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To the Graduate Council:

I am submitting herewith a dissertation written by David Hothersall entitled "Operant Conditioning of Heart Rate Changes in Curarized Rats with Brain Stimulation Reinforcement." I recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Psychology.

Major Professor

We have read this dissertation and recommend its acceptance:

Accepted for the Council:

Vice Chancellor for
Graduate Studies and Research

OPERANT CONDITIONING OF HEART RATE CHANGES IN CURARIZED RATS WITH BRAIN STIMULATION REINFORCEMENT

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

David Hothersall

August 1968

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Finally I would like to thank my wife, Lesley.

ABSTRACT

Many learning theorists have distinguished between autonomic responses which can be conditioned using classical conditioning procedures, and skeletal responses which can be conditioned using operant or instrumental conditioning procedures. In recent years this distinction has been challenged since such autonomic responses as changes in heart rate, GSRs, vasoconstriction and vasodilation, and changes in blood pressure have all been conditioned as operants.

The aim of this study was to investigate the possibility that both increases and decreases in heart rate could be conditioned in groups of curarized rats using an operant conditioning procedure with brain stimulation as the reinforcement for the heart rate changes. Curarized rats were used in order to markedly reduce the possibility of skeletal mediation of the heart rate changes which were conditioned. Brain stimulation was used since it has been found to have powerful reinforcing effects for more typical operant responses. A feedback stimulus was presented to the rats during the curare conditioning sessions whenever they were emitting criterion heart rates, i.e. heart rates which if sustained for a fixed number of beats would be followed by the brain stimulation. Two groups of six rats were used, one group being reinforced for heart rate increases, and one for decreases. All rats were given four curare conditioning sessions during which heart rate changes were reinforced.

During the first curare conditioning session no evidence of operant conditioning of heart rate changes was found. On the three

later conditioning sessions significant differences were found in the heart rates of the two groups, with the group reinforced for heart rate increases showing the higher heart rate. There was also some evidence of an extinction effect during the later curare sessions in that the heart rates of the two groups which diverged during conditioning, tended to converge during extinction. The results of this experiment show that changes in heart rate can be conditioned in curarized rats using an operant conditioning procedure with brain stimulation as the reinforcement for the heart rate changes.

In a second experiment three rats were used in an attempt to first condition a heart rate change in one direction, and then to reinforce heart rate changes in the opposite direction. Although the three rats showed good acquisition of the initial heart rate change none of them showed any evidence of acquisition of the heart rate change in the opposite direction. The results from these three rats suggest that heart rate reversals will only be conditioned with great difficulty.

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INTRODUCTION

Traditionally many learning theorists have held that instrumental or operant conditioning procedures are effective only in the modification of skeletal responses under the control of the somatic nervous system, and not in generating control over the smooth muscles, cardiac muscles and glands. In short responses under the control of the autonomic nervous system are thought not to obey the law of effect.[1] Konorski and Miller (1937), Schlosberg (1937) and Skinner (1938) all distinguished between skeletal responses which can be conditioned as operants, and autonomic responses, which can only be conditioned using classical conditioning procedures. For over twenty years this distinction was widely accepted (Mowrer, 1950; Keller and Schoenfeld, 1950; Solomon and Wynne, 1954; Morgan, 1965). Bugelski (1956), in discussing the experiments of Pavlov on classical conditioning, and of Skinner and Mowrer on operant and instrumental conditioning, wrote:

> For Mowrer and Skinner, Pavlov might account satisfactorily for the learning of autonomic nervous system responses...but for any behavior involving essentially the whole organism, some kind of reward learning is presumed (Bugelski, 1956, p. 68).

The clearest statement of this distinction was made by Kimble (1961)

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[1]The first clear statement of the law of effect is to be found in Thorndike (1911, p. 24). He proposed that responses which have satisfying consequences are strengthened and those followed by discomfort or annoyance are weakened. More recent statements of this law differ from Thorndike's original one in that other terms (reward, reinforcement, punishment) have been substituted for satisfaction and annoyance.

in his text on Conditioning and Learning. Kimble stated:

> Thus for autonomically mediated behavior, the evidence points unequivocally to the conclusion that such responses can be modified by classical, but not instrumental, training methods (Kimble, 1961, p. 100).

