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The development of voluntary cardiovascular control

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To the Graduate Council:

I am submitting herewith a dissertation written by Roger A. Kleinman entitled "The development of voluntary cardiovascular control." I have examined the final electronic copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Psychology.

Jasper Brener, Major Professor

We have read this dissertation and recommend its acceptance:

Accepted for the Council:

Carolyn R. Hodges

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

June 8, 1970

To the Graduate Council:

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We have read this dissertation
and recommend its acceptance:

Accepted for the Council:

Vice Chancellor for
Graduate Studies and Research

THE DEVELOPMENT OF VOLUNTARY CARDIOVASCULAR CONTROL

A Dissertation

Presented to

the Graduate Council of

The University of Tennessee

In Partial Fulfillment

of the Requirements for the Degree

Doctor of Philosophy

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by

Roger A. Kleinman

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ABSTRACT

Several lines of evidence have suggested that the normally involuntary status of the autonomic nervous system is due to a lack of discriminable afferent information to the central nervous system. This proposition has been implicitly supported by many behavioral studies all of which provided extrinsic feedback of cardiovascular performance in an attempt to produce learned cardiovascular control.

In order to explicitly determine whether discrimination of afferent information from the heart facilitates subsequent learned heart rate control, therefore, the first experiment of this dissertation was performed. During the first phase of this experiment, human subjects were trained to discriminate their pulses, and during the second phase, these same subjects were required to increase and decrease their heart rates under conditions of augmented sensory feedback of heart rate, and under conditions when no extrinsic feedback was provided. The results of this experiment demonstrated that a significant improvement in heart rate control during the second phase developed as a function of previous pulse discrimination training. In addition, pulse discrimination training facilitated the development of learned heart rate control under conditions of augmented sensory feedback. These findings were taken to support the original hypothesis that training designed to facilitate discrimination of the internal afferent information associated with a visceral response would also facilitate the

development-learned control over that response. The results of this experiment together with other reports indicating that voluntary visceral control is more effective under conditions of extrinsic response-contingent feedback of visceral responding suggested the possibility that voluntary control might be developed over other aspects of cardiovascular functioning following the implementation of procedures which utilized augmented sensory feedback of the relevant response process.

The second experiment in this dissertation investigated the possibility of producing voluntary control over changes in systolic blood pressure. During two training sessions, two groups of subjects were instructed to increase or decrease their systolic blood pressure while receiving virtually continuous extrinsic information of blood pressure changes. Heart rate was continuously monitored in all subjects to determine the relationship between changes in this cardiac response and systolic blood pressure. The results of this study demonstrated that following the implementation of a simple operant procedure, reliable increases and decreases in systolic blood pressure may be obtained. Moreover, such changes developed according to an instructional requirement and not as a function of habituation to the experimental environment, or as a result of unconditioned stimulus effects. Although these blood pressure changes were produced in the absence of any systematic changes in heart rate, it was found that increases and decreases in blood pressure ware associated with greater

heart rate differences during certain periods in the development of learned blood pressure control than during other periods.

The implications of these findings were discussed with respect to the use of visceral response discrimination and operant training procedures in the therapeutic control of emotional and psychosomatic behavior patterns.

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CHAPTER I

INTRODUCTION

During the last ten years there has been an accumulation of experimental evidence demonstrating that autonomically-mediated responses are modifiable according to the law of effect. In general, these procedures have placed the cardiovascular response system in control of response contingent external stimulus events (For reviews: Katkin and Murray, 1968; Kimmel, 1967).

The Voluntary-Involuntary Distinction

Reports of instrumental autonomic conditioning can be viewed as opposing a two-factor approach to response acquisition whereby it is conceived that visceral responses can be experimentally controlled only according to the laws of classical conditioning (Kimble, 1961; Konorski and Miller, 1937; Skinner, 1938). The major issue in this controversy concerns what sort of response is being conditioned in studies which purport to demonstrate autonomic conditioning.

In 1954, Kendon Smith ("Conditioning as an Artifact") argued that although autonomic responses can be experimentally controlled following operant procedures, these responses may be reflexive artifacts of striate muscle responding, and occur only secondarily to an instrumentally conditioned somatic response. Essentially this argument describes a conceptual nervous system in which proprioceptive feedback

from the striate musculature produces reflexive visceral effects in either one of two ways: (1) direct neural connections may exist between striate muscles and visceral organs at a spinal level, or (2) afferent information from the striate musculature to central areas may result in efferent control of the visceral system.

For several reasons which are discussed in a later section, the arguments put forth in this somatic-mediation controversy are not adequately addressed to the question of whether autonomic responses can be conditioned. In view of the untenability of the somatic-mediation argument, a response which can be demonstrated to be under the control of response contingent reinforcement will be defined, here, as an autonomic operant, regardless of other observed behavioral correlates or antecedents.

Within the context of this dissertation, a "Voluntary Response" is defined as a response which is produced or inhibited following the occurrence of an appropriate verbal instruction. Thus if a subject raises his arm when he is told to, arm-raising qualifies as a voluntary response. Similarly, if a subject is told to decrease his heart rate and he does so, then this response would qualify as voluntary.

Although the operations which define a voluntary response are different from those defining an operant, it is argued here that the processes involved in the modification of these two classes of behavior are very similar. Specifically, both types of behavior can be influenced by response contingent stimulation (reinforcing stimulus).

The meaning of a "Voluntary Response" will be clarified by reference to the types of reinforcing stimuli used in operant conditioning procedures. Studies which have used animal subjects in an effort to produce autonomic operants have typically provided biologically significant reinforcing stimuli (intracranial electrical stimulation; shock) which were contingent upon the emission of the required response. Thus, Dicara and Miller (1968a) employed a shock avoidance design in which criterion heart rate changes in rats allowed the animal to escape or avoid electric shock. As a rule, biologically significant reinforcing events (food, shock) have not been used with human subjects. Humans will associate implicit value to the occurrence of a stimulus event if that event is defined as worthwhile or desirable by the experimenter. Operant procedures which conform to this latter scheme have been termed "Voluntary Control Procedures," and although some redundancy exists with the terms "Instrumental Control" or "Operant Control," the term "Voluntary" specifically refers, operationally, to a response which is inhibited or produced following the appropriate verbal instruction. The development of voluntary heart rate control was demonstrated by Hnatiow and Lang (1965) who taught subjects to reduce their heart rate variability by instructing them to stabilize a beam of light whose variability of movement was contingent upon the subjects' heart rate variability. "Decreasing-variability" of a beam of light had acquired reinforcing properties only after the experimenters instructed subjects as to its significance. As a consequence, the variability of the visual

display acquired control over the emission of decreases in heart rate variability.

Feedback and Voluntary Control

The importance of informational feedback of effector responding for the acquisition and maintenance of voluntary control over that effector was demonstrated as early as 1895 by Mott and Sherrington. These investigators found that when single limbs in monkeys were deafferented, voluntary control over the limb was lost. Clinical demonstrations, however, have provided evidence that afferent information from a particular effector organ is not required in order to maintain voluntary control over that organ; sensory feedback via other modalities may be sufficient. Individuals suffering from tabes dorsalis, a disease of the dorsal spinal column which interrupts striate afferentation, cannot maintain an erect standing posture without visual feedback of their position in space. Although some evidence suggests that the loss of voluntary control in this case is due to hyperactivity of the deafferented limb and not directly a function of the lack of striate proprioception (Taub and Berman, 1968), afferent information via the visual mode was nevertheless necessary to regain voluntary effector control. Thus, it appears that the maintenance of voluntary control over an effector organ depends less on the channel by which afferent information is delivered to the central nervous system (CNS), and more upon the informational content of the feedback per se.

Evidence to be presented below supports the proposition that the normally involuntary status of the autonomic nervous system is due to a lack of discriminable afferent information to the CNS from autonomic effectors. Within this framework, the heart is viewed functionally as a deafferented striate muscle. This proposition has been implicitly supported by the various studies, to be discussed, all of which utilized extrinsic feedback of cardiovascular performance in an effort to develop cardiovascular operants.

The Purpose of this Dissertation

The purpose of the experiments reported in this dissertation, therefore, will be first, to determine whether intensive training designed to facilitate the discrimination of afferent information from the heart enhances the subsequent development of cardiac rate control. Secondly, to determine whether operant procedures which incorporate augmented sensory feedback of cardiovascular performance can result in the acquisition of voluntary control over systolic blood pressure changes.

A description of these investigations will be preceded, however, by an account of the problems which have been raised with respect to what constitutes a demonstration of autonomic conditioning. Secondly, the train of logic will be explored which underlies the proposition that the normally involuntary status of the autonomic nervous system is a consequence of a lack of discriminable afferent information from

visceral response processes, and not merely due to afferent or efferent insufficiency. These discussions constitute the following two sections respectively.

The Somatic-Mediation Problem

The criticism made by Smith (1954) that experimentally controlled autonomic responses were in reality reflexive artifacts of conditioned striate responding was originally predicated upon the observation that somatic acts may provide the necessary antecedent conditions for the performance of the autonomic response. Smith contended, as an example, that increases in muscular tension could have, and probably did, reflexively initiate the increments in heart rate, galvanic skin responding, salivation, and other changes in autonomic responding which various investigators claimed to have conditioned. In order to determine the validity of Smith's criticism, and ascertain the role which somatic response processes play in autonomic learning, the somatic component would perforce have to be either systematically varied or eliminated entirely.

Systematic variation of the somatic component. Sinus arrhythmias are known to be associated with normal respiratory cycles, and cardiac accelerations and decelerations occur respectively during inspirations and expirations (Westcott and Huttenlocher, 1961). The following studies attempted to determine whether respiratory changes constituted a necessary antecedent for the establishment of a conditioned cardiac response.

Wood and Obrist (1964) investigated a classically conditioned heart rate response in humans under conditions of controlled and uncontrolled respiration. These investigators noted a biphasic conditioned heart rate response, characterized by an initial acceleration following CS presentation and a secondary deceleratory phase. Increases in respiratory activity were directly related to the cardiac acceleration during the first phase of the conditioned cardiac response. In a second procedure, respiration amplitude and frequency were controlled, and the acceleratory phase was not observed. Conditioned cardiac rate decelerations, however, were obtained.

Using conditions of unpaced and paced respiration, Brener and Hothersall (1967) taught human subjects to successfully increase and decrease their heart rates when presented with augmented sensory feedback of their cardiac performance regardless of whether unpaced or paced respiratory behavior was specified.

Despite the relationship which exists between respiratory acts and heart rate, the results of these two studies have demonstrated that respiratory changes do not provide a necessary condition for the development of a conditioned heart rate response. It might be argued that other somatic muscle groups could have been conditioned and perhaps mediated the observed heart rate changes. The logic of this argument, however, proceeds ad infinitum and ad nauseam, thwarting efforts to critically evaluate the results of autonomic conditioning studies which systematically manipulate particular somatic responses.

Elimination of the somatic component. The experimental response to this logical problem has involved the technique of curarization which effectively blocks neural transmission at the motor end plate and produces a state of flaccid paralysis in the somatic musculature. Classical conditioning of heart rate changes has been successfully produced when animal subjects were curarized with d-tubocurarine (Black, Carlson, and Solomon, 1962; Solomon and Turner, 1962). Following the publication of these findings, it was argued by Smith (1964) that in light of the large amount of evidence favoring a motor theory of learning, it would appear that even radical curarization does not abolish skeletal responding. In response, Black and Lang (1964) recorded electromygraphic (EMG) activity and produced reliable classically conditioned cardiac rate increases in curarized dogs in the absence of any EMG responding.

A series of studies (Dicara and Miller, 1968a; Hothersall and Brener, 1969; Miller and Dicara, 1967; Miller and Banuazizi, 1968; Trowill, 1967) have successfully demonstrated operantly conditioned heart rate changes in curarized animals. Both a reward (intracranial electrical stimulation, ICS) and a shock avoidance paradigm have been used effectively. Black (1966) had suggested that although striate paralysis followed curarization, the heart rate response might be mediated merely by conditioned activity of the motor cortex. In response, Miller and Banuazizi using ICS contingent upon systematic changes in heart rate or spontaneous intestinal contractions, operantly conditioned one response mode in the apparent absence of systematic

changes in the other. The authors cited these results as evidence for two independent response systems, striate and visceral, since if Black's criticism was valid, conditioned activity of the motor cortex would have similarly affected both autonomic response modes. Their interpretation is somewhat tenuous since it is evident that somatic activity is accompanied by a need for an increased blood flow to, and consequent vasodilation of those blood vessels which supply the striate muscles. The adaptive needs of the organism, therefore, dictate that increased somatic activity would result in a dissimilar responding of the cardiovascular and gastrointestinal systems.

It must be emphasized here that curare blocks neural transmission at the myoneural junction, and although response-produced feedback from striate musculature is effectively blocked, the use of curare does not exclude the possibility first, of peripheral feedback proximal to the myoneural junction, or secondly that a central structure (functionally defined) may control both response processes simultaneously. The results of studies which show autonomic conditioning in curarized subjects do not necessarily support the concept of two response systems controlled independently by two central structures. The "efferent nervous system" has been dichotomized according to the degree to which an organism can discriminate afferent feedback from or control those responses mediated by autonomic or somatic mediators. Such a dual classification gains little support from anatomical, physiological, as well as psychological evidence (Rushmer, 1961).

Somatic-Autonomic Coupling

The proposition that a central area controls both somatically and autonomically-mediated responses in a parallel fashion has acquired considerable experimental support. A high correlation between somatic activity and cardiac rate has been obtained in noncurarized and partially curarized dogs (Black, 1965, 1967; Obrist and Webb, 1967) and humans (Obrist and Webb, 1967; Westcott and Huttenlocher, 1961). Black (1965) found that during recovery from flaccid paralysis, the striate component of a previously conditioned heart rate response under curare gradually appeared. Obrist and Webb observed that anticipatory cardiac responses to aversive stimulation produced decelerations in humans, but accelerations in dogs. Directly associated with these changes in cardiac behavior, however, were regular changes in EMG activity. Cardiac accelerations were associated with increases in EMG activity, while decelerations correlated with decreases in EMG activity. It thus appeared that cardiac behavior was not associated with emotion (shock anticipation) as much as motion (somatic activity). In other words, changes in heart rate were predicted by somatic activity and not by the motivational antecedents of the cardiac response. Because increases in heart rate were proportional to skeletal muscle activity, these investigators concluded that heart rate can be used as an index of skeletal muscle activity, both response processes being controlled largely by the same central neural structures. In light of the findings by Westcott and Huttenlocher, Wood and Obrist, particularly the Black (1965)

and Obrist and Webb results, and in consideration of the fact that autonomic conditioning can proceed in the absence of peripheral somatic-mediation (curare studies), the proposition that somatic and autonomic response processes are coupled at a central as well as a peripheral level seems highly tenable.

This proposition has been experimentally investigated by Brener and Goesling (1968) who hypothesized that if the cardiovascular and skeletal response are merely different aspects of the same response process, then if one response is instrumentally conditioned, the probability of the second response occurring under the same contingencies as the first would be greater than if the contingency for the first response had not gone into effect. An active-passive shock avoidance design with rats was used. During active (wheel turning) and passive (immobilization) training, significant increments and decrements respectively in heart rate were obtained. When avoidance training was completed, animals were curarized and their heart rates were placed in control of the reinforcing stimulus (shock). Although conditioning procedures under curare did not result in <u>differential</u> heart rate changes, previous somatic learning determined heart rate performance during cardiac conditioning. In other words, despite the inability of the curarized animals to emit skeletal responses, the heart rate changes which had accompanied skeletal learning continued to exert control over the reinforcing stimulus during heart rate conditioning. Thus the striate and the cardiovascular response can be appreciated as functionally

integrated components of what Smith (1967) called the entire "somatic-autonomic Gestalt."

Aside from the fact that anatomical, physiological, and psychological evidence belies the existence of two independently functioning response systems, the virtual untestibility of such a notion must also be emphasized. Experimental support for two independent response systems would derive from evidence which demonstrated that a central neural structure which mediated the development and maintenance of a visceral operant was not necessary for the development and maintenance of a striate operant.

