration. Previous studies report that for a stimulus to be effective in eliciting a startle response, the maximum intensity level of the stimulus must be reached within 12 milliseconds of onset (Graham and Clifton 1966). Rapid onsets were observed to elicit steep and large accelerations in heart rate. Gradual stimulus onsets produced a period of deceleration and a more gradual and smaller phase of acceleration (Hatton et al. 1970).

Modification of the cardiovascular response to startle stimuli has been reported to be controlled by the length of the lead time between the startle eliciting stimulus and the pre-startle stimulus. Graham (1975) reported that interstimulus intervals of less than 240 milliseconds tended to facilitate heart rate, while lead times of greater than 240 milliseconds were observed to elicit heart rate inhibition. Using electrical shocks as a pre-startle stimulus that terminated upon onset of an auditory startle stimulus, Kanfer (1958) reported that a progressive acceleration in heart rate occurred from the pre-tone to the post-tone periods. Deceleration was seen in investigations using an interstimulus interval of 2000 milliseconds. Heart rates measured during the interval between the stimuli were

reported to decrease sharply (Graham 1975; Bohlin et al. 1981).

In addition to changes in heart rate, other cardiovascular responses to startle stimuli have been observed. Bartlett (1934) reported that intense auditory excitation directly increased arterial pressure, especially in the systolic phase. The increase in pressure was most likely the result of vasoconstriction. Constriction of blood vessels has been reported to occur each time a noise stimulus was applied, and occurred whether the subject was awake or asleep (Still, 1970). Vasoconstriction is reported to occur independent of annoyance or any other emotion (Rosen 1970). The vasoconstriction observed may be partially the result of another physiological response to startle stimuli; the stimulation of the adrenal gland to secrete epinephrine and norepinephrine. Epinephrine causes selective peripheral vasoconstriction and norepinephrine causes a more general vasoconstrictive response (May 1978; Sternbach 1986).

Other involuntary responses elicited by startle stimuli include marked changes in galvanic skin resistance and respiration rate. These responses were reported to be elicited slightly slower than the muscular response (May 1978). A sudden and intense stimulus was reported to cause a decrease in the rate of respiration (Cohen 1977) and an

increase in breathing amplitude was observed, indicating that inspirations were becoming very deep (Kryter 1970).

Phares (1933) observed the effect of a startling stimulus upon galvanic skin resistance when one of the subjects she was testing was startled by the sound of a window shade that had suddenly sprung loose. Skin resistance was observed to decrease relatively immediately after the stimulus. The change in skin resistance was also observed to be susceptible to modification when the startle stimulus was preceded by a weak, neutral stimulus (Grings and Schell 1969).

Neural Pathway

Observations of the latencies of the fastest units of the startle response (15-25 milliseconds) indicate that the response probably does not involve the cerebral cortex, which shows a minimum latency of 8 milliseconds to auditory stimulation (Prosser and Hunter 1936). This is supported by evidence that startle reactions have been recorded in anencephalic children and decerebrated animals (Rossignol 1975). Responses observed in anencephalics indicate that startle responses are probably mediated by the lower extrapyramidal centers within the reticular formation (Jung 1960).

Electrical stimulation of various loci in the reticular nucleus concurrently with reflex elicitation indicate that facilitation of the startle response is controlled by the

nucleus reticularis pontalis oralis and the nucleus reticularis caudalis. Inhibition of the response was elicited by stimulation of the nucleus reticularis gigantocellularis region of the midbrain (Ison and Hammond 1971). The cell bodies of the axons in the reticularis nucleus project into the final relay of the neuronal pathway, the reticulo-spinal tract. Stimulation of the tract produces spinal excitability changes similar to changes induced by natural stimuli like sound (Rossignol 1975; Hoffman and Ison 1980). From the spinal tract, efferent fibers carry impulses to the various systems of the body.

On the sensory side of the response pathway, electrical stimulation of the ventral cochlear nucleus has elicited a response similar to acoustic startle, showing habituation and sensitization, and prepulse facilitation and inhibition (Davies et al. 1986). Based on information accumulated from several previous investigations, it seems likely that the startle response involves the following neural pathway: ventral cochlear nucleus; inferior colliculus; the lower extrapyramidal region of the reticular nucleus, where facilitation and inhibition are controlled; the reticulo-spinal tract; anterior horn of the spinal cord and efferent fibers to the rest of the body (Prosser and Hunter 1936; Jung 1960; Ison and Hammond 1971; Hoffman and Ison 1980; Davies et al. 1986).

CHAPTER III

MATERIALS AND METHODS

A total of eighteen males were used in this investigation, all were volunteers solicited at random from the General Zoology and the Anatomy & Physiology classes at Pittsburg State University. Interested volunteers were asked to select a convenient test time from a schedule of experimental times provided by the investigator. The time selected by the subjects dictated the order in which the subjects were tested. Subjects ranged in age from eighteen to twenty years of age.

Acoustic Stimuli used

The sounds used in this investigation were generated using a Texas Instrument Computer (TI99/4A). The "CALL SOUND" program built into the computer was used to produce the pre-startle and the startle eliciting stimuli, and the specific interstimulus intervals upon which this project is based. All sounds, interstimulus intervals and intertreatment intervals were programed and copied onto a memory tape to allow the same sounds and intervals to be recalled when necessary.