The validity of this long-standing distinction has been challenged, especially in recent years, by the results of a number of experiments which apparently demonstrate operant conditioning of autonomic responses.[2] The general procedure used in these experiments has been to place a reinforcing stimulus under the control of an autonomic response such as heart rate, vasodilation or vasoconstriction, the galvanic skin response, or intestinal motility. Using this operant conditioning procedure systematic changes in the frequency of occurrence of these autonomic responses have been observed. However, some other investigators have reported negative results from attempts to condition autonomic responses as operants.[3] Still other investigators have challenged the 'positive' results claiming that they do not in fact demonstrate operant conditioning of autonomic responses, but conditioning of some other aspect of the organism's behavior which in turn elicits the autonomic changes which have been observed. Such critics believe that the original distinction between classical and operant conditioning in terms of

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[2]The results of these experiments will be presented and discussed in Chapters I and II of this dissertation.

[3]The results of these experiments will be presented and discussed in Chapters I and II of this dissertation.

the responses to which these procedures can be applied retains its validity, and that all of the results seemingly showing operant conditioning of autonomic responses can be accounted for in terms of the conditioning of some form of mediating response. The foremost proponent of this point of view has been Kendon Smith. In 1954 he formulated his mediation or artifact hypothesis which states that only skeletal responses can be conditioned and:

> . . . every 'conditioned visceral response' is in reality an artifact, an innate accompaniment of the skeletal responses inculcated by the conditioning process (Smith, 1954, p. 217).

Smith recently made this point even more strongly in his supplementary comments on his paper 'Conditioning as an Artifact'. He stated:

> The broad idea that autonomic conditioning does not really exist and that it is merely counterfeited by somatic learning is suggested so strongly . . . that there seems little distinction in being the one to formalize it (Smith in Kimble, 1967, p. 100).

Briefly stated the aim of the experiments reported in this dissertation was to demonstrate operant conditioning of an autonomic response under conditions where the possibility that the autonomic changes were 'counterfeited by somatic learning' was minimized.

There are at least two positions which might be adopted in replying to the proponents of the mediation or artifact hypothesis.

1. Results showing, for example, operant conditioning of heart rate changes might be regarded as of interest and significance without any consideration of the manner in which the changes in heart rate were actually produced. The important thing, it might be claimed, is that such results show that changes in heart rate can be conditioned in

accordance with the principles of operant conditioning. How such conditioning is mediated might be regarded as of little or no interest. An analogy might be drawn between this and the situation in a Skinner box in which the experimenter is rarely interested in, or even aware of, the manner in which the rat actually presses the lever. What is of interest is how the rate of lever pressing can be controlled, not how the response is made. For the present investigator such a position, though methodologically defensible, does not seem satisfactory. The analogy between a discrete and arbitrary response such as lever pressing and a continuous response such as heart rate is tentative. In addition due to the controversial nature of results showing operant conditioning of changes in heart rate, it seems that the onus should rest on the investigator to consider the possibility of skeletal mediation of the autonomic changes which he has obtained, before claiming that operant conditioning of an autonomic response has been demonstrated.

2. The position which was adopted in planning the experiments reported in this dissertation was to give close consideration to the arguments proposed by mediation theorists, and to specifically consider the behaviors which could possibly mediate changes in the response being studied. Such a consideration led to an attempt to eliminate, or at least to markedly reduce, the possibility of such mediation and thus, at least in the ideal case, to the study of operant conditioning of an autonomic response isolated from possible mediating behaviors. However, in adopting this position the investigator is faced with the unhappy task of attempting to establish a negative proposition, i.e., that operant

conditioning of an autonomic response has occurred in the absence of skeletal mediation. Logically such negative propositions have a dubious status and just how impregnable they can be is shown by the exchange of papers between Black and Lang (1964) and Smith (1964). In spite of this difficulty in the experiments reported in this dissertation an attempt was made to at least reduce the possibility of skeletal mediation of the autonomic changes which were conditioned by using curarized Ss.