The controversy concerning somatic-mediation has been discussed briefly because its elucidation is a necessary requirement for the description of what constitutes an autonomic operant. It has been argued that autonomic and somatic responses are centrally as well as peripherally coupled, constituting different components of the same response system. Within this context, one response cannot be considered to take precedence over or reflexively initiate the other. Consequently, a cardiovascular response which, after the implementation of operant procedures, acquires reliable control over the occurrence of a response-contingent external stimulus event will be defined as a cardiovascular operant irrespective of other observed behavioral correlates or antecedents.

The task remaining then, is to define the experimental procedures which facilitate the development and maintenance of a cardiovascular

operant. Toward this end, the concept of informational feedback of cardiovascular performance is of central importance.

The Development of Voluntary Effector Control

The development and maintenance of a voluntary response requires that information concerning the various dimensions or aspects of the response, such as magnitude, extent, form, or topography, be fed back to a central system which integrates the information and modifies the response characteristics to conform with the original instructional demands. Ample proprioception is normally made available from the striate musculature to permit the CNS to evaluate and correct motor acts.

The importance of proprioceptive feedback to voluntary effector control, however, has been questioned by Taub and Berman (1963) and Taub, Bacon, and Berman (1965), who trained monkeys with deafferented forelimbs (dorsal root section) to emit a forelimb flexion response to avoid shock. Although visual cues were eliminated all subjects learned the avoidance response. In the free situation, however, coordinated and adaptive responses such as grasping and other digital manipulations were ". . . abortive, tending to be jerky, inaccurately directive and short in excursion," (Taub and Berman, 1963, p. 1014). In fact, by the end of the first postoperative week, the deafferented limbs were held almost immobile. In another study (Taub and Berman, 1964) monkeys had both forelimbs deafferented. In the free situation during the two post-operative weeks, the limbs remained virtually unused. Maximal recovery

required from two to six months and was characterized by the ability to use the limbs in slow and moderately rapid body movement. Fine digital manipulations recovered to the extent that, "Several monkeys were even able to pick up raisins between the thumb and forefinger from a shallow well," (Taub and Berman in S. J. Freedman, 1968, p. 177). It is clear from the descriptions of the recovery of muscular coordination made in these studies that deafferentation resulted in moderate to serious impairment in coordinated motor functioning. Totally abolishing peripheral afferentation (spinal cord deafferentation, section of the right vagus, and procaine block of the left vagus) did not prevent an animal from emitting a previously learned avoidance response. It was concluded from this and other experiments that there is considerable autonomy between the CNS and the periphery. Once a response pattern is programmed into the CNS, the behavior can be maintained without any afferentation from the periphery. The authors suggested that purely central feedback mechanisms which are established during peripheral functioning could maintain instrumental behavior subsequent to deafferentation. These central feedback loops would provide neural information concerning the activity of the motor cortex back to the sensory cortical areas before motor impulses reach the periphery. As will be described in a later section, Chang (1955) and Kuypers (1960) have demonstrated such central mechanisms utilizing electrophysiological and neural degeneration techniques.

In none of the studies reported by Taub and Berman (1968) did the totally deafferented animal learn an instrumental response. At least two channels of feedback information to the CNS must be associated for such learning to occur; information concerning the characteristics of the response itself, and information about the effects of the response. The former afferentation may be topographic (proprioception from relevant muscle groups) or nontopographic (indirect afferentation of a muscle contraction such as kinesthetic feedback from the nondeafferented joints and subcutaneous tissue; afferent information of body position from the organs of equilibration within the middle ear; vision; or perhaps extrinsic feedback of deafferented muscle contractions, such as the click of a feeder mechanism which is contingent upon the animal emitting the required response). Animals with both forelimbs deafferented did learn an avoidance response when blindfolded, but these two types of feedback information were probably available to these monkeys. First, neural feedback of response characteristics could have occurred centrally since the animals had ample experience with the required response (forelimb flexion) and could have established central associations with the topographic feedback information available prior to deafferentation. In addition, the deafferented animals had access to free play situations where several forms of nontopographic feedback were available (vision, audition, equilibration). Secondly, it could be argued that information concerning the effect of the response (shock avoidance) was also available. This feedback would have been represented

by the nonoccurrence of the aversive stimulus. The animal which was subjected to total peripheral deafferentation performed an avoidance response, but this response was previously learned when only forelimbs were deafferented. No reports were made of response acquisition under conditions of total peripheral deafferentation.

Taken together the Taub and Berman papers suggest first, that following deafferentation and recovery, instrumental responding may be exhibited, albeit moderately to seriously impaired. Secondly, feedback to the CNS concerning the effects of an instrumental response must be available from the periphery for learning to occur. Thirdly, subsequent to complete deafferentation, previously learned instrumental responses may be displayed, but central feedback mechanisms are necessary.

Autonomic effectors have been generally thought to provide no feedback at all, or feedback too diffuse to relay any precise information concerning autonomic functioning to the CNS. According to this opinion the CNS cannot evaluate and modify autonomic responding, and voluntary control over visceral activity is normally not possible. Many physiological studies, particularly the Russian literature on interoception, have demonstrated that neural afferentation normally exists between visceral organs and the CNS. Exploring the relationship between interoceptive reflexes and the cortex, I. I. Kaplan (Chernigovskiy, 1967, p. 605) created a dominant focus of excitation in the motor area by applying phenol to the anterior columns of the spinal cord. Stimulation of interoceptors in the bladder before phenol

application produced no somatic muscular activity, whereas following treatment with phenol, interoceptive stimulation evoked gross somatic activity. O. S. Merkulova (Chernigovskiy, 1967, p. 610) applied a strychnine solution to the motor and premotor cortical areas in the cat and found a lowered threshold for skeletal responding to interoceptive stimulation following treatment with strychnine. Washing or undercutting the cortex removed the effects of the drug. Ukhtomskiy (Chernigovskiy, 1967, p. 581), in a series of experiments concerned with alimentary functions, showed that contraction of antagonistic muscles of the lower limb produced by stimulation of the motor cortex are inhibited when swallowing or defecation accompanies motor cortex stimulation. The experiments just cited have not clearly specified whether visceral afferents affect the cortex directly or, in a reflexive fashion, elicit somatic changes at the spinal level. They have, however, demonstrated that afferent information from the viscera is normally made available to the CNS, and that changes in visceral activity can produce changes in striate responding. These and other findings have led Chernigovskiy to conclude that ". . . such a mobile and corticalized function as the function of the motor apparatus does not remain indifferent to the activity of internal organs. Moreover, stimulation of the interoceptors may bring about rather diversified changes in activity of skeletal musculature," (p. 619).

Although the Russian studies have not indicated whether visceral afferentation affects somatic activity by acting on the cortex, or

reflexively at the spinal level, it should be noted that almost all interoceptive information, even that concerning many spinal reflexes, and particularly of the vital functions (cardiovascular, respiratory, etc.), is made available via the sensory spinal tracts to at least the supraspinal sensory nuclei. Several studies which incorporated electrophysiological and neural degeneration techniques have determined that the cortex receives fibers from the supraspinal sensory nuclei and in turn provides neural information over corticofugal pathways to the subcortical regulatory areas associated with visceral functioning. The electrophysiological evidence of Kaada (1960) and Lofving (1961) indicated that the cortex does exert a controlling influence (inhibitory) over cardiovascular function. Chang (1955), using electrophysiological techniques and Kuypers (1960) demonstrated the existence of neural connections between the motor cortex and subcortical areas via afferent collaterals from cortical pyramidal fibers. Kuypers lesioned various cortical areas in the Rhesus monkey, and observed the degeneration of corticospinal fibers. Neural degenerations demonstrated the existence of projections from the precentral (motor) gyrus to the lateral reticular nucleus of the medulla oblongata and the pontine and medullary tegmentum. Projections of the precentral, and postcentral (somato-sensory) areas synapsed with the nucleus cuneatus, gracilis, and main sensory nucleus of the trigeminus. It was also found that the same cortical areas may mediate both sensory and motor functions, and it was suggested that corticofugal fibers synapsing with sensory nuclei

may represent sensory "feedback" mechanisms. Of particular importance here are the connections which the motor and sensory-motor cortex make with the medulla oblongata, since the areas which regulate cardiovascular functioning are located in this part of the brain stem.

Taken together, these several studies support a physiological model whereby afferent information concerning visceral activity is made available to the cortex where it can be evaluated and utilized to exert corticofugal control over those subcortical areas which regulate visceral functioning.

The empirical fact is, however, that the autonomic nervous system does not normally or usually function as a voluntary response system. In light of the physiological evidence to the contrary, it cannot be said that the normally involuntary status of the autonomic nervous system is due to a lack of visceral afferentation to the higher brain areas. Rather, the distinct possibility arises that the existing neural feedback to the CNS is not sufficiently discriminable. Furthermore, it appears appropriate to suggest that if the discriminability of this feedback information could be enhanced, the implementation of appropriate operant procedures might result in the development of voluntary control over autonomic effectors.

In summary, it has been proposed that, whether peripheral or central, feedback information concerning the characteristics and effect of an emitted response is necessary for the acquisition and maintenance of that response. The fact that autonomic responses do not usually

operate according to the law of effect is not due so much to an insufficiency of afferent information by which the CNS can evaluate response effects, but to an inability to effectively discriminate the afferentation which is normally made available to the higher brain areas. Neural fibers from the cortex extend to subcortical areas which regulate visceral responding as participants in central somatosensory feedback systems. Thus, autonomic afferent and efferent systems exist, completing the anatomical requirements for the evaluative and control functions which the CNS can perform on the visceral response system.

The demonstration that visual feedback of bodily orientation is sufficient to permit coordinated extensor activity following functional deafferentation (tabes dorsalis) suggests that exteroceptive feedback may perform a similar function by providing the necessary information over an external route which permits behavioral control. What is also suggested by this demonstration is that in the autonomic nervous system where afferentation is normally made available to the cortex but cannot be adequately discriminated, the use of extrinsic feedback of visceral responding might enhance this discriminability and thereby facilitate the development of learned visceral control. Several behavioral studies, which attempted to instrumentally condition cardiovascular changes support this proposition. Hnatiow and Lang (1965) and Lang, Sroufe, and Hastings (1967) presented subjects with extrinsic (visual) feedback of heart rate variability and succeeded in conditioning decreases in the variability of subjects' heart rates. Providing subjects with

visual feedback of heart rate performance on a beat-to-beat basis, Engel and Hansen (1966) and Engel and Chism (1967) instrumentally conditioned cardiac decelerations and accelerations respectively. In these studies a monetary reward was also made contingent upon the appropriate heart rate response. Brener and Hothersall (1966, 1967) demonstrated the development of voluntary control over heart rate increments and decrements when augmented sensory feedback (auditory) was presented to their subjects on a beat-to-beat basis. Relevant to this discussion was the finding of Harwood (1962) who rewarded cardiac decelerations with a sum of money, but failed to adequately demonstrate operant conditioning. Since subjects were not given reinforcement on a beat-to-beat basis, the emission of incorrect responses (short inter-heartbeat intervals) might have been rewarded. In other words, subjects were not provided with response contingent feedback of cardiac rate performance and failed to develop operant control. Snyder and Nobel (1968) provided subjects with extrinsic feedback of spontaneous changes in finger blood volume and obtained clear evidence of instrumental conditioning.

Although all of these studies have argued implicitly for the necessity of employing extrinsic feedback information in order to facilitate the identification of those internal cues associated with cardiovascular responding thereby facilitating learned cardiovascular control, none have directly assessed this proposition. Relevant to this issue was the finding by Lisina (1961) that subjects learned to instrumentally control increases in finger volume only when they were

provided with visual (plethysmographic) feedback of blood volume changes. Brener, Kleinman, and Goesling (1969) endeavored to assess the degree to which augmented sensory feedback is needed for the development of voluntary control of cardiac rate. If such feedback information were facilitatory, then increased exposure to augmented sensory feedback information of cardiac rate during training should bear a positive relationship to the degree of voluntary heart rate control. In this experiment subjects were instructed to increase their heart rate to a light on their right and decrease their heart rate to a light on their left. Augmented sensory feedback consisted of high-pitched tones which were presented to the right ear (via headphones) contingent on short IBI's (high heart rate) and low-pitched tones which were delivered to the left ear contingent upon long IBI's (low heart rate). A criterion which defined short and long IBI's was individually chosen for each person. Each subject was run for two sessions separated by about 72 hours and on each session received nine trial pairs. A trial pair was defined as a right-light, left-light trial sequence, each individual trial lasting for a time period which was required for the subject to emit 50 IBI's. The subjects were divided into three experimental groups each receiving a different sequence of feedback or no-feedback trial pairs. The second, sixth and ninth trial pairs were test trial pairs during which no feedback was presented. Each of the three experimental groups was characterized by a different percentage of non-test trial pairs during which feedback was made available to subjects. The

100 percent group received feedback on all nontest trial pairs, the 50 percent group on only half of the nontest trial pairs, and the 0 percent group were presented with no feedback at all. With the aid of a histogram compiler, IBI distributions were collected during every test trial for each subject, and the median IBI of each distribution was used as the descriptive measure of heart rate performance during each trial. A measure of heart rate control for each test trial pair was obtained in the following manner: For every right-light, left-light trial pair, the median IBI for the right-light trial was subtracted from the median IBI of the left-light trial. Since long IBI's were required on the left-light trials, and short IBI's were required on right-light trials, large positive intermedian differences indicated good heart rate control, whereas small intermedian differences indicated poor heart rate control.

The mean intermedian IBI differences for each group of subjects on Sessions I and II are presented in Figure 1. Three results will especially be noted. First, all groups on both sessions show positive mean intermedian IBI difference scores, indicating that heart rate performance changed in the direction that is predicted by the law of effect. Second, heart rate control improved from Session I to Session II. Thirdly, on Session II, the degree of heart rate control observed was a direct function of the amount of exposure to augmented sensory feedback during training.

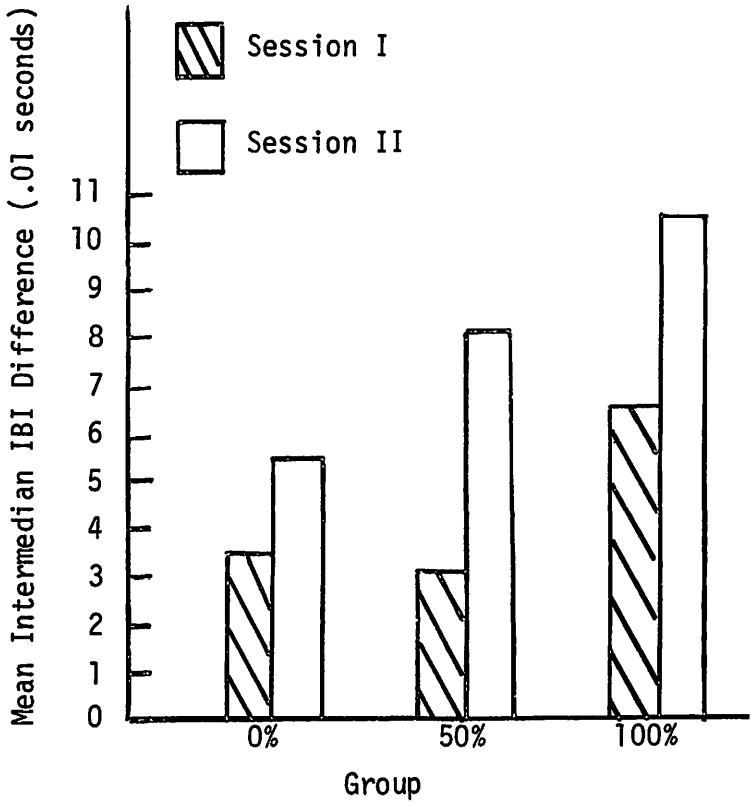

Figure 1. Mean intermedian difference scores for 100%, 50%, and 0% feedback groups on Session I and Session II.