Both the pre-startle and the startle stimuli were generated with a frequency of 1000 hertz (cycles/second). This frequency has been used in several previous studies and resulted in definite responses. Still (1970) reports that

while a normal, young human ear can hear sounds with frequencies from 20-20000 hertz, the human ear is most sensitive to frequencies between 1000 and 4000 hertz (May 1978). Most human speech occurs in the frequency bands from 600-4800 hz (Still 1970) and Kryter (1970) notes that at a sound pressure level of approximately 70 decibels, a 1000 hz tone becomes very effective in eliciting the startle response.

Loudness of a sound is considered a psychological measurement rather than an actual physical measurement of the strength of sensation received by the eardrum and transmitted to the brain (Still 1970). Since loudness would be a subjective measurement, sound intensity is used to compare the strength of sensations elicited by various sounds.

The intensity of a sound is a measurement of the amount of energy present in the sound waves. There are two basic scales against which sounds are measured. One is Sound Power Level, measured in Watts of electricity radiated by the sound source; and the other is Sound Pressure Level, which is a measurement, in terms of atmospheric pressure, of sound as it passes a point any given distance from the source (Still 1970). Sound Pressure Level has become the primary mode of measurement for sound, although Sound Power Level is still used in certain instances. The basic unit of measurement of the Sound Pressure Scale is the decibel (db),

which is a logarithmic unit that expresses the ratio between two sound pressures (Lipscomb 1974). The most common reference point for the Sound Pressure scale is 0.0002 microbars, where one bar is equal to the atmospheric pressure at sea level; this pressure represents zero on the decibel scale (Still 1970).

One approach to measuring sounds in a way that reflects on their loudness is to alter the measured frequency spectrum of the sound. By modification of some of the upper and lower frequency bands detected by the human ear, a weighted sound level can be obtained which to some extent correlates with the loudness of the sound (May 1978). The "A" scale is regarded to approximate the human ears frequency response by slightly filtering out the lower frequencies. In previous studies, the "A" scale has been used most favorably as the means for determining the human correlates of sound (Lipscomb 1974).

In this investigation, all sound measurements were made using the "A" weighting scale. The pre-startle stimulus, the startle eliciting stimulus and the background noise level were all calibrated and measured using a Sound Level Meter (Model 1551-C) manufactured by the General Radio Company of Concord, Massachusetts. The pre-startle stimulus was generated with an intensity of 73 db(A) and a duration time of 20 milliseconds. The startle eliciting

stimulus was 60 milliseconds in duration and was generated at a sound pressure level of 92 db(A).

A background noise level of 61 db was heard throughout the entire test. While the background sound was not initially included in the experimental design, previous studies report that a continuous level of low intensity noise superimposed over the pre-startle and the startle stimuli caused a greater response to the stimuli than was observed with a background of complete silence (Hoffman and Flesher 1963; Kryter 1970; Stitt et al. 1973).

Treatments consisted of both a pre-startle and a startle stimulus, with an interstimulus interval of either 0, 40, 120, or 1000 milliseconds. Exposure to the startle stimulus only was used as a control. Ison and Hammond (1971) reported that an interstimulus interval of 40 milliseconds elicited the maximum amount of inhibition of the startle response in rats. In man, an interstimulus interval of 120 milliseconds was observed to cause the greatest amount of response inhibition (Graham 1975). Previous studies of the effect of interstimulus intervals upon startle response modification report that as the interval between the stimuli increased, the amount of response inhibition was observed to increase until the maximum inhibition interval was reached. Interstimulus intervals beyond the optimum range were observed to elicit a

decreased amount of inhibition (Ison and Hammond 1971; Krauter et al. 1973; Stitt et al. 1973).

All treatments and the control were separated by an intertreatment interval of 60 seconds. A 60 second pre-test interval preceded the control and a 60 second post-test interval followed the last treatment. The entire test, consisting of all treatments, intervals and the control, was recorded on a TDK cassette tape (120 microsecond bias).

Electromyograph

The muscle response to the treatments was recorded using an Electromyograph, which consisted of a set of three surface electrodes and a Physiograph preamplifier. While the amplitude of the muscle action potentials appears to dependent to some degree on the thickness of the tissue interposed between the muscle and the skin, previous studies indicate that the data secured using surface electrodes are essentially the same as those derived from needle electrodes which are inserted directly into the muscle (Bierman and Yamshon 1948).

Pulse Rate

Pulse rate was monitored using a Photoelectric Pulse Pick-up, which is a plethysmographic transducer for detecting and recording the cardiovascular pulse pressure wave. The transducer detects changes in light intensity within the tissues cause by pulse variations in blood volume; these changes are recorded as pulse rate.

All physiological responses elicited by the startle stimuli were recorded using an E & M PHYSIOGRAPH-SIX (Narco Biosystems of Houston, Texas) (Figure 1). Although several studies report that ink writing recording devices like the Physiograph have a much more limited response to than a Cathode Ray Oscilloscope, almost all agree that the disadvantage is outweighed by the fact that the ink writing recording device produces an inexpensive, immediate and permanent record (Figure 2) which can be observed during and after the investigation (Bierman and Yamshon 1948; Jones and Kennedy 1951).