In the experiments reported in this dissertation both increases and decreases in heart rate have been conditioned in curarized rats using an operant conditioning procedure. Black (1965) termed the heart a 'promiscuous organ' since it is subject to so many different influences. Heart rate is influenced by[4]:

(a) respiration - heart rate increases during inspiration and decreases during expiration (sinus arrythmia);

(b) skeletal tension;

(c) blood pressure level - under resting conditions there is normally an inverse relationship between blood pressure level and heart rate. This inverse relationship is sometimes described as Marey's law of the heart;

(d) oxygen deficiencies - anoxia leads to acceleration of heart rate (anoxic tachycardia);

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[4]This information is taken from pages 123 and 124 of 'Samson Wright's Applied Physiology', 11th edition by Cyril A. Keele and Eric Neil.

(e) endocrine factors;

(f) temperature - a rise in body temperature leads to an increase in heart rate.

In curarized rats the possibility of changes occurring in respiration and skeletal tension is at least considerably reduced.

Having decided to attempt to operantly condition changes in heart rate in curarized rats one further decision which had to be made concerned the reinforcer to be used in these experiments. Due to the nature of the curarized preparation the number of potential reinforcers is extremely limited. It was decided to use positively reinforcing brain stimulation since this reinforcer had a number of definite advantages. It could be made contingent on heart rate changes in a reliable and controlled manner, thus eliminating any delay between the change in heart rate and the delivery of the reinforcement. Secondly it seems desirable that powerful reinforcers be used in attempts to condition autonomic responses as operants. Brain stimulation has been found to yield powerful reinforcing effects in a number of experiments in which typical operant responses have been conditioned (Olds, 1958; Valenstein and Beer, 1964). All of the rats used in the experiments reported in this dissertation were first trained to lever press for brain stimulation, and this same intensity of stimulation was subsequently used to reinforce heart rate changes during the curare conditioning sessions.

A second important feature of the experiments reported in this dissertation was that a feedback stimulus was presented during the

conditioning phase of each curare conditioning session. This stimulus was contingent on criterion heart rates, i.e., heart rates which if sustained for a fixed number of beats would produce the brain stimulation reinforcement. This feedback stimulus was used since the results of a number of previous experiments (Lisina, 1961; Hnatiow and Lang, 1965; Lang, Sroufe and Hastings, 1967; Brener and Hothersall, 1966, 1967) have shown the importance of feedback for the modification and control of autonomic responses.

A third feature of this experiment was that the animals were given repeated curare conditioning sessions. This was done since it was felt that extent and length of training might be an important factor in the development of autonomic responses as operants. The alternative to using repeated curare conditioning sessions would have been either:

1. To use a large dose of curare which would have maintained the Ss in a completely paralyzed condition for a longer period of time, thus allowing the conditioning phase of the curare session to be prolonged. This procedure was not used in the experiments reported in this dissertation since Black (1967) has shown that large doses of d-tubocurarine have a direct effect on the neural innervation of the heart. In three deeply curarized dogs Black found that cardiac responses to direct vagal stimulation were markedly attenuated. At medium and light levels of curarization the cardiac response to this stimulation was recovered.

2. A second procedure which could have been used would have been to infuse curare at a slow rate throughout the curare conditioning session

by means of a venous cannula and an infusion pump. This technique would allow a constant level of curarization to be maintained throughout the curare session.

The repeated curare conditioning sessions which were used allowed a study of the development over sessions of the heart rate changes as operant responses. In addition this procedure allowed an assessment of the 'extinction' effect within each session, and the carry-over of the conditioned heart rate changes from one session to the next.

Finally I investigated the possibility that both increases and decreases in heart rate could be conditioned in the same rat under different stimulus conditions. A number of different procedures were used, and the results of the application of these procedures are reported.