An analysis of variance, performed on the mean intermedian IBI's during test trials revealed a nonsignificant groups effect. This result could have been caused by one of two principal factors. First, augmented sensory feedback of heart rate performance might not have had a facilitatory effect on the development of heart rate control. Secondly, the facilitatory effects of extrinsic feedback were being obscured by individual subject differences within each group, or by experimental error which was too great to permit a significant contribution to between subject variability by the differential treatment procedures.

The results of other studies which have utilized extrinsic feedback toward the development of learned cardiovascular control support the latter position. As will be recalled, Lisina (1961) found that subjects learned to produce increases in finger blood volume only when they were provided with plethysmographic feedback of changes in finger volume. Hnatiow and Lang (1965) reported that standard deviations of subjects' IBI distributions were significantly smaller during feedback than during nonfeedback training periods. It was observed by Brener et al. (1969, Experiment 1) that larger conditioned heart rate changes are acquired when training is carried out under conditions of augmented sensory feedback than when no extrinsic feedback is made available. As was mentioned earlier, none of these studies have specifically investigated the degree to which artificial feedback affects the development of learned cardiovascular control. The fact that cardiovascular performance was consistently superior in these studies during feedback

conditions, however, strongly supports a facilitatory role for extrinsic feedback in the production of learned autonomic responding. In view of the evidence which recommends the utility of augmented sensory feedback in the establishment of cardiovascular control, it is difficult to attribute to chance effects, the direct relationship observed in the Brener et al. experiment between amount of exposure to feedback and the degree to which subjects learned to control their cardiac rates. Rather, it is suggested that facilitatory effects were obscured by a large within group variability and that sampling a greater number of subjects would have reduced this error according to the random error theory of measurement.

A significant improvement in cardiac rate control was observed from Session I to Session II. In addition, it was noted (Figure 1, page 24) that all groups displayed heart rate control in the predicted direction during the first session. These results demonstrate and support other findings (Brener and Hothersall, 1966, 1967; Hnatiow and Lang, 1965; Lang, Sroufe and Hastings, 1967) that following the implementation of appropriate operant procedures, individuals can rapidly gain voluntary control over cardiac behavior. Furthermore, it was demonstrated that the ability to voluntarily control cardiac behavior can be retained and significantly improved as a function of further participation in an operant training program.

In light of the several studies just cited which made extensive use of extrinsic feedback of cardiovascular behavior in the development

of learned cardiovascular control, and because all have implicitly supported the discriminative function of augmented sensory feedback toward this end, the question arose as to whether intensive training designed to promote discrimination of afferent information from the heart would facilitate the development of subsequent learned heart rate control. In order to answer this question the following experiment was performed.

CHAPTER II

EXPERIMENT I: PULSE DISCRIMINATION AND THE DEVELOPMENT OF LEARNED HEART RATE CONTROL

I. METHOD

Subjects

The subjects were 21 undergraduates (8 females, 13 males) who reported no personal histories of cardiovascular disease.

Apparatus

The basic pulse discrimination procedure employed in this study required the subject to press a microswitch button every time he felt his heart beat. The level of pulse discrimination was assessed by examining the distribution of latencies from heart beats to button presses.

Logic modules (BRS) were programmed so as to record the time interval occurring between each button press and the heartbeat which immediately preceded it. These latencies were recorded in 0.18 second interval categories. Thus if a button press occurred within 0.18 seconds of a heartbeat, a one was added to the total of the first category counter. If the button press followed the heartbeat with a latency of >0.18 <0.36 seconds, a one was added to the second category. Latencies of >0.36 <0.54 seconds, >0.54 <0.72 seconds, >0.72 <0.90 seconds were recorded on the third, fourth, and fifth counters respectively. Latencies of longer than 0.90 seconds were not recorded.

Electromagnetic counters also recorded the frequency of various sequences of button presses and heartbeats. One counter counted the number of heartbeats which followed a heartbeat, another counter counted the number of button presses which followed a heartbeat (correct response) and a third counter counted the number of button presses which followed a button press. Finally a Hunter decade timer was employed to time the period it took for subject to emit 51 heartbeats (50 inter-heartbeat intervals). An accurate measure of heart rate was thus obtained.

Procedure

Pulse discrimination training. The Experimental group (4 females, 3 males) was run for two sessions (Session I and II) separated by about one week; a session consisted of 28 trials lasting for the time it took subject to emit 50 IBI's. The 28 trials were divided into 7 blocks of 4 trials each. Each block was defined by 3 no-feedback trials (identified to the subject by the onset of a small green light) followed by one trial (identified to the subject by the onset of a small amber light) on which discreet feedback (1130 Hz tones) of heartbeats were presented to subjects via earphones.

Subjects were ushered into a sound-deadened cubicle (7' × 8-3/4' × 4-3/4') and asked to sit in a reclining chair. They were told that the experiment was designed to investigate certain internal, physiological responses and that some small recording devices (EKG

electrodes) would be attached to the surface of their skin. All subjects were assured that no shock or other aversive stimulation would be employed (actual instructions appear in Appendix, Note 1). They were shown how to hold the microswitch with their left hand and to press the lever with the thumb. Subjects held the switch in their left hand since one of the EKG placements was on the right wrist (lead II) and pressing the lever with the right hand often introduced electrical and movement artifact into the EKG records. Experimental subjects were then informed that when the green light (no feedback) came on they were to press the "button" in their hand each time they thought they felt their heart beat. In addition, they were instructed not to wait until they were absolutely sure they felt a heartbeat before pressing the button, but to do so whenever they were fairly certain. Experimental subjects were instructed as to the significance of the tones which were presented to them on amber light trials. During subsequent green light periods they were told to press the button whenever they experienced any internal feelings or sensations which previously were discriminated during the amber light period when tones occurred.

The measure of pulse discrimination employed was based on the distribution of heartbeat-button press latencies. It was assumed that if the subject was not discriminating his heartbeats, his button presses would be rectangularly (equally) distributed in time with respect to his heartbeats. In other words, each button press would have an equal chance of falling into any of the 0.18 second latency categories

(within the constraints set by the IBI distribution). If however, the latency distribution peaked in one of the latency categories, this mode would indicate that the subject's button presses were being timed with respect to the occurrence of his heartbeats. The degree of pulse discrimination displayed by subjects was measured by calculating the percentage of button presses above chance which appeared in the modal category. The rationale underlying this modality discrimination index is based upon the assumption that if a subject is not discriminating heartbeats, the inter-button press and IBI distributions will be independent, and consequently any inter-button press interval will have an equal chance of being associated with any IBI. In order to empirically investigate this assumption, two control groups were run. The first of these groups (Regular Control) heard tones (1130 Hz) at a regular rate of 72 presentations per minute. The second group (Irregular Control) heard an irregular sequence of tones (1130 Hz) which was repeated at 6.4 second intervals. Both groups were instructed to press the button in synchrony with these tone sequences on green light (no-tone) trials. Their task then, was to match the rate of the tones heard on amber light (tone) trials.

Heartbeat-button press latencies were recorded for both these groups as well as for the Experimental group which heard tones contingent on their heartbeats.

It will be recognized that the chances of obtaining a completely rectangular distribution of latencies is very remote. The control

groups, therefore, served to empirically establish the modality scores that would be obtained by chance.

Preliminary Considerations Concerning the Methods By Which the Index of Pulse Discrimination Was Obtained

In order to continuously record the latencies between heartbeats and button presses, the category time base was automatically reset each time a heartbeat occurred. A relatively short IBI, therefore, might have resulted in the resetting of the timer mechanism before the time base had reached the upper limit of a particular category. Consequently the probability of a button press falling in that category was less than for each of the preceding categories. In the absence of pulse discrimination, the assumption that heartbeat-button press latencies will be rectangularly distributed over the latency categories holds only for those categories which have an equal chance that button presses will fall into them. Therefore, to include the number of button presses within a certain category in statistical calculations, the shortest IBI produced during each trial had to be longer than the upper limit of that category. In order to make this determination, information concerning the variability of the IBI distributions during each trial was required. Although IBI distribution variability was not measured in the present experiment, normative data were available from a study performed by Brener (1964) in which it was also indicated that IBI variability fluctuated in a systematic fashion with heart rate. Thus only those variability scores were chosen from subjects in the Brener

study whose mean IBI's equaled the mean IBI's for subjects in the present experiment. The average standard deviation thus calculated was 0.058 seconds. Three standard deviations or 0.174 seconds below the mean was taken as a reasonable lower limit to the IBI distributions obtained in the present experiment on each trial. Thus, in order to include a particular category the mean of the IBI distribution for that trial had to exceed the upper limit of the category by 0.17 seconds. This mean IBI value was obtained directly by dividing the duration of each trial (in seconds) by 50 (IBI's per trial). Thus category five was rejected if the trial time was less than 53.50 seconds, category four if less than 44.50 seconds, and category three if less than 35.50 seconds. If, during any trial, the category to be dropped contained more button presses than the categories retained, either the variability of the IBI distribution on that trial was smaller than 0.58 seconds, or a relatively larger number of heartbeat-button press latencies fell into that category, or both, and it was deemed inappropriate to drop that category.

The specific measure of pulse discrimination was calculated by summing the number of button presses in each accepted category over the 3 no-feedback trials which constituted each block, and then obtaining the percentage of presses above chance which appeared in the modal category for that particular block. Thus 7 discrimination scores were calculated for each subject on each session.

It should be noted that the procedure of summing the number of button presses in each category over the 3 trials constituting each block served two important functions. First, the probability of obtaining a perfectly rectangular distribution on each trial is small, and indeed zero if the chance expectation is not an integral value. A mode will probably appear on any given trial, and if percentage above chance scores were computed separately for every trial and then averaged, the resultant value would have probably been unduly inflated. Second, if pulses were not being discriminated, these trial modal values, varying from one category to another over trials, would be effectively obscured by summing the number of button presses over a few trials.

In summary, the procedures outlined above were designed to assess the ability of subjects to discriminate their heartbeats while at the same time controlling for the effects of individual differences in heart rate and heart rate variability.

Heart rate control training. About one week following Session II the Experimental and the Irregular Control group underwent one session of heart rate control training (Session III). Session III consisted of 8 trials when subjects were instructed to increase their heart rate, and 8 trials when a decrease was required. Increase trials were identified by the onset of a light to the right of the subject, and decrease trials by the onset of a light to their left. The 16 training trials were divided into 4 blocks of 4 trials each. On two

blocks extrinsic feedback of heart rate was made available via headphones to subjects (Feedback Period); the remaining blocks provided no feedback (No-Feedback Period). Feedback and No-Feedback periods alternated and were partially counterbalanced. In each group, four subjects were presented with a No-Feedback period first, and three subjects were first presented with a Feedback period. Right-light (increase) and left-light (decrease) trials were alternated within each period.

Before training was begun, several 50 IBI heart rate samples were taken (until heart rate stabilized) and an IBI criterion was chosen such that about half of the number of IBI's recorded during each sample were shorter and half were longer than the criterion. During training, if less than twenty IBI's accumulated in the short IBI category during two successive right-light (or less than twenty IBI's in the long IBI category on two successive left-light trials) the criterion was lengthened (or shortened) by 0.02 seconds so that the IBI criterion could be achieved more easily by the subject. When feedback tones were made available to subjects, high heart rates (short IBI's) were accompanied by high-pitched tones (1685 Hz) in the right ear; low heart rates (long IBI's) by low-pitched tones (1130 Hz) in the left ear. All tones were contingent upon heartbeats.

Subjects were ushered into the experimental cubicle, and seated; the EKG electrodes were then attached and the nature of the experiment was explained. Subjects were instructed to increase their heart rates on right-light trials and decrease their heart rates when the light on

their left appeared. They were also informed that on some trials they would hear tones through the headphones and were instructed as to their significance. Subjects were asked to produce high-pitched tones on right light feedback trials and low-pitched tones during left-light trials. They were, finally, instructed not to engage in any abnormal breathing and to refrain from bodily movement or other maneuvers involving muscular tension in their efforts to control their heart rates.

II. RESULTS

Pulse Discrimination Data

The mean discrimination scores for each session for each subject were submitted to analysis of variance. The results of this analysis are described in Table 1. It will be seen that a significant groups effect was obtained ($F = 8.703$, $df = 2/18$, $p < .01$) indicating that the discrimination scores were significantly higher for the Experimental group. This is interpreted as evidence that the discrimination procedure led to heartbeat discrimination in the Experimental group. Further evidence in support of this contention is contained in the significant groups by sessions interaction ($F = 4.231$, $df = 2/18$, $p < .05$). As may be seen from Figure 2 the Experimental group alone displays an increase in discrimination as a function of sessions. Thus the discrimination procedure may be seen to lead to an improvement in heartbeat discrimination as a function of training.

Table 1

Summary of the Analysis of Variance on the mean percent above chance pulse discrimination scores for each session for each subject.

Source	df	Mean Square	F
Between Subjects	20		
Groups (A)	2	.0322	8.703**
Subjects within groups	18	.0037	
Within Subjects	21		
Sessions (B)	1	.0029	2.231
A × B	2	.0055	4.231*
B × subjects within groups	18	.0013	

* = $p < .05$ ** = $p < .01$

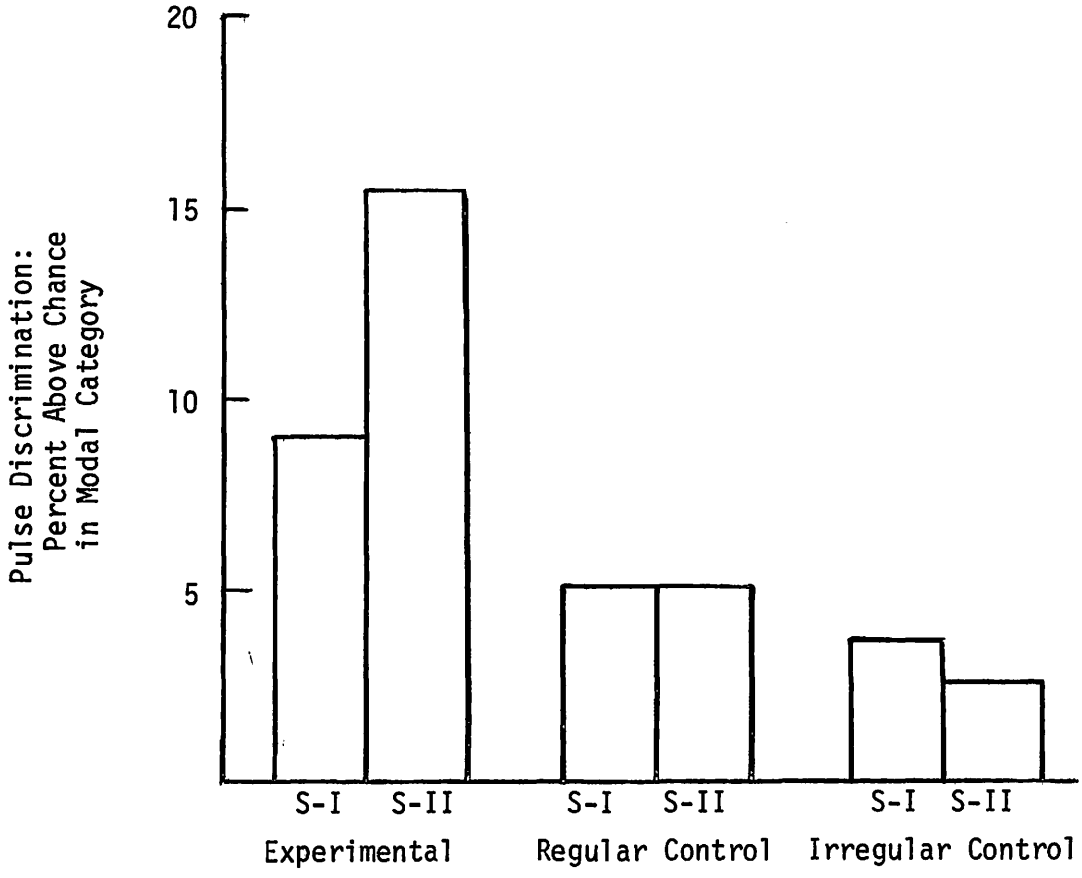

Figure 2. Percent above chance pulse discrimination on Session I (S-I) and Session II (S-II) for the Experimental and two Control Groups.