The investigation required that all subjects be tested three times, in one week increments, to ensure statistical validity. However, during the first week of testing, a problem with the tape cassette of the recorded sound program prompted the rerecording of the sounds onto a new tape cassette. As several subjects had already been tested with the flawed tape, it was decided that only the data recorded in the second and third tests (using the new cassette tape) would be used in the statistical analysis of the data.

Prior to testing, the subjects were prepared by attaching the surface electrodes to the skin overlying the Upper Trapezius muscle in the dorsal region of the neck. The Upper Trapezius was selected based on information cited in an earlier study on the electromyographic responses to sudden and intense stimuli. In this study, Jones and

Kennedy (1951) reported that a startle response was obtained most readily and consistently from the region of the neck. In many instances, a startling stimulus too mild to produce an electromyographic response elsewhere in the body produced one in the neck. Among the different placements of the electrodes upon the neck, the strongest responses were recorded from the upper part of the Trapezius (Jones and Kennedy 1951). The first electrode was placed approximately 3.0 cm. below the mastoid process. The electrodes were spaced 3.0 cm apart to allow for easy application and comfort, and were within the 2.5 to 5.0 centimeter spacing suggested by earlier electromyographic investigations (Bierman and Yamshon 1948; Jones and Kennedy 1951). The electrodes were fastened to the skin with adhesive bandage strips and an electrode gel was used to maintain good electrical contact between the skin and the electrode.

Once the electrodes were in place, the subject was instructed to lie down on a lawn chair that had been grounded and covered with a sleeping bag. Once in the lawn chair, the subject's head was roughly at a 20 degree angle from the horizontal plane. The photoelectric pulse pick-up was attached to the distal portion of the right index finger.

After attachment of the pulse pick-up, subjects were asked to relax while the electromyograph and the pulse pick-up systems were calibrated. The electromyograph was

calibrated at 0.05 mV/cm of amplitude. While the analysis of the muscular response would be performed using a measurement of amplitude size (in millimeters), calibration using millivolts allowed the sensitivity of the system to be the same in each of the three tests. As only the pulse rate was to be analyzed, the pulse pick-up sensitivity was adjusted until a clear series of pulse waves could be recorded.

After calibration, a stereophonic head set was placed on the subjects ears and the cassette tape of the recorded sound program was played on an Audiotronics-136 Cassette Tape Player. The sounds on the tape cassette were calibrated through the headset using the sound level meter before each subject was tested. Paper speed on the Physiograph was set at 2.5 cm/sec. The start of the test, treatment onset and the end of the test were marked on the tracing using an event marker.

Statistical Analysis

Electromyographic response to each treatment was measured in millimeters of response amplitude. An average response amplitude for each treatment was calculated from the data in tests I and II. A Two-way Analysis of Variance was performed on the data to determine if treatment responses differed significantly from each other and to determine if there was significant difference in the responses elicited between subjects. The average values of the responses were then analyzed using a Student-Newman-Kuel

Multiple Range Test to determine if the treatment responses differed significantly from the control response.

Pulse rate was analyzed by counting the pulse waves in 2 second increments after stimulus onset. The values in each 2 second increment were then corrected to beats/minute. Eves and Gruzelier (1984) report that a maximum change in heart rate was observed around 17 seconds after stimulus onset and returned to near baseline by 50 seconds post-stimulus. Based on this information, pulse rates recorded at intervals 2 seconds, 18 seconds and 50 seconds after stimulus onset were analyzed to determine if the stimuli elicited a change in pulse rate. The pulse rate response to the stimuli was analyzed using a Nested Classification Analysis of Variance.

Figure 1

VIEW OF EXPERIMENTAL APPARATUS

- (1) E & M PHYSIOGRAPH "SIX"
- (2) Audiotronics-136 Cassette Tape Player
- (3) EMG Preamplifier
- (4) Audiotronics HS-20V Stereo Headphones
- (5) Photoelectric Pulse Pick-up



FIGURE 1.

Figure 2

VIEW OF PHYSIOGRAPH TRACING

- (1) Time Line (1.0 second increments)
- (2) Electromyograph (with ECG artifact)
- (3) Pulse Rate