CHAPTER I

OPERANT CONDITIONING OF CHANGES IN HEART RATE

Harwood (1962) published the first report of an experiment concerned with operant conditioning of changes in heart rate. He used human Ss and attempted to condition heart rate decreases. If a cardiac deceleration occurred in the presence of a blue light a nickel was given to the S. Periods of light and no light alternated with reinforcement only being given in the presence of the light. No evidence of operant conditioning of decreases in heart rate was found in comparisons of heart rates during periods when the light was and was not on. Harwood pointed out that the method which he had used to monitor and record heart rate had resulted in a variable delay between the occurrence of a cardiac deceleration - the operant response - and the delivery of the reinforcement. The average length of these delays was three seconds. It seems likely that with a continuous response such as heart rate delays of this magnitude could have resulted in the reinforcement of a heart rate response other than the one being conditioned. In view of this technical weakness the results of his experiment can not be regarded as in any way conclusive.

The first major attempt to condition heart rate as an operant was reported in 1960 in a doctoral dissertation by Shearn. A brief report of this experiment was published in 1962 (Shearn, 1962). Shearn used a Sidman avoidance procedure in which temporary heart rate accelerations avoided an electric shock. Yoked control Ss were paired with the

experimental Ss. The experimental Ss showed an increase in the number of temporary accelerations in heart rate, while the control Ss showed a decrease. Although this result might be regarded as evidence of operant conditioning of a change in heart rate, there was no decrease in the number of shocks received by the experimental Ss - one criterion of successful avoidance conditioning. In addition all of the Ss, both experimental and yoked control, showed an overall decrease in heart rate during the experimental session.

Brener (1966) used an escape avoidance procedure in an attempt to condition changes in heart rate as an operant. In his first experiment low heart rates, within the repertoire of the S, served to terminate or avoid an electric shock. In the second experiment the effective response was an increase in heart rate. Yoked control Ss were used. Significant heart rate differences were found between the experimental and yoked control Ss in both these experiments. These differences were consonant with predictions based on the principles of operant conditioning. Brener found it easier in these experiments to condition heart rate decreases. An important feature of the procedure of these experiments was the use of visual feedback presented to the Ss when criterion heart beats were emitted. Brener concluded that both increases and decreases in heart rate can be conditioned as operants. The results of these two experiments presented the first clear challenge to the widely held belief that autonomic responses can not be conditioned as operants.

Frazier (1966) used a discriminated avoidance conditioning technique with human Ss. During training ten twenty minute 'epochs', during which a light was present and punishment was available (S^D) periods, alternated with no light periods during which the contingencies were not in effect (S^Δ). If during successive minutes of the 'epoch' S's heart rate did not increase, at the end of the period he was given an electric shock. Frazier attempted to achieve the goal of 'conditioning without awareness' by giving the S a monitoring task to perform during the conditioning period. This goal appears to have been met in that none of the Ss reported that they realized that a change in heart rate was the effective response. In acquisition a steady increase in heart rate was found. In addition there was a maintenance of a high heart rate during the 'epoch' and a return to a lower heart rate during the S^Δ period. During the last training period the light was presented alone without any shocks, but the Ss were not told that they would not receive shocks during this period. Sustained high heart rates were observed. Frazier claims that this demonstrates that the changes in heart rate during the S^D periods were due to the conditioning and were not due to some type of sensitizing effect of the electric shocks. Frazier reports that clear increases in heart rate were found during the S^D periods, but they were not accompanied by parallel changes in respiration rate or amplitude. Frazier was also able to demonstrate what he termed 'cardiovascular oscillation'. Following training repeated presentations of the S^D stimulus elicited clear heart rate increases, with the S's heart rate decreasing when the stimulus was withdrawn. These results represent an

impressive demonstration of stimulus control over changes in heart rate. It would be interesting to see whether similar results could be obtained in an experiment in which a similar procedure was used but with a decrease in heart rate as the effective response. As yet the results of such an experiment have not been published.

Engel and Hansen (1966) used human Ss and reinforced them on a beat to beat basis for changes in heart rate. If the S's heart rate decreased a light in the S's room was switched on and a timer which the S could see began to run. The light served as a cue for the S and the sight of the timer running was assumed to be reinforcing since the S was later paid a sum of money proportional to the elapsed time shown on the timer. In this experiment five of the ten Ss learned to slow their hearts under these conditions. Four of the five non-learners correctly inferred that a decrease in heart rate was the effective response and so it appears that, at least in this experiment, Ss were better able to modify their heart rates when they were 'unaware' that heart rate changes were actually being conditioned.