A Duncan's Multiple Range Test was applied to the means of the three groups in order to determine differences in the mean performance of the Experimental and two Control groups. It was found that the Experimental group which had a mean discrimination score of 12.28 percent above chance differed significantly from the Regular Control group (5.05 percent above chance) and the Irregular Control group (3.21 percent), $p < .005$, $S_{\bar{x}} = 1.62$ percent. The means of the two control groups were found however not to be significantly different.

It is concluded, therefore, that subjects may be trained to discriminate their heartbeats.

The next phase of this experiment was to submit subjects who had been trained to discriminate their pulses to heart rate control training and determine whether or not this prior training facilitated their abilities to control their heart rates. The Control group selected for comparison was that which displayed the lowest discrimination scores, i.e., the Control group which revealed tones at an irregular rate during the first phase of the experiment.

Heart Rate Control Data

The heart rate (bpm) during the left-light (low heart rate) trials were subtracted from the heart rates produced during the right-light (high heart rate) trials for each trial pair. Thus the resultant heart rate difference scores were large and positive if subjects demonstrated good heart rate control, and small if subjects displayed poor heart rate control. An analysis of variance was applied to the mean

heart rate difference scores for the two trial pairs within each Feedback and No-Feedback period during Session I and Session II (Table 2). The nonsignificant groups effect ($F = 2.045$, $df = 1/12$, $p > .05$) indicated that mean heart rate control did not differ significantly between the two groups. A significant groups by periods interaction ($F = 6.710$, $df = 1/12$, $p < .05$) meant however that heart rate control on Periods I and II was not independent of the treatment groups involved. As will be observed in Figure 3, only the Experimental group displayed an improvement in heart rate control over both Feedback and No-Feedback periods. The heart rate difference scores averaged over the Feedback and No-Feedback conditions for the Experimental group on Period I were compared with the same scores for the control group. This comparison revealed that heart rate control on Period I did not differ significantly between the two groups ($t = 0.543$, $df = 12$, $p > .05$). Another comparison between the Experimental and Control groups was made on these scores for Period II, and a significant difference was found ($t = 2.181$, $df = 12$, $p < .05$). Thus a significantly greater heart rate control was displayed on Period II by the Experimental group. The significant feedback effect ($F = 11.135$, $df = 1/12$, $p < .01$) indicated that heart rate control for the two groups was differentially dependent upon the availability of feedback. From Figure 3 it can be seen that the change in heart rate control for the Experimental group from the No-Feedback to the Feedback period was larger than for the Control group. This observation supports the significant groups by feedback interaction, indicating that the change in heart rate control from the No-Feedback to the Feedback periods

Table 2

Summary of the Analysis of Variance on the mean heart rate difference scores between right and left-light trials for two trial pairs within each Feedback and No-Feedback block during the first and second periods for the Experimental (discrimination) and Control Groups.

Source	df	Mean Square	F
Between Subjects	13		
Groups (A)	1	124.80	2.045
Subjects within groups	12	61.02	
Within Subjects	42		
Periods (B)	1	1.14	--
A × B	1	44.64	6.710*
B × subjects within groups	12	6.65	
Feedback (C)	1	21.63	11.135**
A × C	1	13.21	6.803*
C × subjects within groups	12	1.94	
B × C	1	1.79	--
A × B × C	1	8.64	1.203
B × C × subjects within groups	12	7.18	

* = $p < .05$ ** = $p < .01$

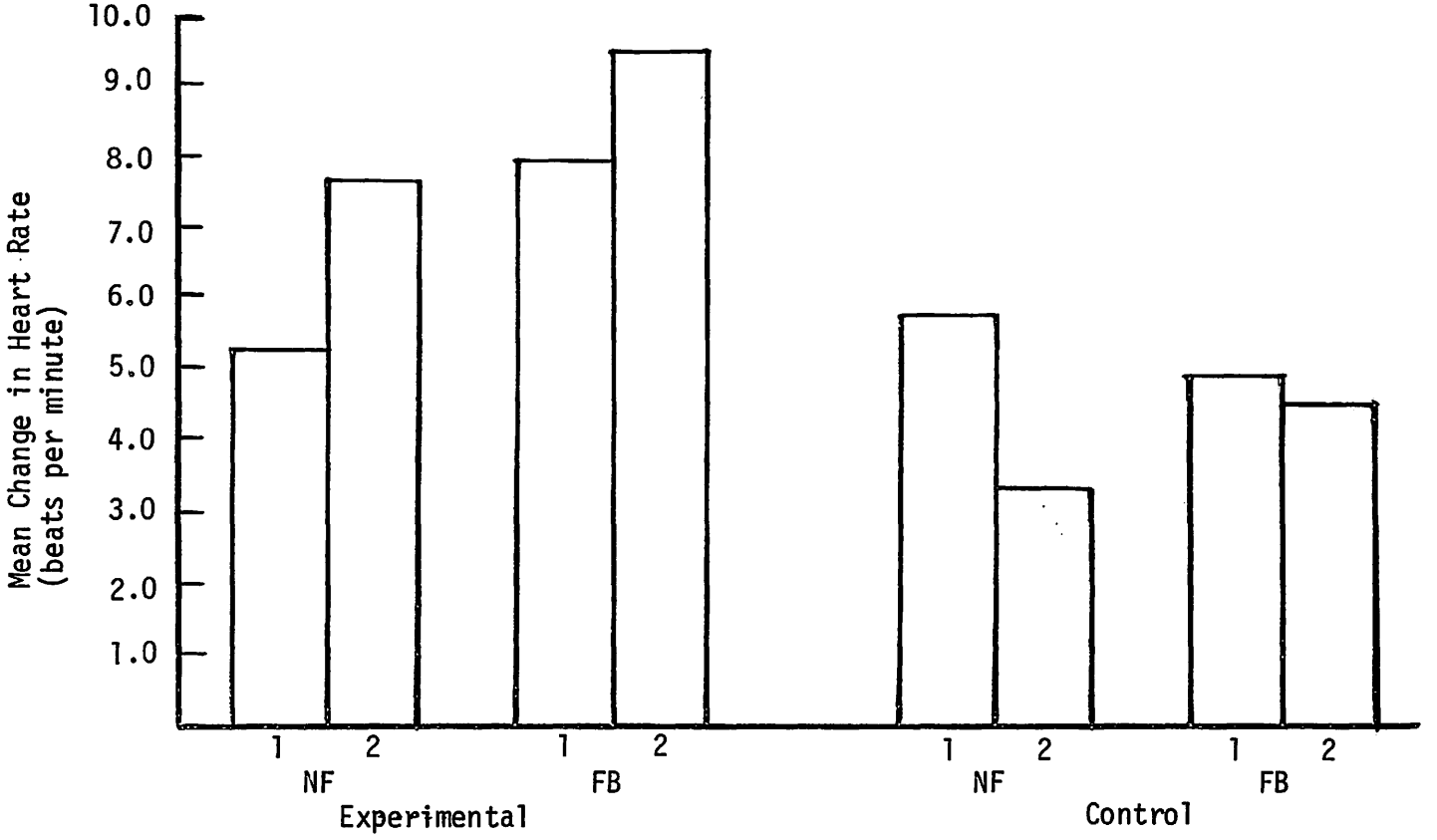

Figure 3. Mean heart rate difference scores between right and left-light trials for two trial pairs within each Feedback (FB) and No-Feedback (NF) block for the Experimental and Control Group.

was not independent of the treatment group involved. The heart rate difference scores averaged over periods for the Experimental group during the No-Feedback condition were compared to the same scores for the Control group. It was found that heart rate control did not differ significantly between the two groups during No-Feedback conditions ($t = 0.991$, $df = 12$, $p > .05$). Another comparison for the two groups was made for the Feedback condition and a significant difference resulted ($t = 1.856$, $df = 12$, $p < .05$). The Experimental group, therefore, demonstrated significantly better heart rate control than the Control group during the Feedback condition. A nonsignificant periods by feedback interaction ($F < 1.0$) indicated that the change in heart rate control as a function of training, averaged over groups, was similar under the No-Feedback and the Feedback periods. The nonsignificant three factor interaction revealed that the improvement in heart rate control performance as a function of the feedback conditions during each period was not dependent upon the different treatment procedures ($F = 1.203$, $df = 1/12$, $p > .05$).

From the pulse discrimination and heart rate control data, the following conclusions were drawn. First, a significantly greater improvement in heart rate control developed as a function of previous pulse discrimination training. During the first stages (Period I) of heart rate control the Experimental group performed at the same level as the Control group. But during the second stages (Period II) the Experimental group performed significantly better on heart rate control.

Secondly, significantly greater heart rate control was displayed during Feedback periods. Thirdly, pulse discrimination training led to a significant improvement in heart rate control under conditions of augmented sensory feedback. During the No-Feedback conditions, the Experimental and Control heart rate performances were similar. But during Feedback conditions the Experimental group demonstrated significantly greater heart rate control.

III. DISCUSSION

Particularly relevant to the purposes of this experiment were the Group, Feedback, Groups by Periods, and Groups by Feedback effects. The highly significant improvement in heart rate control displayed by both groups from the No-Feedback to the Feedback periods argues for the facilitating effect of feedback information toward the development of voluntary heart rate control. The groups by periods result and the t-tests performed on the mean scores for the two groups on Period I and on Period II indicated previous discrimination training resulted in significantly better heart rate control during the heart rate training procedure. Although the group means did not differ significantly, the groups by feedback interaction and the t-tests performed for the means of the two groups for the No-Feedback and for the Feedback conditions provided evidence that heart rate control during Feedback periods developed to a significantly greater degree as a function of prior pulse discrimination training. Thus the effectiveness of extrinsic

feedback information of heart rate in facilitating the development of learned heart rate control was a function of the procedures in which subjects had previously learned to discriminate their heartbeats.

The rationale underlying the use of extrinsic feedback involves the necessity of providing a means by which the intrinsic stimulus consequences of an internal response may be identified and controlled in an instrumental fashion. Since response contingent feedback (direct or indirect) is requisite to the acquisition of an instrumental response, it appears reasonable to suggest that the significantly greater improvement in heart rate control demonstrated by the Experimental group during Feedback periods may well have been due to the facilitating effects of the extrinsic feedback of heart beats made available during the discrimination training procedure. Thus it may be speculated that the Experimental group learned to identify intrinsic pulse contingent afferentation by associating the extrinsic feedback stimulus with the internal event, and effectively transferred this learning to the rate dimension of the new feedback stimuli during the heart rate control procedure. The Control group was not provided with pulse contingent feedback during the first phase of the experiment and might not have been able to utilize the discriminative function of the extrinsic feedback made available during heart rate training as effectively as the Experimental group. The fact that the Experimental group improved to a significantly greater extent than the Control group during Feedback periods supports this contention.

In summary, the present experiment demonstrated first, that significantly better heart rate control was demonstrated during Feedback periods. Secondly, that a significantly greater improvement in heart rate control developed as a function of previous pulse discrimination training. Discrimination training also led to a significantly greater improvement in heart rate control during conditions of augmented sensory feedback. The results of this experiment are taken to support the hypothesis that if the discrimination of internal afferent information associated with a visceral response is necessary to the development of instrumental control over that response, then training designed to facilitate this discrimination should also facilitate the development of learned visceral control. It was suggested that augmented sensory feedback may have facilitated this discrimination and subsequent control.

The finding in the present study that pulse discrimination training facilitated the development of learned heart rate control, and that such control was better during Feedback conditions, suggested the possibility that learned control over other aspects of cardiovascular responding might develop following the implementation of operant procedures which utilized augmented sensory feedback of the relevant response process. The following experiment addressed itself to this question by attempting to establish voluntary control over systolic blood pressure changes.

CHAPTER III

EXPERIMENT II: LEARNED CONTROL OF INCREASES AND DECREASES IN SYSTOLIC BLOOD PRESSURE

I. METHOD

Subjects

The subjects were fifteen normotensive male undergraduates who reported no histories of cardiovascular disease.

Apparatus

Rapid sampling of systolic blood pressure was accomplished by monitoring blood pressure changes from the left index finger rather than the brachial artery, since the latter technique does not allow for prolonged measurements without ischemic discomfort. The digital cuff measured slightly less than the circumference of the finger leaving about 1/4 inch of the dorsal digit unoccluded. Because venous flow in the index finger is accomplished primarily via dorsal vessels, the digital cuff employed thus obviated discomfort due to blood pooling by permitting venous return from the partially occluded digit. A crystal pulse sensor (Beckman Infraton Pickup) was attached with surgical tape to the finger distal to the cuff, and produced an electrical signal when an arterial pulse wave was detected. This signal was amplified (Grass Model P511 A. C. Preamplifier) and used to drive a solenoid valve which gated air flow at 17 psi (produced by a compressor) to a

plexiglass air pressure reservoir. This reservoir distributed pneumatic pressure to the cuff, a manometer, and a bleed valve which was continuous with atmospheric pressure. Each pulse detected by the pulse sensor produced a cuff inflation of about 5 mm. Hg until the cuff pressure was sufficient to occlude arterial blood vessels (cuff pressure higher than systolic pressure) thus preventing the activation of the pulse sensor. In the absence of a signal from the sensor, the cuff pressure was bled off through the bleed valve at about 3 mm. Hg per second until the cuff pressure just permitted an arterial pulse to pass and activate the sensor. The operation of this feedback system thus resulted in a cuff pressure which oscillated about the systolic arterial pressure. Bleed rates were adjusted to subject's heart rate so as to maximize the number of blood pressure determinations to about one determination every 2 - 3 heartbeats. No ischemic discomfort was reported by any subject. Distortion of the arterial pulse wave results in progressively higher systolic peaks as the pulse wave traverses to the periphery (Hurst and Logue, 1966) so that systolic values obtained via this technique were consistently higher (about 15 mm. Hg) than these blood pressure values obtained simultaneously at the brachial artery. A pressure transducer (E and M Model ESG-300) was pneumatically linked to the cuff and presented an electrical signal to a D. C. amplifier (E and M Transducer-Monitor-Coupler) which operated a voltage to frequency converter. The output pulses of the converter, which were produced at a rate directly proportional to cuff pressure, were converted

to square waves of constant duration, amplified (BRS logic modules), and used to operate an electromagnetic counter. By this technique blood pressure could be rapidly sampled for a predetermined time interval and an integrated measure of blood pressure displayed on the counter at the end of the sampling period. The value which appeared on the counter was then converted to the absolute systolic level. The counter and manometer were displayed to subjects and constituted two forms of augmented sensory feedback of blood pressure performance.

Heart rate was continuously monitored by recording electrocardiographic R - waves (lead II) which were converted to square waves of constant duration (BRS logic modules) and displayed on an electromagnetic counter.

Procedure

An Increase, Decrease, and Control group were each constituted by five subjects. Subjects sat in a reclining chair in a sound deadened cubicle (7' × 8-3/4' × 4-3/4') and were run for two sessions separated by about 48 hours. A session consisted of 20 training trials, each 50 seconds in duration, and separated by a variable intertrial interval of about 30 seconds. Immediately before the onset of the first trial, 4-6 blood pressure determinations were made on experimental subjects in the absence of feedback displays in order to assess subject's basal systolic level. Three signal lamps and the feedback displays were operative only during the trial periods. During the intertrial interval,

the feedback displays were illuminated by one dim lamp. Continuous records of blood pressure and heart rate were taken throughout each trial.

Experimental subjects were told that the experiment was designed to investigate whether or not they could increase (Increase group) or decrease (Decrease group) their blood pressures, and were instructed as to the significance of the feedback displays (actual instructions are reproduced in the Appendix, Note 2). Increase subjects were told to keep the manometer reading as high as possible throughout each training trial and to accumulate a higher counter total on successive trials, and then to reset the counter by pushing a button on the counter face. Decrease subjects were given the same instructions except that the direction of their required response was a decrease in blood pressure. A control group was run in order to assess the effects on systolic blood pressure of exposure to the experimental environment and the feedback stimuli employed. Control subjects were told that the experiment was designed to investigate their cardiovascular processes and all they were to do was to pay close attention to the feedback display during trial periods, and to reset the counter at the end of each trial.