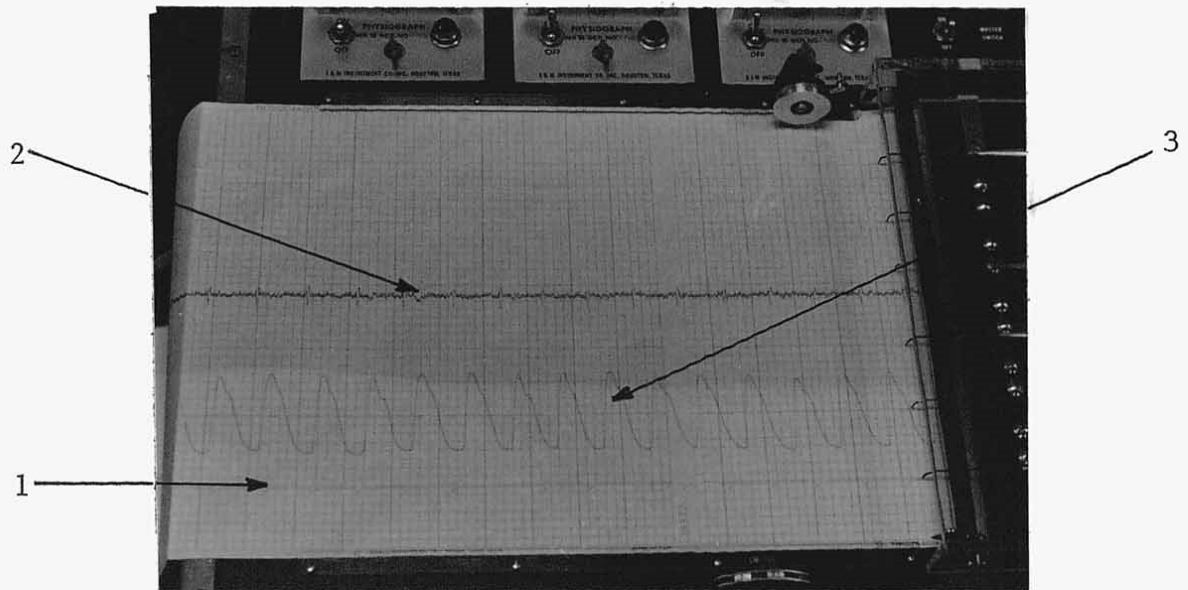


FIGURE 2.

CHAPTER IV

RESULTS

Electromyograph

Mean values of 33.2, 11.5, 4.8, 4.0 and 6.9 (mm) were calculated from the response amplitudes elicited by the control and the four treatments.

An Analysis of Variance was performed on the electromyographic data to determine if there was a significant difference in the muscular response elicited by each treatment. Also examined were the responses between the subjects to determine if the subjects themselves differed in their response to the treatments. F-value of the treatments ($F = 21.82$, $df = 4/68$, $P < 0.05$) did indicate that there was a significant difference in the responses elicited by the treatments. F-value of the subjects ($F = 1.05$, $df = 17/68$, $P < 0.05$) indicated that there was no significant difference in the responses elicited by each subject. A Multiple Range Test was performed to determine which treatments differed. Analysis showed that the response elicited by the four treatments did not differ significantly, but that the treatment responses did differ significantly from the control response. The mean values of the response amplitude are plotted in Figure 3. While not significantly different from the other treatment responses, the response amplitude elicited with an interstimulus

interval of 120 milliseconds appears to have been inhibited the most.

Pulse Rate

Pulse rates were recorded at 2, 18 and 50 seconds post-stimulus during each treatment. Mean values of 68.5, 67.8 and 64.0 (beats/min) were recorded during the control treatment; 65.7, 67.4, 66.7 during Treatment 1; 64.3, 68.4, 67.3 during Treatment 2; 66.3, 70.3, 64.7 during Treatment 3; and 65.1, 67.3, 65.6 during Treatment 4.

Analysis of the pulse rate data involved the use of a Nested Classification to determine if the changes in pulse rate elicited by the treatments differed significantly. Also analyzed were the changes between the subjects and changes in pulse rate that were recorded at 2, 18, and 50 seconds intervals in each treatment.

F-values for the treatments, times and individuals ($F(\text{treat}) = 0.41$, $df = 4/270$, $P < 0.05$; $F(\text{time}) = 0.29$, $df = 10/270$, $P < 0.05$; $F(\text{Indiv.}) = 0.02$, $df = 255/270$, $P < 0.05$) showed no significance. The mean values of pulse rate recorded at 2, 18 and 50 second intervals for each treatment are plotted in Figure 4. While not significant, the pulse rates recorded during the 18 second intervals appear to be much faster than rates recorded at the 2 second and 50 second interval.