In a second experiment Engel and Chism (1967) used a similar technique in an attempt to condition heart rate increases. In this experiment all five of the Ss learned to increase their heart rates. Since all of the Ss acquired the response in this second experiment, whereas only 50 percent of the Ss acquired the response in their earlier experiment Engel and his co-workers concluded that it is easier to condition heart rate increases as an operant than it is to condition heart rate decreases. This conclusion is in opposition to the results

reported by Brener (1966). Two of the Ss in Engel and Chism's experiment were observed to increase their respiration rates, and so it seems that at least in these Ss a somatic manoeuvre had been employed. It is interesting that when two of the Ss who had successfully increased their heart rates were asked how they had achieved control over the light and the timer, they reported that they had tried to relax completely, a form of behavior which might have been expected on a priori grounds to have led to a decrease rather than to an increase in heart rate. Engel and his co-workers concluded on the basis of the results of these two experiments that human Ss can learn to both increase and decrease their heart rates, and that the techniques which are used to regulate heart rate vary widely from one S to another.

Human Ss were used in all of the experiments which have been presented thus far. There have been six reports of experiments on operant conditioning of heart rate changes in which animal Ss have been used. Shearn and Clifford (1964) used rabbits and recorded frequency distributions of inter heart beat intervals (IBIs) under constant conditions and with no experimental contingencies in effect. Within sessions they found that the IBI distributions tended to drift towards the longer IBIs, i.e., there was a decrease in heart rate. During later sessions different Ss were shocked whenever they emitted either a long or a short IBI. The rabbits shocked for short IBIs became 'trapped' and continued to emit short IBIs throughout the session. Consequently there was no decrease in the number of shocks

they received. The Ss shocked for long IBIs oscillated between short and long IBIs. These results are contrary to what might have been expected on the basis of the principles of operant conditioning. The results of this experiment provide little evidence of operant conditioning of a change in heart rate.[1]

Black (1967) reported the results of a series of experiments on operant conditioning of changes in heart rate in curarized dogs. In the first of these experiments two groups of dogs were used, one of which - the pretraining group - was operantly conditioned during the first phase of the experiment to press a pedal when a Conditioned Stimulus (CS) was presented in order to avoid a shock; the other group was simply given a series of presentations of the CS alone. In the second phase of the experiment all of the dogs were curarized and each group divided into two sub-groups one of which was reinforced for heart rate increases and one for heart rate decreases. A six second CS - UCS interval was used and if a criterion heart rate response occurred during this interval S avoided the electric shock. If such a response did not occur the shock was presented at the end of the interval. During the second phase of the experiment there was a significant increase in the number of

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[1]Miller and Carmona (1967) and Miller and DiCara (1967) suggest that the results of this experiment, together with the results of a number of others, seriously challenge the view that visceral responses can not be conditioned as operants. Inclusion of Shearn and Clifford's experiment in a list of experiments giving positive results from attempts to condition autonomic responses as operants does not seem justified.

successful avoidance responses made by both the increase and decrease groups, indicating that both increases and decreases in heart rate could be conditioned in curarized dogs. However increases in heart rate were found to be easier to condition than decreases. A non-significant difference favoring the dogs given pre-training in the pedal pressing situation was found. It is interesting that although the responses of the dogs in the increase and decrease groups during the CS - UCS interval were similar, an increase in heart rate followed by a decrease, they were very differnt in form: the duration of the increase in the dogs trained to increase their heart rates was significantly longer than the duration of the increase in dogs for whom the avoidance response was a decrease in heart rate. In later experiments Black used different levels of curarization, light, medium and deep. He found that operant conditioning of heart rate changes occurred more readily at the light level of curarization, than it did at either of the other two. In addition, conditioned cardiac responses which had been formed under light levels of curarization were found to be attenuated when they were transferred to medium or deep levels of curarization. Black suggests that this may have been due to the fact that with large doses of curare there may be a direct blocking of the vagus. Support for this suggestion came from results obtained by Black showing that in a deeply curarized dog the cardiac response to direct electrical stimulation of the vagus was weakened. In general the results of Black's experiments provide a convincing demonstration of operant conditioning of both increases and

decreases in heart rate, under conditions in which the possibility of skeletal mediation of the cardiac changes had been, at least, markedly reduced.