II. RESULTS AND DISCUSSION

Blood Pressure Data

Figures 4 and 5, respectively present systolic blood pressure changes (training trial minus basal values) during Sessions I and II

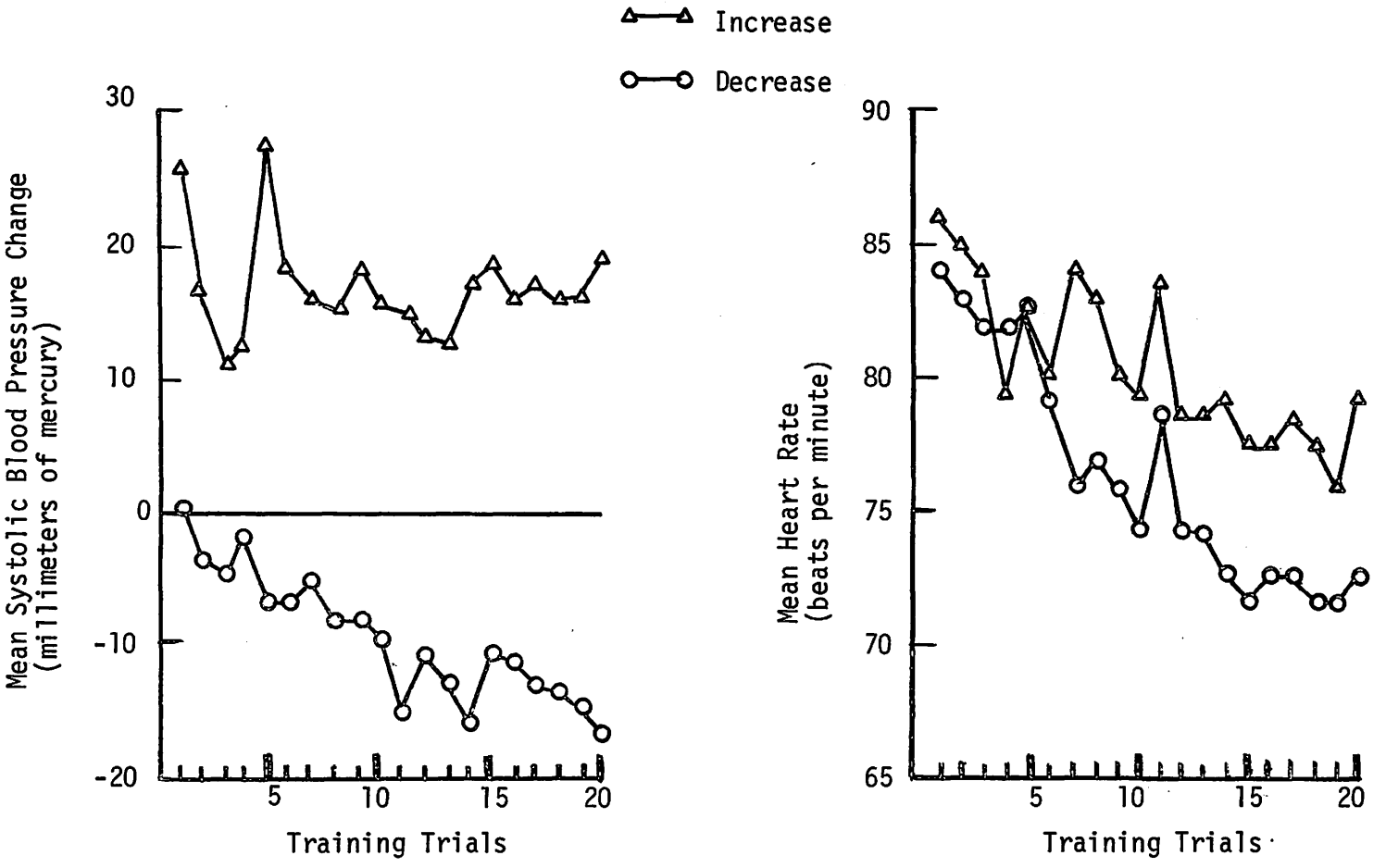

Figure 4. Mean systolic blood pressure change from baseline and mean heart rate for Increase and Decrease blood pressure groups for Session I.

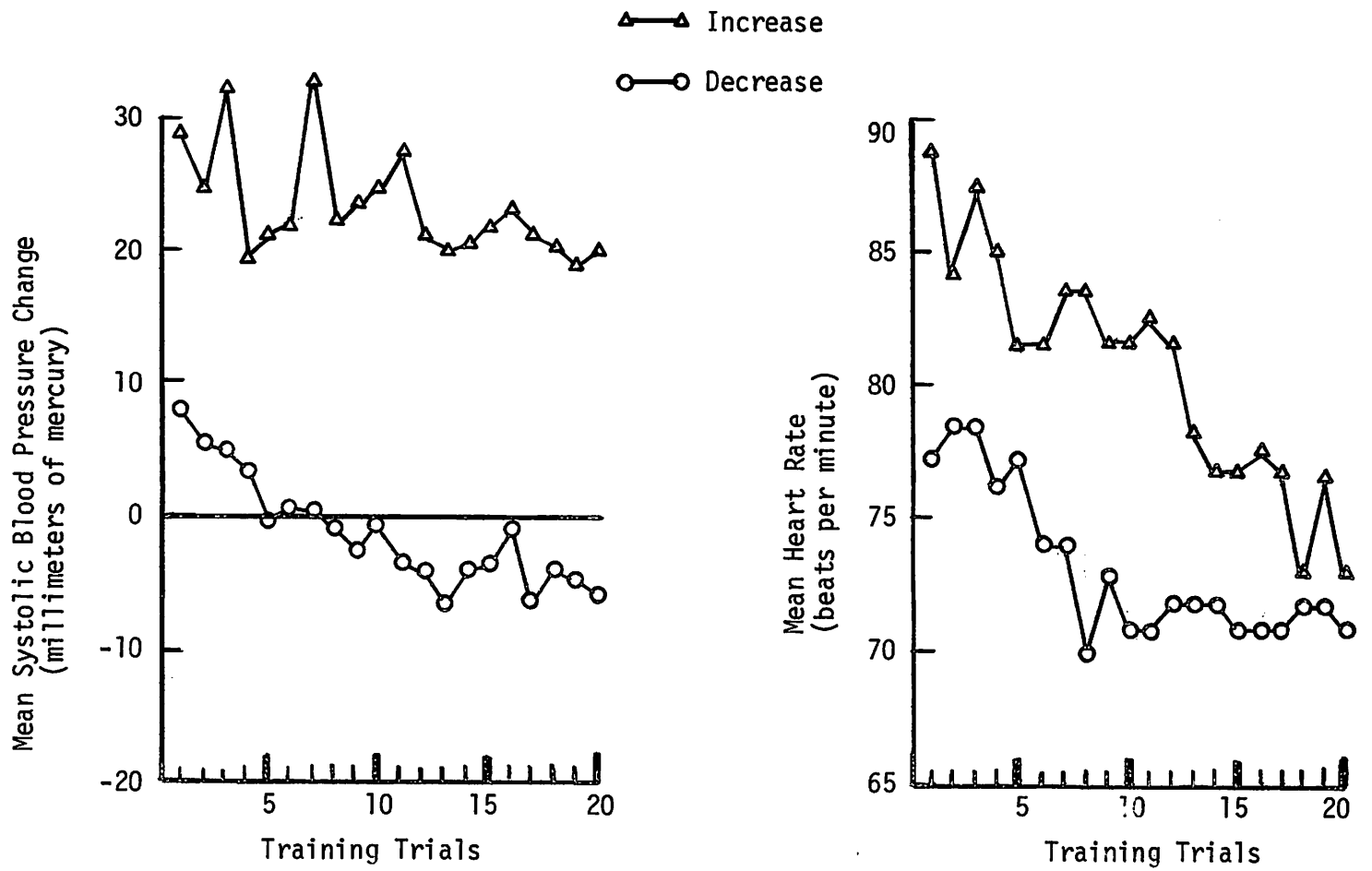

Figure 5. Mean systolic blood pressure change from baseline and mean heart rate for Increase and Decrease blood pressure groups for Session II.

for the Increase and Decrease groups. Both groups exhibited blood pressure changes which were in the predicted direction as a function of training. An analysis of variance (Winer, 1962) (Table 3) revealed significant differential blood pressure performances for the two groups ($F = 10.368$, $df = 1,8$, $p < .05$). The decreasing trend exhibited by each group over trials for both sessions resulted in a significant trials effect ($F = 3.391$, $df = 19,152$, $p < .01$) but an insignificant groups by trials interaction ($F = 1.165$, $df = 19,152$, $p > .05$). The two groups demonstrated differential blood pressure changes with respect to their performances on Sessions I and II. Increase subjects produced generally higher systolic changes from baseline on Session II than on Session I (Figure 5), whereas the Decrease group demonstrated greater decrements on Session I (Figure 4). Consequently, the mean blood pressure performances for Sessions I and II were not significantly different ($F = 4.368$, $df = 1,8$, $p > .05$). In addition, the larger increment noted for the Increase group on Session II was paralleled by the decrease in the degree of blood pressure decrement exhibited by the Decrease group on Session II and a nonsignificant groups by sessions interaction resulted ($F < 1.0$). An examination of Figures 4 and 5 reveals that the combined trends of both groups on Sessions I and II are of the same (decreasing) form, supporting the nonsignificant sessions by trials interaction ($F < 1.0$). In addition, the trends for the Increase and Decrease groups considered separately on Sessions I and II are also of the same (decreasing) form, an observation which supports the nonsignificant groups by sessions by trials interaction ($F < 1.0$).

Table 3

Summary of the Analysis of Variance on the systolic blood pressure difference scores from baseline for the Increase and Decrease Groups during training trials on two sessions.

Source	df	Mean Square	F
Between Subjects	9		
Group (A)	1	6433.25	10.368*
Subjects within groups	8	6205.26	
Within Subjects	390		
Sessions (B)	1	5464.17	4.368
A × B	1	181.17	--
B × subjects within groups	8	1251.09	
Trials (C)	19	195.68	3.391**
A × C	19	67.23	1.165
C × subjects within groups	152	57.70	
B × C	19	47.70	--
A × B × C	19	46.81	--
B × C × subjects within groups	152	85.54	

* = $p < .05$ ** = $p < .01$

Since both groups exhibited a decreasing trend in blood pressure values as a function of training within each session, it might be argued that the data represent a differential habituation to the experimental environment. The Control subjects provided data indicating, however, that sustained elevations or decrements in systolic blood pressure are not associated with exposure to the experimental environment. Figure 6 presents this data for Sessions I and II respectively. It will be observed that blood pressure levels do not change in a systematic way within sessions as a function of exposure to the experimental environment. The mean blood pressure values for the first and last blocks of five trials are respectively 134.5 mm. Hg and 136.9 mm. Hg for Session I, and 126.2 mm. Hg and 126.6 mm. Hg for Session II.

Heart Rate Data

Since systolic blood pressure is primarily influenced by cardiac output (heart rate × stroke volume) (Hurst and Logue, 1966; Rushmer, 1961), the relationship between heart rate and systolic blood pressure was analyzed in order to determine to what extent systolic changes were associated with changes in heart rate. Figures 4 and 5, pages 51 and 52, present the heart rates for the Increase and Decrease groups as a function of training for Sessions I and II respectively. Although the heart rates over Sessions for the Increase group were generally higher than the Decrease group, (mean values = 80 bpm. and 75 bpm. respectively), within group variability did not permit a significant group difference ($F = 1.351$, $df = 1,9$, $p > .05$) (Table 4). Since decreasing heart rates

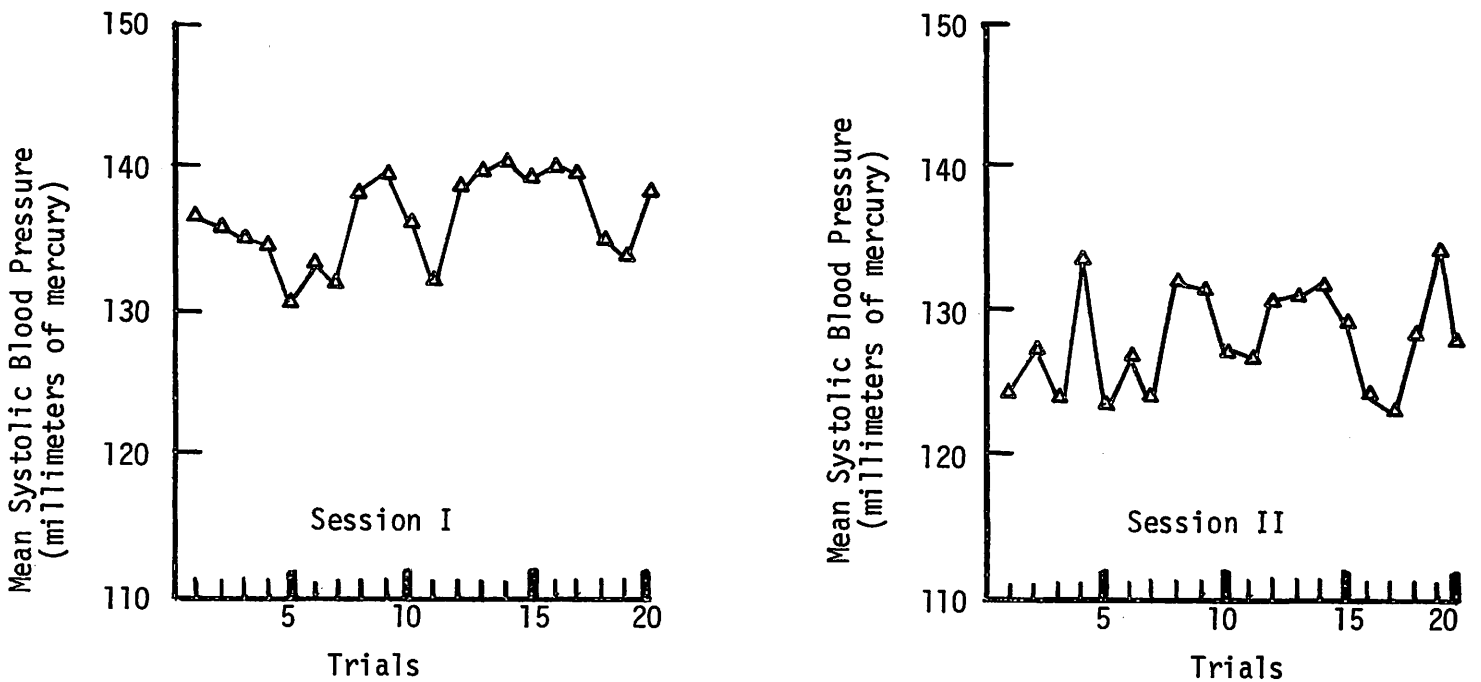

Figure 6. Mean systolic blood pressure for Control Group for Session I and Session II.

Table 4

Summary of the Analysis of Variance on the heart rate scores for the Increase and Decrease blood pressure Groups during training trials on two sessions.

Source	df	Mean Square	F
Between Subjects	9		
Group (A)	1	2381.40	1.351
Subjects within groups	8	1762.49	
Within Subjects	390		
Sessions (B)	1	176.90	--
A × B	1	169.00	--
B × subjects within groups	8	2087.73	
Trials (C)	19	164.95	9.048**
A × C	19	15.21	--
C × subjects within groups	152	18.23	
B × C	19	6.10	--
A × B × C	19	21.37	
B × C × subjects within groups	152	10.69	1.999*

* = $p < .05$ ** = $p < .01$

over training trials characterized both groups on both sessions, no significant sessions effect ($F = < 1.00$) or groups by sessions interaction ($F = < 1.00$) resulted. As might be expected, the decreasing trend for heart rate during all experimental conditions was reflected in a significant trials effect ($F = 9.050$, $df = 19.152$, $p < .01$), but a nonsignificant groups by trials interaction ($F < 1.00$) and sessions by trials interaction ($F < 1.00$). Since heart rate for the two groups on Session I diverges as a function of blood pressure training, whereas heart rate on Session II converges, the significant groups by sessions by trials interaction was not unexpected ($F = 1.999$, $df = 19,152$, $p < .05$).