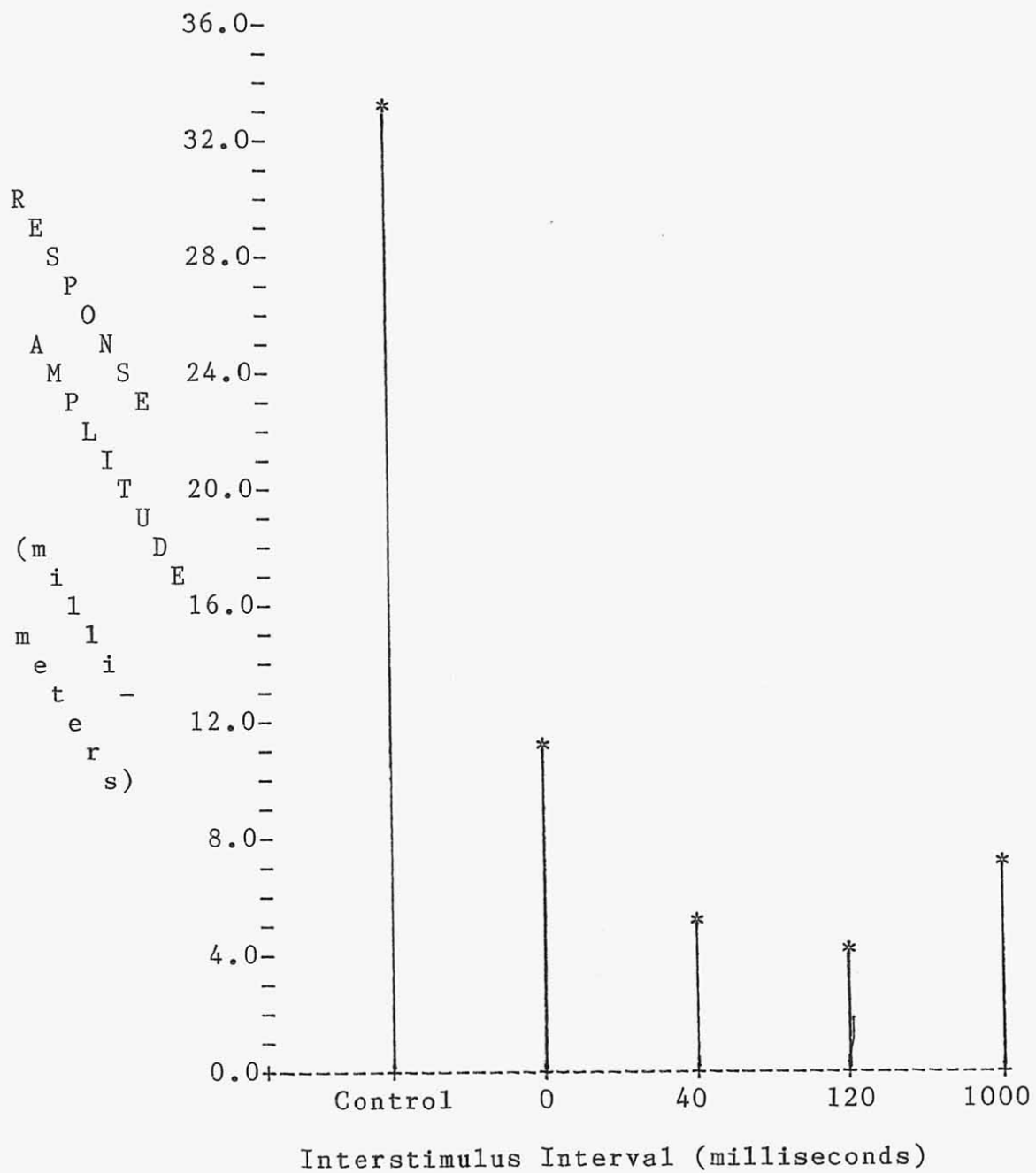


Figure 3. Average amplitude of EMG response to control and treatments.

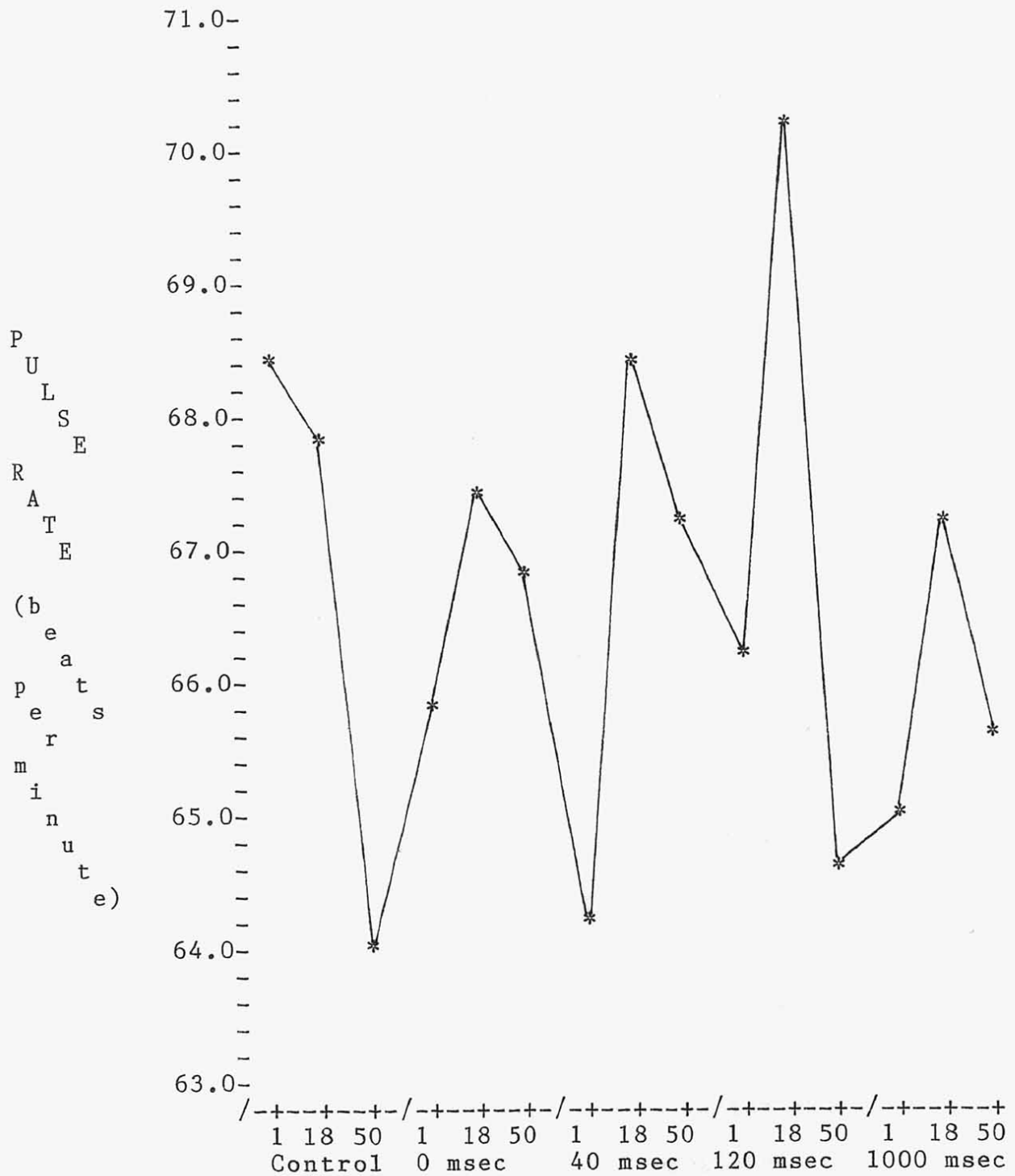


Figure 4. Average pulse rates recorded at 2 sec., 18 sec., and 50 sec. during each treatment

CHAPTER V

DISCUSSION

The startle reflex system, far from producing a mere "reflection" of the eliciting stimulus, yields a reaction which is lawfully modulated by diverse shifts in the environment (Ison and Hammond 1971). Reflex modification refers to the phenomenon whereby the reflex elicited by one stimulus is modified by the prior presentation, withdrawal or change of another (usually weaker) stimulus (Hoffman and Ison 1980).

That reflex modification is not due to pre-stimulus initiated modification of sensory input is evidenced by examination of the Intratympanic reflex (Graham 1975). Contraction of the middle ear muscles in the presence of an intense acoustic stimulus dampens the vibrations of the ossicles and thus modulates the deformation of the oval window made by the sound pressure waves. However, the maximum level of protection offered by the reflex is not more than 15 decibels, a reduction which is not sufficient to produce the level of response inhibition obtained with a pre-startle stimulus (Ison and Hammond 1971).

The results of the present investigation show a significant difference in the electromyographic response of the four treatments when compared to the control response. Figure 3 illustrates that the treatment responses all show

signs of inhibition when compared to the control. The results are supportive of work performed by Davies (1974) who reported that the startle response is reduced when a relatively weak pre-stimulus precedes a startle eliciting stimulus by 10-2000 milliseconds. Comparison of Treatment 1 to the control response illustrates that the startle response is modified by the pre-startle stimulus. Kanfer (1958) recorded a startle response using a pre-startle stimulus that terminated upon onset of the startle stimulus. The results of Kanfers experiment, along with other studies report that responses obtained with an interstimulus interval of less than 5 milliseconds had reduced latency periods, but inhibition of the response was not as profound (Davies 1974; Braff et al. 1978).

While the difference between the treatment responses was not found to be significant, Figure 3 does show an apparent difference in the amplitude of each response. Maximum inhibition of the startle response seems to have occurred during the third treatment when the interstimulus interval was 120 milliseconds; followed by treatment 2, which, with an interstimulus interval of 40 milliseconds, resulted in the next greatest amount of response inhibition. Hoffman and Ison (1980), while studying the startle response of rats, observed that maximum inhibition of the response occurred when an interstimulus interval of 40 milliseconds was used. Intervals with time lengths less than or greater

than 40 milliseconds resulted in a greater response amplitude. The optimum inhibition interval for man is reported to be much longer than the interval for the rat. Investigations of the startle response in humans have reported that maximum inhibition occurred when the pre-startle stimuli presented approximately 120 milliseconds before a startle eliciting stimulus (Braff et al. 1978; Hoffman and Ison 1980).

Analysis of pulse rate indicates that no significant change was elicited by treatments. Previous investigators have reported that the length of the interstimulus interval dictates whether the change in heart rate will be facilitation or inhibition. Lead times between stimuli of less than 240 milliseconds are reported to have elicited a facilitation of the heart rate. Lead times greater than 240 milliseconds have been observed to inhibit the heart rate response to a startle stimuli (Graham 1975). An investigation using pre-startle and a startle stimuli with no interstimulus interval resulted in an acceleration in heart rate, while studies using a 2000 millisecond lead time report that heart rate decelerated upon onset of the stimuli (Kanfer 1958; Bohlin et al. 1981).

Figure 4 illustrates the pulse rates measured at 2, 18 and 50 second intervals within each treatment. While not significant, the graph does show an apparent increase in heart rate during the 18 second intervals in each treatment.

Eves and Gruzelier (1984) report that two cardiac responses are elicited by startle stimuli. The primary cardiac response is an acceleration in heart rate which is usually seen 10 post-stimulus. The secondary response is either an increase or a decrease in heart rate which usually occurs around 18 seconds post-stimulus and returns to near baseline by 50 seconds post-stimulus. While not significant, it does appear that a secondary pulse rate response was observed in this investigation.