Peak systolic pressure is primarily a function of the left ventricular stroke volume, rate of ejection, and the distensibility of the aortic walls (Rushmer, 1961). Thus elevations in systolic pressure can be brought about by increases in cardiac rate and conversely, decrements in heart rate may be associated with systolic decreases. Although cardiac rates were generally higher for the Increase group, the fact that group means differed significantly on the blood pressure variable but not on heart rate, indicates that the latter measure did not contribute significantly to the observed group blood pressure differences. On Session I, however, group differences in blood pressure increased as a function of training, and so did heart rate. Group differences on heart rate diverged from complete overlap during the first five trials to a difference of 5 bpm. averaged over the last five trials; the Increase group exhibiting the higher cardiac rate.

Conversely, on Session II, while group blood pressure differences increased again as a function of training, the heart rates for the two groups converged from 8 bpm. to 4 bpm. averaged over the first and last five trials respectively. The Increase group still retained the higher heart rates. That these observed differences in heart rate performance as a function of training for the Increase and Decrease groups were not independent of the two Sessions was supported by the significant three factor interaction. Thus it appears that the differences in systolic blood pressure observed for the two groups were correlated with heart rate changes to a greater extent during training on Session I than on Session II.

Since a decrement in the association between heart rate and blood pressure was displayed during the second session, it would be difficult to determine which aspect of cardiovascular responding mediated the systolic blood pressure changes during Session II. Although changes in stroke volume may have influenced blood pressure levels on Session II, two points mitigate this argument. First, increases in diastolic filling pressure resulting in a greater stroke volume are normally produced by the "muscular pumping" action in the lower limbs which forces blood back to the heart. The subjects in the present experiment, however, assumed a semi-reclining position, remained relaxed and relatively immobile. Thus it is doubtful whether stroke volume was increased by this means. Secondly, an increased myocardial contractility might have reduced the end systolic ventricular reserve, thus

increasing the stroke volume, but this effect, however, is minimal and most likely could not have maintained the large elevations in blood pressure (about 20 mm. Hg) observed on Session II. An increased venous return and stroke volume might have resulted from an elevated intra-abdominal and a decreased intra-thoracic pressure both of which are attendant upon deep inspirations. At the end of each training session, however, subjects reported that they had not engaged in irregular or "abnormal" respiratory behavior patterns while attempting to control their blood pressure. It is possible that an increase in peripheral vascular resistance may have resulted in larger systolic peaks by presenting arterial pulse waves with a greater arterial diastolic pressure. This possibility should be systematically investigated. Unfortunately, the only cardiovascular variables monitored were systolic blood pressure and heart rate. Any statements concerning the underlying mechanism by which the observed blood pressure changes were mediated, therefore, would be quite speculative, and do require further experimental investigation.

A Control for the Possible Unconditional Effects of the Feedback Stimuli Employed

The dramatic blood pressure increments produced by the Increase group on trial one for both sessions does not conform to the usual exponential learning curve, and suggested the possibility that increase subjects were responding to some unconditional effect of the feedback stimuli and the instructions. Although the Decrease group experienced

the same environmental stimuli as did the increase subjects, and evidenced no similar increases, it was decided to run an additional control. Five male undergraduates were run according to the same procedure as the experiment described immediately above, except that eight basal trials preceded, and four basal trials followed the twenty training trials. The eight pretraining basal trials were divided into two blocks of four trials each. During one block the signal lamps and feedback stimuli (manometer dial and counter) were operative, while during the other block these stimuli were not. Block presentation sequences were partially counterbalanced between subjects. Post-training basal trials monitored blood pressure levels in the absence of extrinsic feedback. Following the presentation of all eight pretraining basal trials, subjects were given the same instruction as previously described for the Increase group. Since subjects were familiar with both the required task, the signal lamps, and feedback stimuli prior to Session II, the non-feedback pretraining basal trials were omitted on this Session. Post-training basal measurements were obtained as in Session I. Figures 7 and 8 present the pretraining, training, and post-training trial blood pressure values respectively for Sessions I and II. It will be observed that on Session I the systolic level decreases as a function of pretraining baseline trials regardless of whether signal lamps or feedback stimuli were presented to subjects. Systolic levels also exhibit a decrement during feedback pretraining basal trials on Session II. As in the previously presented

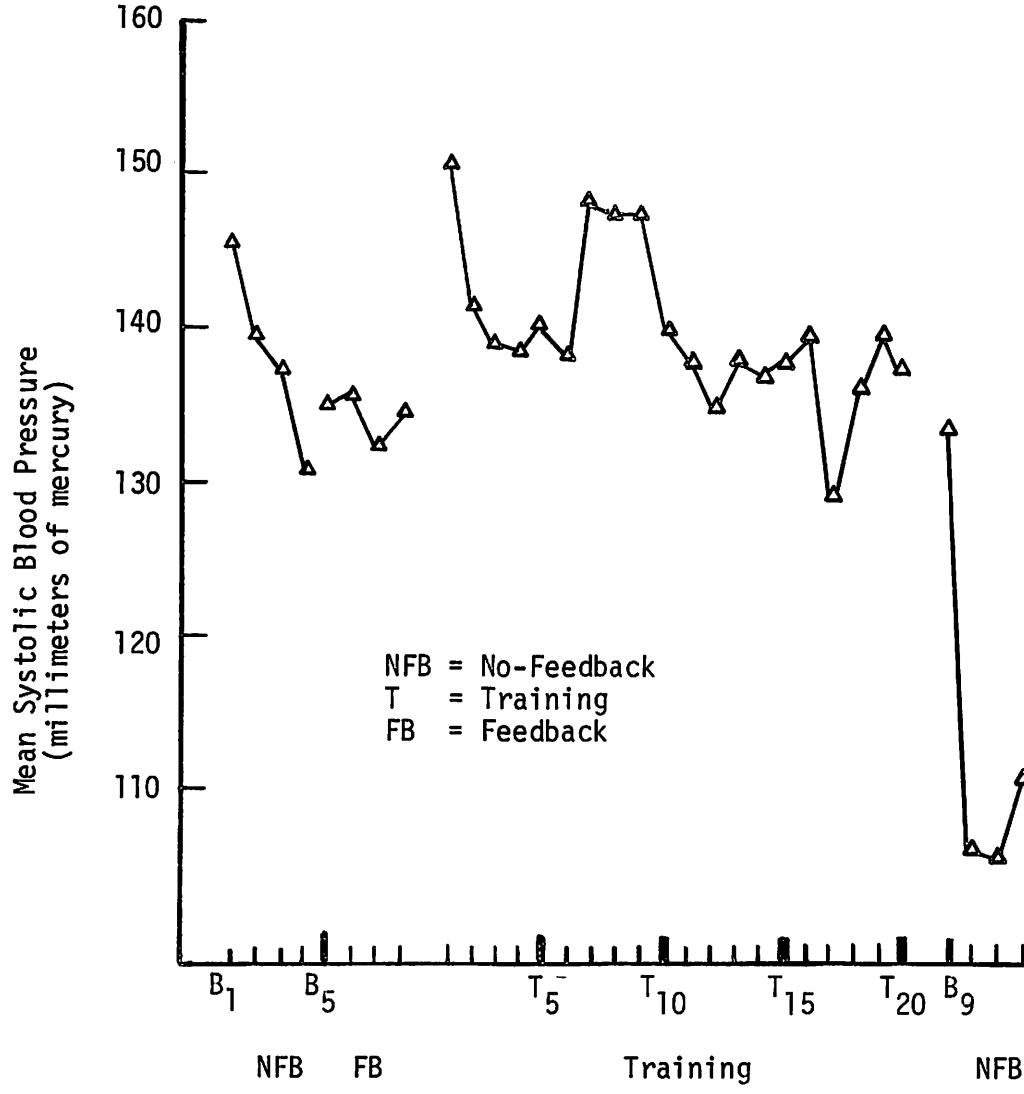

Figure 7. Mean systolic blood pressure for feedback stimulus Control Group on Session I.

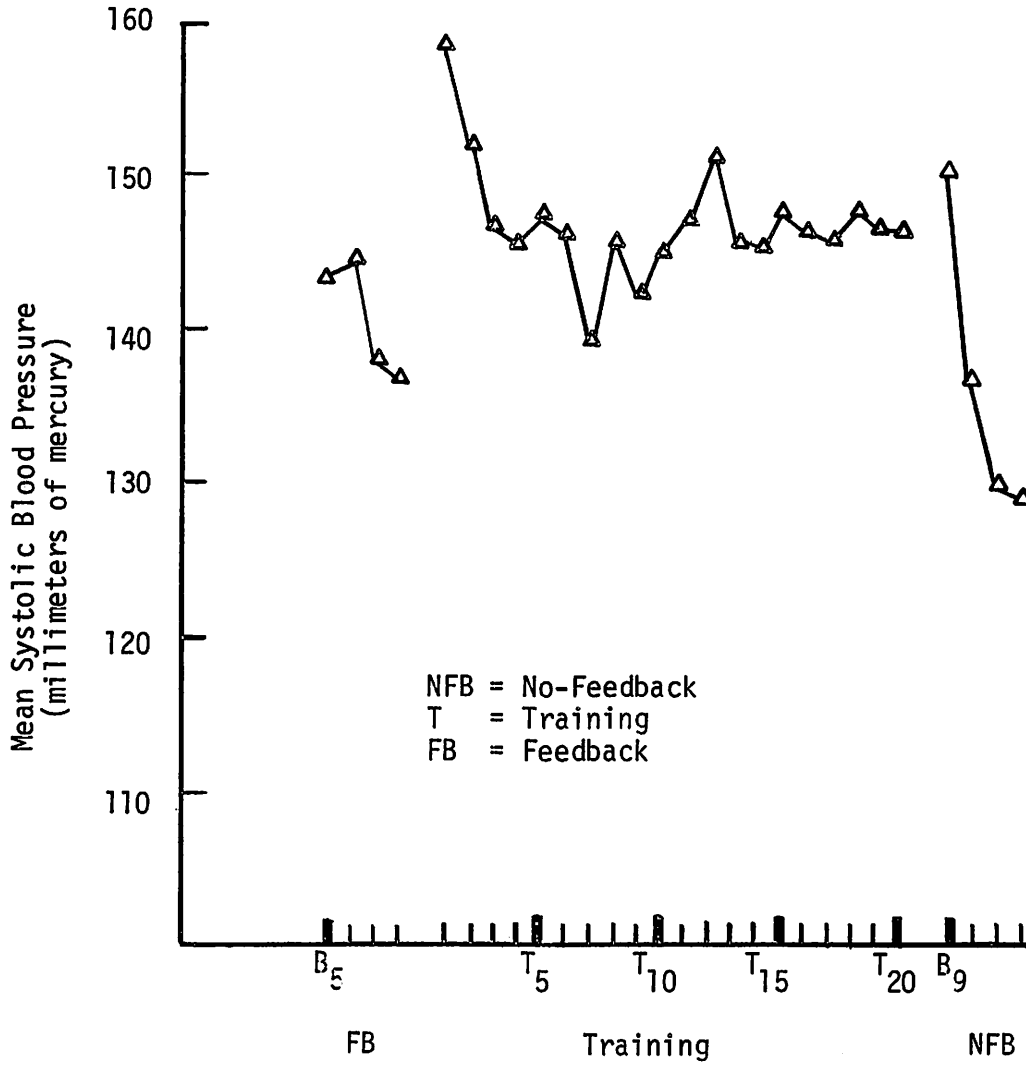

Figure 8. Mean systolic blood pressure for feedback stimulus Control Group on Session II.

blood pressure increase data, training trial one was characterized on both Sessions by a dramatic and immediate increase in systolic values. These control data thus indicated that the sharp increment obtained on training trial one was not due to any unconditional effects of the signal lamps or feedback stimuli, but rather to the instructional requirements. The post-training baseline data demonstrated, in addition, that termination of the instructional requirements was associated with a sharp decrease in systolic values to baseline levels.

In summary, these results demonstrated that following the implementation of a simple operant procedure, significant elevations and decrements in systolic blood pressure levels may be obtained. Furthermore, such changes can develop according to an instructional requirement and not as a function of unconditioned stimulus effects or differential habituation to the experimental environment. The heart rate data indicated that systolic blood pressure levels may be operantly modified independently of any systematic changes in heart rate. This finding supports other research which demonstrated independence of instrumentally conditioned changes in systolic blood pressure and heart rate (Dicara and Miller, 1968; Shapiro, Tursky, Gershon, and Stern, 1969; Shapiro, Tursky, and Schwartz, Circulation, now in press), and instrumentally conditioned changes in heart rate and systolic blood pressure (Shapiro, Tursky, and Schwartz, Psychosomatic Medicine, now in press). In addition, the results of the present experiment indicated that although average heart rates may not differ significantly

between subjects required to increase and decrease their systolic blood pressure, such changes may nevertheless be associated with differences in heart rate to a greater extent during certain periods in the development of learned blood pressure control than during other periods. Moreover, this learning process may be characterized by the development of considerable cardiovascular response specificity, i.e., the gradual elimination of those responses (in the present experiment heart rate) which may mediate the required blood pressure change less efficiently. In light of the work by Lacy and Lacy (1958) which demonstrated that hierarchies and patterns of autonomic responding develop which are individualistic, it seems probable that individual cardiovascular response patterns might also emerge during the development of learned blood pressure control. Research designed to investigate the relative importance of such variables as heart rate, stroke volume, and peripheral vascular resistance in determining blood pressure changes would provide the basic data for the identification of individual cardiovascular response patterns. The presentation of augmented sensory feedback of these individual response patterns to subjects who were required by curious investigators or in more dire circumstances by the presence of chronic essential hypertension to acquire learned blood pressure control might provide the optimal conditions for such learning to develop.

CHAPTER IV

SUMMARY AND CONCLUSIONS

Summary of Results

The first experiment in this dissertation investigated the hypothesis that the discrimination of afferent information associated with a visceral response will facilitate the development of learned control over that response. During the first phase of the experiment, subjects were presented with extrinsic feedback of their heartbeats on seven trials. Each of these trials was preceded by three no-feedback trials during which subjects were not presented with feedback, but were asked to press a button each time they discriminated a heartbeat. Two control groups received extrinsic stimulus presentations which were not contingent upon any aspect of their cardiac behavior. During the second phase, Experimental subjects and the subjects in one of the Control groups were instructed to increase or decrease their heart rates under conditions of augmented sensory feedback of heart rate, and under conditions when no extrinsic feedback was provided. The results of this experiment demonstrated that significantly better heart rate control developed during Feedback periods regardless of the group involved. A significant improvement in heart rate control also developed as a function of previous pulse discrimination training. Finally, pulse discrimination training led to a statistically reliable improvement in heart rate control during conditions of augmented sensory feedback.

These results were taken to support the original hypothesis that training designed to facilitate discrimination of the internal afferent information associated with a visceral response would also facilitate the development of learned control over that response. The findings of Experiment I taken together with other reports that instrumental visceral control is more effective under conditions of extrinsic response-contingent feedback of visceral responding suggested the possibility that voluntary control might be developed over other aspects of cardiovascular functioning following the implementation of procedures which utilized augmented sensory feedback of the relevant response process.

The second experiment in this dissertation investigated the possibility of producing voluntary control over changes in systolic blood pressure. During two training sessions, two groups of subjects were instructed to increase or decrease their systolic blood pressure while receiving virtually continuous extrinsic information of blood pressure changes. Two additional Control groups were run, the first in order to assess the effects on systolic blood pressure of habituation to the experimental environment, and the second to test for any unconditioned effects on blood pressure by the feedback stimuli employed. Heart rate was continuously monitored in all subjects to determine the relationships between changes in this cardiac response and systolic blood pressure. The results of this study demonstrated that following the implementation of a simple operant procedure, statistically reliable

increases and decreases in systolic blood pressure may be obtained. Moreover, such changes developed according to an instructional requirement and not as a function of habituation to the experimental environment, or as a result of unconditioned stimulus effects. Although these blood pressure changes were produced in the absence of any systematic changes in heart rate, it was found that increases and decreases in blood pressure were associated with greater heart rate differences during certain periods in the development of learned blood pressure control than during other periods.