The exact mechanism of the startle response has yet to be identified. However, investigators studying the response have proposed a theoretical model of the mechanism involved. The proposed model is composed of two pathways; one directly leading, and the other indirectly leading to the startle response centers in the nucleus reticularis. When pre-startle stimuli elicit activity in the direct pathway, they presumably prime the pathway so that a subsequent stimulus is more effective and thus results in facilitation of the response (Bohlin et al. 1981). It is assumed that the indirect, or inhibitory pathway transmits signals more slowly than the direct pathway, perhaps because it incorporates more synapses or consists of neurons of a smaller diameter. Maximum inhibition of the response would occur when the interval between the pre-startle stimulus and the startle eliciting stimulus is just long enough for the transmission of the pre-startle signal along the inhibitory

neurons to have produced its maximal effect at the moment that the excitatory input from the startle stimulus arrived (Hoffman and Ison 1980).

CHAPTER VI

CONCLUSION

Based on the results of this investigation, it is concluded that a weak pre-startle stimulus does significantly modify the muscular response elicited by a startle stimulus. While not significant, the amount of inhibition observed is apparently dependent upon the interstimulus interval length; maximum inhibition seems to have occurred with an interval of 120 milliseconds.

A significant modification in pulse rate was not observed to have been elicited by the pre-startle stimulus in any of the treatments. While not significant, a secondary cardiac response seems to have been elicited during each treatment.

Investigations of the physiological response to sudden or intense stimuli have provided valuable information on the effect of noise and other startle stimuli upon health and performance. A startle response may prove to be detrimental to a patient suffering from severe heart trouble, or equally pernicious in those professions which involve the use of precision tasks. Investigations of the startle response may develop new techniques to alleviate the problems caused by startle stimuli. Use of a background of low level, pulsed noise has been observed to reduce the startle response, and it has been recommended that professions which require

precision work use a background of low level, pulsed noise to reduce the chance that a sudden, intense noise will produce startle and disrupt work (Hoffman and Flesher 1963). Startle stimuli have also been reported to cause accidents in flying, especially in situations which require rapid analysis, correct decision and sensitive reactions. It has been proposed that further research on the effect of startle stimuli on efficiency reduction be conducted, particularly in those professions where a short reduction in efficiency would threaten safety (Vlasak 1969).

While the physiological response to startling stimuli has been studied using various types of stimuli (acoustic, visual, tactile) at different levels of intensity, frequency and duration; the setting in which they have been presented has remained the same. Ison and Krauter (1975) write that in several experiments, the subject has been tested in an austere and impoverished environment. While this is a natural consequence of experimental routine of simplification and control, it is difficult to extend these findings to the more general, non-laboratory environment. Based on their recommendations, it is also recommended by this investigator that further research into the startle response be conducted using startle stimuli that are representative of those found in a work or residential setting. Stimuli such as a car horn, a dropped glass, a flash of lightning, or even a loud shout, are more likely to

be encountered in a non-laboratory setting than a single tone of a specific intensity, frequency and duration. By using stimuli such as these, a much more accurate description can be drawn of the physiological response to sudden or intense stimuli.

LITERATURE CITED

- Bartlett, F.C. 1934. The problem with noise. Cambridge University Press, London, 87 pp.
- Basmajian, J.V. 1967. Muscles alive: their function revealed by electromyography. The Williams and Wilkins Company, Baltimore, Maryland, 421 pp.
- Bierman, W., and L.J. Yamshon. 1948. Electromyography in kinesiology evaluations. Archives of Physical Medicine 29:206-211.
- Berg, R.L., and J.G. Beebe-Center. 1941. Cardiac startle in man. Journal of Experimental Psychology 28:262-279.
- Bohlin, G., F.K. Graham, L.D. Silverstein, and S.A. Huckley. 1980. Cardiac orienting and startle blink modification in novel and signal situations. Psychophysiology 18:603-611.
- Braff, D., C. Stone, E. Calloway, M. Geyer, I. Glick, and L. Bali. 1978. Prestimulus effects on human startle reflex in normals and schizophrenics. Psychophysiology 15:339-343.
- Cohen, A. Extraauditory effects of acoustic stimulation. In: Lee, D.H.K., H.L. Falk, S.D. Murphy, and S.R. Geiger, eds. 1977. Handbook of physiology - section 9: reactions to environmental agents. American Physiological Society, Bethesda, Maryland, p31-41.
- Davies, M. 1970. Effects of interstimulus interval length and variability of the startle response habituation in the rat. Journal of Comparative and Physiological Psychology 72:177-192.
- Davies, M. 1974. Signal to noise ratio as a predictor of startle amplitude and habituation in the rat. Journal of Comparative and Physiological Psychology 86:812-819.
- Davies, M., T. Parisi, D.S. Gendelman, M. Tischer, and J.H. Kehne. 1982. Habituation and sensitization of startle reflexes elicited electrically from the brainstem. Science 218:688-690.
- Davies, M., R.L. Commisaris, J.V. Casella, S. Yang, L. Dember and T.P. Harty. 1986. Differential effects of dopamine agonists on acoustic and electrically elicited startle responses: comparison to effects of strychnine. Brain Research 371:58-69.

- Eves, F.F., and J.H. Gruzelier. 1984. Individual differences in the cardiac response to high intensity auditory stimulation. *Psychophysiology* 21:342-352.
- Graham, F.K., and R.K. Clifton. 1966. Heart rate change as a component of the orienting response. *Psychological Bulletin* 65:305-320.
- Graham, F.K. 1975. The more or less startling effects of weak prestimulation. *Psychophysiology* 12:238-248.
- Graham, F.K., L.E. Putnam, and L.A. Leavitt. 1975. Lead stimulation effects on human cardiac orienting and blink reflexes. *Journal of Experimental Psychology: Human Perceptions and Behavior* 1:161-169.
- Grings, W.W., and A.M. Schell. 1969. Magnitude of electrodermal response to a standard stimulus as a function of intensity and proximity of a prior stimulus. *Journal of Comparative and Physiological Psychology* 67:77-82.
- Hatton, H.M., W.R. Berg, and F.K. Graham. 1970. Effects of Acoustic rise time on heart rate response. *Psychonomic Science* 19:101-103.
- Hoffman, H.S., and M. Flesher. 1963. Startle reaction: modification by background acoustic stimulation. *Science* 141:928-930.
- Hoffman, H.S., and M. Flesher. 1964. An apparatus for the measurement of the startle response in the rat. *American Journal of Psychology* 77:307-309.
- Hoffman, H.S., and B.L. Wible. 1969. Role of weak signals in acoustic startle. *Journal of the Acoustical Society of America* 47:481-497.
- Hoffman, H.S., and J.R. Ison. 1980. Reflex modification in the domain of startle: I. Some imperical findings and their implication for how the nervous system processes sensory input. *Psychological Reviews* 87:175-189.
- Ison, J.R., and G. Hammond. 1971. Modification of the startle reflex in the rat by changes in the auditory and visual environments. *Journal of Comparative and Physiological Psychology* 75:435-452.

- Ison, J.R., and E.E. Krauter. 1975. Acoustic startle reflexes in the rat during consummatory behavior. *Journal of Comparative and Physiological Psychology* 89:39-49.
- Jones, F.P., and J.L. Kennedy. 1951. A electromyographic technique for recording the startle pattern. *Journal of Psychology* 32:63-68.
- Jung, R. The extrapyramidal motor systems. In: Field, J., H.W. Magoun, and V.E. Hall, eds. 1960. *Handbook of physiology - section 1: neurophysiology, Vol II.* American Physiological Society, Washington, D.C., p863-928.
- Kanfer, F.H. 1958. Effect of warning signal preceding a noxious stimulus on verbal rate and heart rate. *Journal of Experimental Psychology* 55:73-80.
- Kryter, K.D. 1970. *The effects of noise on man.* Academic Press, Inc., New York, 633 pp.
- Landis, C., and M.M. Bolles. 1949. *Textbook of abnormal psychology.* MacMillian Company, New York, 556 pp.
- Leaton, R.N., and W.F. Wipple, Jr. 1986. Cerebellar vermis: habituation of the acoustic startle response. *Science* 232:513-515.
- Lipscomb, D.M. 1974. *Noise, the unwanted sounds.* Nelson-Hall Company, Chicago, 342 pp.
- May, D.N., ed. 1978. *Handbook of noise assessment.* Van Nostrand-Reinhold Company, New York, 391 pp.
- Nixon, C. Man and sonic boom: environmental change. In: Welch, B.L., and A.S. Welch, eds. 1970. *Physiological effects of noise.* Plenum Press, New York, p309-332.
- Phares, M. 1933. Analysis of music appreciation by means of psychogalvanic reflex technique. *Journal of Experimental Psychology* 17:117-140.
- Pretorius, P.J., and J.J. Van der Walt. 1967. Influence of a loud acoustic stimulus on the ultra low frequency acceleration ballistocardiogram in man. In: Welch, B.L. and A.S. Welch, eds. 1970. *Physiological effects of noise.* Plenum Press, New York, p342. (Abstract).

- Prosser, C.L., and W.S. Hunter. 1936. The extinction of startle responses and spinal reflexes in the white rat. *American Journal of Physiology* 117:609-618.
- Rosen, S. Noise, hearing and cardiovascular function. In: Welch, B.L., and A.S. Welch, eds. 1970. *Physiological effects of noise*. Plenum Press, New York, p57-66.
- Rossignol, S. 1975. Startle responses recorded in the leg of man. *Electroencephalography and Clinical Neurophysiology* 39:389-397.
- Russo, J.M., and J.R. Ison. 1979. Sensitization of the rat acoustic startle response by repetition of a photic stimulus. *Physiological Psychology* 6:204-209.
- Semotan, J., M. Semotanova, and M. Oldman. 1969. Startle and other human responses to noise. *Journal of Sound and Vibrations* 10:480-489.
- Sternbach, R.A. 1960. A comparative analysis of autonomic responses in startle. *Psychosomatic Medicine* 22:204-210.
- Still, H. 1970. *In quest of quiet*. Stackpole Books, Harrisburg, Pennsylvania, 321 pp.
- Stitt, C.L., H.S. Hoffman, and R. Marsh. 1973. Modification of the rat startle reaction by termination of anticident acoustic signals. *Journal of Comparative and Physiological Psychology* 84:207-215.
- U.S. Environmental Protection Agency. 1971. *Public hearings on noise abatement and control: Vol. VII - physiological and psychological effects*. U.S. Government Printing Office, Washington, D.C., 453 pp.
- Vlasak, M. 1969. Effect of startle stimuli on performance. *Aerospace Medicine* 40:124-128.
- Watson, P.J. 1978. Nonmotor functions of the cerebellum. *Psychological Bulletin* 85:944-964.