Theoretical Significance of These Findings

The results which are of central importance to this dissertation are these: First, that the ability to discriminate the intrinsic stimulus consequences of an internal response can be experimentally altered. Secondly, as a consequence of such changes in discriminative ability, voluntary control over that visceral response is altered. Thirdly, operant procedures which result in voluntary autonomic control are not limited to the control of a particular aspect of autonomic functioning. The theoretical import of these findings pertains specifically to the manner in which somatic and autonomic response processes have been viewed with respect to their status as voluntary control systems. In psychological literature the traditional opinion concerning somatic and autonomic behavior has been that autonomic effectors provide either no afferentation or afferent information too

diffuse to permit an organism to discriminate and exert control over the intrinsic stimulus consequences of an autonomically-mediated response. This opinion gained support from the common observation that visceral responses could not be voluntarily controlled. Thus the somatic nervous system was classified as a voluntary response system, while the autonomic nervous system was assigned to an involuntary position.

A strict theoretical distinction between the voluntary and involuntary aspects of somatic and autonomic functioning is severely compromised by the finding that individuals can learn to discriminate the occurrence of an autonomically-mediated response (heartbeat) and then, as a function of this discriminative ability, learn to voluntarily control that response. Moreover, the generalization of voluntary control to other aspects of cardiovascular functioning (blood pressure) lays strain upon this theoretical position. The results of Experiment I as well as the finding by other investigators that voluntary cardiovascular control is facilitated under conditions of extrinsic feedback from the relevant response, suggest further that extrinsic feedback functions to augment the discrimination of the intrinsic afferentation which normally reaches the CNS from visceral organs. In light of the physiological, anatomical, and the present psychological evidence to the contrary, it is difficult to accept a theoretical position which maintains that a qualitative distinction exists between the autonomic and somatic response processes according to their status as voluntary control systems.

Rather, a theoretical viewpoint has been adopted which supports the existence of a functionally integrated efferent nervous system, all aspects of which may yield to voluntary control.

Potential Significance of the Results of this Dissertation

In general, the results reported herein are directly relevant to the possibility that discriminative training and operant control procedures similar to those described, can be utilized to duplicate and thereby gain an understanding of the development of emotional and psychosomatic behavior. In addition, the possibility of applying these techniques in the clinical setting is also indicated.

Development of voluntary control of subjective emotional states. It is readily observable that subjective emotional states are often manifested by changes in autonomic behavior. The extensive work of Hans Selye is notable for the descriptions which it has provided of the various autonomic changes which are associated with prolonged stress. In light of the results of Experiment I, it is suggested that emotional behavior manifested as visceral responding may also be voluntarily controlled following the implementation of appropriate discrimination training and operant control procedures. According to Mandler (1962), the instrumental control of emotional behavior is dependent upon the ability to identify the stimulus consequences of autonomic responding which are associated with a particular emotional event. Mandler added, however, that the only internal stimuli

discriminable were probably those deriving from a generalized sympathetic arousal. According to this opinion, the internal stimuli associated with a particular visceral response can not be discriminated to a degree that would permit the development of specific visceral control. The present findings demonstrated, however, that discrimination training resulted in the identification of the stimulus consequences of a rather specific aspect of cardiovascular functioning, namely the heartbeat; and further that voluntary control over a particular response dimension of the heartbeat (rate) developed as a consequence of pulse discrimination training. Thus it may be possible to train individuals to discriminate the stimulus consequences of a particular visceral response which is associated with a specific subjective emotional state.

Relevant to this suggestion is the concept of Stimulus-Response Specificity. Although the various autonomic responses (palmar sweating, brain waves, heart rate, gastric motility, etc.) exhibit a typical response during a generalized arousal or activation, several studies have made it clear that particular patterns of autonomic responding reliably accompany certain external stimulus conditions. Ax (1953) created fear producing and anger producing experimental situations by exposing subjects to an "incompetent" polygraph operator who insulted and criticized them (anger situation) or expressed apprehension over the danger of high voltage electric shock being inadvertently delivered to them during the experimental procedures (fear situation). Ax found that the number of transient decreases in skin resistance, increases in

electromyograph activity, decreases in heart rate, and diastolic blood pressure increases were greater for the anger than the fear conditions. Wenger and Cullen (1958) measured nine autonomic variables as a function of fourteen stimulus conditions (electric shock, insertion of hypodermic needle, mental arithmetic, carbon dioxide inhalation, cold pressor, etc.) and obtained just as many autonomic response patterns as there were stimulus conditions. Lacy (1959) observed that particular autonomic response patterns were produced by functional classes of stimuli. The author stated that increases in the number of heart rate accelerations accompanied thinking and mental arithmetic tasks, whereas decelerations were associated with tasks requiring visual and auditory attention. These and other findings which demonstrated Stimulus-Response Specificity in autonomic responding led Sternbach (1966) to conclude that "The evidence is now quite convincing that there is such a thing as situational stereotopy, or stimulus-response specificity, and . . . the patterns of (autonomic) response vary from situation to situation," (Sternbach, 1966, p. 91, brackets mine). Since particular autonomic response patterns accompany different emotional states, training designed to augment the identification of the afferent cues associated with these response patterns might facilitate their learned control. In each of these studies autonomic response patterns were reliably produced when subjects were presented with stimuli which elicited several subjective emotional states (anger, fear, apprehension, consternation). In view of the findings presented in this dissertation,

it appears reasonable to suggest that following appropriate discrimination training procedures, the intrinsic afferentation from the autonomic response patterns associated with particular emotional states could be effectively controlled. In individuals who display inappropriate emotional responding (anxiety reaction; phobic reaction; manic reaction, etc.) this type of training may serve as an important adjunct to current therapeutic techniques. It has been well established that the cardiovascular and other visceral response systems can be modified according to schedules of reinforcement. The work by Lacy and Lacy (1958) has shown that individuals develop tendencies to respond to stressful situations with particular profiles of autonomic responses. In fact, Ax (1964) has suggested the formulation of a Physiological Learning Aptitude scale and proposed that the development of neuroses and psychoses may be due to faulty or late physiological learning so that inappropriate physiological responses are made to social stimuli. Psychosomatic disorders typically develop within the context of a social interaction. Thus it is conceivable that psychosomatic disorders could be instrumentally acquired and maintained through a process of social reinforcement.

The development of psychosomatic disorders and their control. The possibility of developing discrimination and operant control of the intrinsic response produced stimuli associated with emotional behavior forms the basis for an understanding of the development and control of

psychosomatic disorders. In its traditional meaning, a psychosomatic disorder refers to a set of organic symptoms which are psychogenic in origin. Typically, medical treatment procedures are directed primarily towards the emotional component of the psychosomatic disorder, under the assumption that the emotion somehow precedes and causes the organic disturbance. Clearly, the theoretical position adopted here does not accept this dualistic conception of mental and visceral functioning. In Experiment II, it was demonstrated that large increments and decrements in systolic blood pressure followed the implementation of operant training procedures. This experiment may serve as a model indicating that psychosomatic disorders may be acquired as a result of an individual's history of response contingent reinforcement. It has been estimated (Rushmer, 1961) that 90 percent of all chronic hypertensive disorders are classified as essential hypertensive cases (etiology unknown). The possibility that many of these cases may be psychosomatic in nature cannot be overlooked. Operant procedures which result in learned blood pressure control, therefore, may serve as a particularly appropriate model to describe the development of a psychosomatic disorder.

In the laboratory the discriminative stimulus which sets the condition that a reinforcement contingency is in effect is easily specifiable; so are the required response and the reinforcing stimulus (extrinsic feedback) which informs the individual that he has emitted the correct response. In the "real world" these parameters are not so easily discernable. Yet an examination of the environmental events

which surround a psychosomatic episode may reveal that the appropriate contingencies are indeed in effect: Children quickly learn to control glandular responding at will in order to gain affection from their parents. Voluntary control over crying (lacrimal glands) is very commonly observed. Likewise, the child who holds his breath, is inadvertently reinforced (succorance from his mother) for an increase in blood pressure. Although the child has no knowledge of the specific facts concerning the cardiovascular changes which were followed by reinforcing attention from his mother, the attention -- a reinforcing stimulus -- serves as extrinsic feedback information which enables the child to emit the same behavior more easily on a subsequent occasion. In fact, the more often that large increments in blood pressure are produced, the more easily can they subsequently be emitted for two reasons. First, by emitting these responses time and again, the child practices the required response and receives feedback which enables him to develop more effective voluntary control. Secondly, large sustained blood pressure increments tend to be self perpetuating. Baroreceptors located in the carotid sinuses which normally respond to increases in blood pressure by producing compensatory decreases in heart rate can gradually reset at a higher level, thus becoming more and more insensitive to blood pressure elevations. Increases in blood pressure may also produce vascular lesions in the renal arteries resulting in reflexive blood pressure elevations due to the initiation of the renin-angiotensin system (Page and McCubbin, 1965; Rushmer, 1961).

One of the primary implications of a model which describes psychosomatic disorders as instrumentally acquired, concerns the possibility that as the principle of reinforcement governs the acquisition of a psychosomatic symptom, so should the principle of differential reinforcement lead to the acquisition of a desirable form of visceral responding, and the extinction of the psychosomatic disorder. Toward this end, the optimal use of augmented sensory feedback of visceral functioning is of paramount importance.

The definition of optimal forms of augmented sensory feedback. The execution of an instrumental act requires that information (topographic, nontopographic, artificial) concerning the effects of the response be discriminated. The results of Experiment I demonstrated that the effectiveness of extrinsic feedback of heart rate in facilitating the development of learned heart rate control was a function of prior pulse discrimination training. It was suggested, from this and other findings which supported the facilitatory role of extrinsic feedback, that during discrimination training pulse contingent extrinsic feedback facilitated the identification of intrinsic response-produced cues which subsequently aided the control of these internal stimuli. A corollary to this suggestion is that if the amount of information is increased concerning the effects of an internal response, the discrimination and control of afferent feedback from that response would be enhanced. In establishing a differential reinforcement schedule to eliminate, as an example, hypertensive symptoms, it would be advisable, therefore, to optimize the feedback information.

There are two ways in which feedback information could be increased. First, increase the number of feedback stimulus presentations, and second, increase the amount of information per presentation. The first approach was utilized in Experiment II by providing subjects with two forms of extrinsic feedback of systolic blood pressure changes (manometer dial and counter). The second method is perhaps better since the subject can receive the same amount or more information without the added distractions introduced by increasing the number of feedback stimuli. This latter technique, however, requires an understanding of the physiological mechanisms which mediate the required response change. Specifically, visceral organs are functionally related to one another so that a particular visceral response may occur due to a change in a variety of other response systems. The cardiovascular system provides a particularly good example of this interactive behavior. Being a closed fluid system, it responds with a change in one of its response aspects to a change in any other. Thus blood pressure elevations and decrements are mediated by changes in heart rate, diastolic filling pressure, ventricular distensibility, ventricular contractility, aortic distensibility, peripheral vascular resistance, hydrostatic pressure, or any combination of these factors. The provision of optimal feedback information would necessitate making the feedback contingent upon the particular aspect of cardiovascular responding which mediates the desired blood pressure response. Blood pressure is a product of the cardiac output (heart rate × stroke volume)

and the peripheral vascular resistance. Cardiovascular dynamics dictate, however, that the form of the arterial pulse wave will be differentially affected by different aspects of cardiovascular functioning. Thus the two principal determinants of systolic blood pressure changes are heart rate and stroke volume (Hurst and Logue, 1966; Rushmer, 1961). The rate at which arterial pressure falls during diastole is a function of the pressure produced during the systolic interval, the rate of outflow through the peripheral resistances, and the diastolic interval (Rushmer, 1961). Obviously these two sets of determinants are not independent. Although heart rate is a principal determinant of both systolic and diastolic pressure, peripheral vascular resistance must certainly affect systolic pressure indirectly by altering the diastolic filling pressure and consequently the stroke volume. Increases in diastolic pressure would also affect systolic pressure directly by presenting a greater arterial pressure to the arterial pulse wave, thus producing a heightened systolic peak as the pressure pulse wave traversed to the periphery (Hurst and Logue, 1966).

In order to bring about effective systolic blood pressure changes, then the general rule might be to provide extrinsic feedback contingent upon heart rate or stroke volume; and for diastolic pressure changes, to provide extrinsic feedback contingent upon heart rate, systolic blood pressure or peripheral vascular resistance. Although each of these physiological responses may affect blood pressure changes, some of these responses may be more effective in determining blood pressure changes

in certain individuals than in others. Providing extrinsic feedback of the most effective determinants, for the individual, of the required blood pressure response might enhance the development of learned blood pressure control. The extensive work of Lacy and Lacy (1958) demonstrates, in fact, that individuals can order their autonomic patterns of responding into a hierarchy. According to these findings, an individual's autonomic response profile, when presented with a variety of stressful situations, may be characterized by an increased heart rate primarily ("cardiac responder"), a change in peripheral vascular resistance ("vasomotor responder"), an increase in gastric motility ("gastric responder"), etc. Furthermore there is evidence that extant somatic or visceral symptomatology will determine the response mode in which individuals respond to a stressful situation. Malmo and Shagass (1949) monitored responses to painful stimuli in psychiatric patients with a history of either cardiovascular complaints or head and neck pains. These investigators found that heart rate, heart rate variability and respiratory variability in response to a painful stimulus were all greater for the group of psychiatric patients with cardiovascular complaints, while electromyographic activity was greatest for the somatic complaint group. Malmo and Shagass concluded that increased responsiveness to a stressful stimulus was specific to the physiological system associated with the complaint. The results of these studies strongly suggest that in order to provide hypertensive individuals with optimal feedback information, to promote maximal blood pressure

control, extrinsic feedback would have to be chosen not necessarily according to the most obvious symptom (high blood pressure) but according to the underlying physiological response or responses -- characteristic to the individual -- which serve as the mechanism by which the blood pressure change is produced.

In summary, the results of the experiments presented in this dissertation support the potential usefulness of discrimination training and operant control procedures in promoting an increased understanding of the nature by which reinforcement contingencies may direct the acquisition of inappropriate emotional and psychosomatic responses. It was suggested that hypertensive disorders of the psychosomatic type could be effectively controlled by providing individuals with optimal forms of feedback contingent upon the particular aspects of cardiovascular functioning -- characteristic to the individual -- which mediate the observed blood pressure changes.

Throughout this dissertation it has been explicitly and implicitly argued that the principles governing the behavior of the autonomic effectors do not stand in qualitative distinction to those relating to the behavior of the somatic musculature. This entire work has been dedicated to that proposition. The argument is of great importance, however, to the psychological community as a whole, and should therefore, be briefly extended.

Traditionally, the psychological position concerning somatic and autonomic behavior has been that visceral organs provide an insufficiency

of discriminable afferent information to the CNS to permit the discrimination and voluntary control of visceral responses. As discussed above, this position gained impetus from the observation that voluntary control of autonomic effectors was not normally possible. Physiological and anatomical investigations, however, have demonstrated the existence of afferentation not only from particular visceral organs to the CNS, but have found discrete neural pathways which relay afferent and efferent information between those areas in the brainstem which regulate visceral functioning and the motor and somatosensory areas of the cortex. The results of this dissertation and other psychological investigations have demonstrated that voluntary control of autonomically-mediated response processes follow the implementation of appropriate discrimination training and operant control procedures. Thus the distinction which persists in certain areas of the scientific community between somatic and autonomic processes with respect to their status as voluntary control systems is based not upon physiological, anatomical, or psychological evidence, but rather upon the dualistic philosophy which characterized the "scientific" thinking during the middle ages. The perpetuation of this dualistic dilemma has resulted in a great deal of wasted time and confusion in trying to solve the scientifically unproductive questions which it has generated. The mind-body problem has no basis in reality: It speaks to philosophy, but not to science.

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APPENDIX

APPENDIX

NOTE I

INSTRUCTIONS TO SUBJECTS: PULSE DISCRIMINATION AND HEART RATE CONTROL

Pulse Discrimination

All subjects received this initial instruction. Hello and thank you for volunteering to act as a subject for this experiment. In this laboratory we study the relationship between mental and physiological processes. In order to do this, the experimenter will attach a few recording devices like these (Experimenter demonstrates) to the surface of your skin. All of these devices are for recording purposes only and no shocks or other stimuli will be presented through them. Nothing unpleasant is going to happen to you in this experiment and so we would like you to relax as completely as possible. Right now the experimenter will attach these recording devices and when he is through he will tell you more about what we would like you to do today (Transducers are attached to subjects).

The Experimental group received the following instructions. Now let us tell you about this experiment. We would like to see if you can tell when your heart is beating. Hold this button in your left hand (Experimenter demonstrates). As you can see there is a small green light on the wall to your right, and an amber light on the wall to your

left. When the green light comes on, we want you to press the button each time you feel your heartbeat. You do not have to wait until you are absolutely sure that you feel a heartbeat; press the button each time you think you feel your heartbeat. You may be wondering at this point how you are going to accomplish this task. When the amber light comes on, you will hear, through a pair of headphones, a short tone each time your heart beats. During these amber light periods, just listen to the tones and try to discover any sensations or feelings which occur when you hear the tones--that is, when your heart beats. Do not press the button during amber light periods, just attend carefully to the tones. During the green light periods you will not hear any tones, and you should press the button whenever you think you feel your heartbeat. By listening carefully to the tones during the amber light period, you may be able to tell even during the green light periods when your heart beats. Remember to press the button during green light periods whenever you think you feel or sense your heartbeat.

Do you have any questions?

The Control group received the following instructions. Now let us tell you about this experiment. As you can see, there is a small green light on the wall to your right, and an amber light on the wall to your left. The green light will come on and go off three times. The amber light will then come on and go off once. This sequence will be repeated throughout the experiment. During these amber light trials just listen to the tones carefully. When the green light comes on, you

will not hear any tones, but press the button at the rhythm which you remember the tones occurred during the amber light trial. Do you have any questions? All right, now the first three trials will be green light trials and consequently you will not have yet heard the tones and won't know exactly at what rhythm to press the button. So, during these first three green light trials, just press the button at a rhythm which you guess the tones will occur when you hear them. Remember, once you have actually heard the tones during the first amber light period, you should during the following green light periods, press the button at the rhythm which you remember the tones occurred during the amber light trial.

Do you have any questions?

Heart Rate Control

All subjects received these instructions. Today we will do something different. The Experimenter will attach the recording devices again and we will proceed. (Transducers are attached to subjects.)

You will notice that today there are two blue lights, one on the wall to your right, and one to your left. When the light to your right comes on we want you to increase the rate at which your heart is beating. When the light to your left comes on, decrease your heart rate. Through these headphones (Experimenter indicates) you will hear high-pitched tones in your right ear, or low-pitched tones in your left ear. The high-pitched tones indicate to you that your heart is going

faster; the low-pitched tones that your heart is beating slower. Listen to the tones carefully, and try to discover any internal feelings or sensations which occur when your heart is beating faster, or when it is going slower. This information may help you in controlling your heart rate today. There will be times when you will hear no tones at all. Regardless of whether you hear the tones or not, try to increase your heart rate when the right light comes on, and decrease your heart rate when the left light comes on. Do you have any questions?

Before we begin, there is something which is very important for you to know. Do not breathe irregularly, tense your muscles, or move around in any way in your efforts to control your heart rate. Just breathe normally and relax. If you breathe irregularly or tense your muscles, the information we collect today will not have any meaning. All right, let's begin.

NOTE II

Instructions to Subjects: Blood Pressure Control

All subjects received this initial instruction. Hello and thank you for volunteering to act as a subject today. In this laboratory we study people's internal responses. In order to record what is going on inside you, it will be necessary for the experimenter to attach several electrodes and other recording devices to your limbs. All of these devices are for recording purposes only and no shocks or other stimuli will be presented through them. Nothing unpleasant is going to happen

to you in this experiment and so we would like you to relax as completely as possible. These devices will transmit information about activity of your internal organs to an analysis and recording system in the adjacent room. This system will provide us with information we need about your internal activity. If you have no objections now the experimenter will go ahead and attach the recording devices to you. When you are connected up, we will tell you what we want you to do in this experiment. (Transducers are now attached to subject.)

Instructions to experimental subjects. In this experiment we are going to investigate whether or not you can control your blood pressure. People generally believe that they cannot control internal responses of this nature, but our data indicates that many people can.

In order to help you with this task we are going to give you continuous information of your blood pressure. This information will be presented to you on these two instruments (Experimenter indicates manometer dial and electromechanical counter). The higher your blood pressure is, the higher will be the indication on the manometer dial and the faster the counter will count. Conversely the lower your blood pressure is, the lower will be the indication on the manometer dial, and slower the counter will count.

What we want you to do in this experiment is to heighten (Increase group) (lower for the Decrease group) your blood pressure and keep it as high (low for the Decrease group) as possible. You will know how successful you are in achieving this goal by watching the dial

and counter. The faster (slower for the Decrease group) the counter counts and the higher (lower) the dial reading, the higher (lower) will be your blood pressure. The experimental session will be divided into fifty-second trials with a rest period of about 30 seconds occurring between each trial. At the beginning of each trial a bright light on the front of this feedback display (Experimenter indicates) will come on and stay on for the duration of the trial period. At the end of the trial period, the light will go off. Only during trial periods will the dial and counter operate, and only during these periods should you attempt to control your blood pressure. So remember, your task is to keep the dial reading as high (low) as possible and to produce a higher (lower) counter total on each successive trial period. At the end of each trial the bright light on the feedback display will go off and the display will be illuminated only by a dim light. When this happens the dial reading will go back to zero and the counter will stop counting. At this point we want you to make a mental note of the counter total and then to reset it by pressing this button (Experimenter indicates). By making this mental note of the counter totals, you can make a trial by trial comparison of how you are doing in your attempts to heighten (lower) your blood pressure.

The question which probably immediately comes to mind is "How do I control the counter and the pointer on the dial?" We cannot tell you how to accomplish this task. We shall however tell you this: you are <u>not</u> to engage in abnormal breathing in your attempts to control the

counter or dial pointer. Also please do not move or tense your muscles. Muscle movement and abnormal breathing will interfere with our recordings and will be immediately detected. We want you to control the counter and the dial by "mental processes" only. We realize that this sounds like an impossible task but many people who we have run through this procedure have been very successful in controlling the counter and the pointer on the dial. Use the information about your blood pressure presented to you on the counter and dial to help you in learning to heighten (lower) your blood pressure. When the counter and the dial are operating, concentrate carefully on any feelings or sensations which occur to you when the reading on the dial and the counter total increases (decreases)--that is, when your blood pressure is going up (down). In this manner, you may be able to identify those feelings inside of you which occur when your blood pressure increases (decreases), learn to control them yourself, and thus be better able to increase (decrease) your blood pressure as the experiment proceeds.

Instructions to control subjects. This experiment is designed to investigate your cardiovascular behavior; that is your blood pressure and the behavior of your heart and blood vessels. In the experiment you are required to do very little. We would like you to just lie there as comfortably and as quietly as possible while we make our recordings of what is going on inside of you.

The experimental session will be made up by a number of recording trials. Each of these trials will last fifty seconds and between each

trial a period of about thirty seconds will occur. When a recording trial begins, a bright light fixed to this display will come on, the pointer on this dial move and this counter will operate (Experimenter indicates). At the end of the trial the bright light will go off and the display will be illuminated by a dim light only. When this happens we would like you to make a mental note of the counter total and reset the counter by pushing this button (Experimenter indicates). At the end of the session we would like you to give us a rough idea of the counter totals and the meter indication. This isn't a test--we just need a rough check of the range in which each of these instruments is operating. So remember you are to lie here as quietly as possible throughout the experimental trials and to reset the counter at the end of each trial. We ask you not to move or to engage in abnormal breathing during the recording trials because these activities will interfere with our measurements and will be immediately detected on our instruments.

TABLE I

Mean percent above chance pulse discrimination scores for the Experimental and two Control Groups on each session for each subject.

Subject	Session I	Session II	Sum	Mean
		EXPERIMENTAL GROUP		
1	5.99	17.29	23.28	11.64
2	9.91	22.41	32.32	16.16
3	8.99	8.57	17.56	8.78
4	10.96	7.29	18.25	9.13
5	4.64	6.46	11.10	5.55
6	16.79	36.84	53.63	26.82
7	6.96	8.80	15.76	7.88
Sum	64.24	107.66	171.90	-----
Mean	9.15	15.38	-----	12.28
		REGULAR CONTROL GROUP		
1	3.93	2.80	6.73	3.37
2	7.49	8.24	15.73	7.87
3	4.81	5.34	10.15	5.08
4	2.93	4.57	7.50	3.75
5	4.94	3.96	8.90	4.45
6	4.03	3.03	7.06	3.53
7	7.24	7.41	14.65	7.33
Sum	35.37	35.35	70.72	-----
Mean	5.05	5.05	-----	5.05
		IRREGULAR CONTROL GROUP		
1	5.73	2.43	8.16	4.08
2	2.53	3.09	5.62	2.81
3	4.64	2.29	6.93	3.47
4	4.83	3.49	8.32	4.16
5	2.36	2.27	4.63	2.32
6	3.33	2.53	5.86	2.93
7	3.07	2.33	5.40	2.70
Sum	26.49	18.43	44.92	-----
Mean	3.78	2.67	-----	3.21

TABLE II

Mean heart rate difference scores (bpm) for two trial pairs within each first No-Feedback (NF-1), second No-Feedback (NF-2), first Feedback (FB-1), and second Feedback (FB-2) block for each subject in the Experimental and Control Groups.

Subject	NF-1	NF-2	Sum	Mean	FB-1	FB-2	Sum	Mean
				EXPERIMENTAL GROUP				
1	5.5	0.0	5.5	2.8	3.0	6.5	9.5	4.8
2	13.0	11.0	24.0	12.0	13.0	15.5	28.5	14.3
3	8.5	13.0	21.5	10.8	12.0	13.0	25.0	12.5
4	2.5	11.0	13.5	6.8	14.5	9.5	24.0	12.0
5	3.5	9.5	13.0	6.5	4.5	10.0	14.5	7.3
6	3.0	3.0	6.0	3.0	5.0	3.0	8.0	4.0
7	0.5	6.5	7.0	3.5	3.0	9.0	12.0	6.0
Sum	36.5	54.0	90.5	----	55.0	66.5	121.5	----
Mean	5.2	7.7	----	6.5	7.9	9.5	-----	8.7
				IRREGULAR CONTROL GROUP				
1	10.2	5.1	15.3	7.7	8.8	4.1	12.9	6.5
2	0.3	1.0	1.3	0.7	- 0.4	0.9	0.5	0.3
3	3.1	0.4	3.5	1.8	1.9	1.0	2.9	1.5
4	13.6	9.1	22.7	11.4	9.9	12.1	22.0	11.0
5	1.3	- 1.7	- 0.4	- 0.2	3.7	- 0.6	3.1	1.6
6	7.6	4.1	11.7	5.9	4.8	6.9	11.7	5.9
7	4.3	3.9	8.2	4.1	5.6	7.4	13.0	6.5
Sum	40.4	21.9	62.3	----	34.3	31.8	66.1	----
Mean	5.8	3.1	----	4.5	4.9	4.5	----	4.7

TABLE III

Mean systolic blood pressure difference scores (training trial minus basal values in mm. Hg) for Increase and Decrease Groups. Mean absolute systolic blood pressure scores (mm. Hg) for Control Group. Values are for each training trial on each session.

	Increase Group		Decrease Group		Control Group	
Trials	S1	S2	S1	S2	S1	S2
1	27.4	28.6	0.2	7.7	136.8	123.6
2	17.8	23.5	- 3.4	5.5	135.9	127.4
3	10.7	31.8	- 4.2	4.9	135.2	123.6
4	12.0	19.2	- 2.2	2.6	134.1	133.2
5	28.1	21.0	- 7.6	-0.2	130.3	123.5
6	18.3	21.6	- 7.6	0.9	133.2	127.2
7	16.1	32.0	- 6.4	0.6	131.8	124.4
8	15.3	21.8	- 8.8	-1.0	137.9	131.8
9	18.0	22.4	- 8.3	-2.8	139.6	131.6
10	15.4	23.3	-10.6	-0.6	136.6	126.7
11	15.0	27.0	-15.6	-3.4	132.1	125.8
12	13.0	21.1	-11.3	-3.6	138.5	130.3
13	12.6	20.0	-12.8	-5.7	139.8	130.5
14	17.5	20.8	-16.2	-3.4	140.1	131.8
15	19.0	22.0	-10.9	-3.1	138.6	128.0
16	15.4	23.3	-11.1	-1.0	139.6	123.7
17	17.1	21.1	-13.1	-5.2	139.3	122.3
18	15.6	20.3	-13.6	-3.8	134.9	127.6
19	15.8	18.8	-15.2	-4.0	133.1	132.4
20	18.8	20.2	-16.6	-4.9	137.6	127.0

TABLE IV

Mean systolic blood pressure difference scores (training trials minus basal in mm. Hg) for Increase and Decrease Groups. Mean absolute systolic blood pressure scores (mm. Hg) for Control Group. Values are for each subject on each session.

Subjects	Increase Group		Decrease Group		Control Group	
	S1	S2	S1	S2	S1	S2
1	11.8	20.1	-11.8	3.9	136.0	128.6
2	1.9	6.2	- 4.2	-5.6	177.0	120.8
3	4.0	16.4	- 2.7	8.5	116.7	142.1
4	30.8	13.7	-14.4	-4.9	114.6	121.2
5	36.2	58.3	-15.8	-7.0	137.0	125.4

TABLE V

Mean heart rate (beats per 50 seconds) for blood pressure Increase, Decrease, and Control Groups on each session for each training trial.

Trials	Increase Group		Decrease Group		Control Group	
	S1	S2	S1	S2	S1	S2
1	72	74	70	64	70	59
2	71	70	69	65	68	62
3	70	73	68	65	67	63
4	66	71	68	63	64	62
5	69	68	69	64	69	58
6	67	68	66	62	66	59
7	70	70	63	62	66	60
8	69	70	64	58	66	60
9	67	68	63	61	66	61
10	66	68	62	59	67	58
11	70	69	65	59	64	57
12	65	68	62	60	63	60
13	65	65	62	60	65	57
14	66	63	60	60	64	56
15	64	63	59	59	63	61
16	64	64	60	59	63	57
17	65	63	60	59	65	58
18	64	61	59	60	64	58
19	63	63	59	60	63	60
20	66	61	60	59	63	58

TABLE VI

Mean heart rate (beats per 50 seconds) for blood pressure Increase, Decrease, and Control Groups on each session for each subject.

Subjects	Increase Group		Decrease Group		Control Group	
	S1	S2	S1	S2	S1	S2
1	61	66	66	56	68	57
2	62	66	65	69	65	60
3	67	83	65	54	72	62
4	64	72	47	54	56	55
5	82	49	74	70	66	62

VITA

Roger Alan Kleinman was born in Brooklyn, New York on December 9, 1942, but moved to Los Angeles before entering school. He was matriculated through grammar, junior, and senior high schools in the Los Angeles area, and promptly embarked upon the collegiate advanture.

In 1965, the University of California endowed him with all the rights and privileges attendant to a Bachelor of Arts degree with a major in Psychology. Thus privileged, he was accepted by and entered as a graduate student at the University of Tennessee, Knoxville. While a graduate student, Kleinman worked as a Research Assistant for Dr. Jasper Brener, who with the help of Bertrand Russell, accomplished a major revision in his life style. He also served as a Graduate Teaching Assistant and Instructor.

Upon completion of his thesis research concerned with operant thermo-regulatory behavior in a cold environment, Kleinman received a Master of Arts degree in 1968. The Ph.D. was earned for the present work in 1970.