Recently Miller and his students and co-workers have reported the results of a number of experiments on operant conditioning of heart rate changes in curarized rats. In three of these experiments positively reinforcing brain stimulation has been used as the reinforcer for the heart rate changes. Trowill (1967) used rats which had previously been trained to lever press for brain stimulation. While completely paralyzed and artifically respirated these rats received brain stimulation on a fixed interval schedule of reinforcement for either relatively slow or relatively fast heart rates. Fifteen of 19 Ss reinforced for fast heart rates increased their rates, and the mean increase for all 19 Ss was 18 beats per minute (b.p.m.); fifteen of 17 Ss reinforced for slow heart rates decreased their rates, and the mean decrease was 19 b.p.m. The difference in the heart rate changes shown by the two groups was highly significant. Yoked control Ss were used in this experiment and a rather disconcerting feature of the results was that the yoked groups showed heart rate changes which were opposite to those of their experimental partners, and hence to each other. Trowill was unable to find any reason for this result and concluded that it may have been due to chance.

Since the heart rate changes conditioned in Trowill's experiment were relatively small, Miller and DiCara (1967) working in the same laboratory conducted a second experiment to see whether larger changes in heart rate could be conditioned. They attempted to 'shape' the heart

rate response by progressively shifting rats to a more difficult criterion heart rate after they had learned to meet an easier one. In addition they used a 20 second Time Out period to allow the rat's heart rate to recover from the effects of the brain stimulation. Two groups of rats were conditioned to either increase or decrease their heart rates while curarized in order to receive the positively reinforcing brain stimulation. Eleven of 12 rats reinforced for increases in heart rate progressively increased their heart rates from an initial rate of 422 to a final one of 510 b.p.m. Ten of the 11 rats reinforced for decreases in heart rate decreased their heart rates from an initial rate of 400 to a final rate of 316 b.p.m. The initial difference in the mean heart rates of the two groups did not approach significance, but the difference at the end of conditioning was highly significant. Since the heart rate changes in the two groups were in different directions they could not be accounted for as due to the unconditioned effects of the brain stimulation. Throughout conditioning Miller and DiCara report that all skeletal responses were completely blocked by the curare.

In a second experiment a discrimination procedure was used with rats being reinforced for either increases or decreases in heart rate in the presence of one stimulus, but no reinforcement being delivered in the absence of the stimulus. The two groups of curarized rats acquired the discrimination of either increasing or decreasing their heart rates in response to the stimulus.

Miller and Banuazizi (1968) reinforced deeply curarized rats maintained on artificial respiration with brain stimulation for either increases or decreases in heart rate. The group reinforced for decreases progressively decreased its heart rate from an average of 425 b.p.m. at the beginning of training to an average of 373 b.p.m. at the end. The group reinforced for increases progressively increased its heart rate from an average of 440 b.p.m. at the beginning of training to an average of 468 b.p.m. at the end.

DiCara and Miller (1968) investigated the possibility of reinforcing changes in heart rate by escape from or avoidance of electric shock. Two groups of curarized rats were used. If on shock trials the criterion heart rate - either a decrease or an increase depending on the S's group membership - was achieved within 5 seconds of the onset of the shock signal the signal was terminated and no shock was delivered. If the S failed to meet the criterion heart rate the shock was presented. As soon as the criterion heart rate was achieved the shock and the signal were terminated. In addition an equal number of non-shock trials were given during which a second non-shock signal was presented for 5 seconds irrespective of the S's heart rate. This signal of course was never followed by an electric shock. Throughout training heart rate in the presence of the shock signal increased in the group reinforced for heart rate decreases. The differences in the average heart rates of the two groups in the presence of the shock signal were statistically significant. In addition there was some evidence that the no-shock stimulus elicited changes in heart rate opposite to those elicited by the shock stimulus.

In this experiment two extremely useful procedures were used by DiCara and Miller. The curare was infused at a slow but constant rate throughout the experimental session thus allowing much more precise control over the level of curarization which was actually achieved. Secondly electromyographic (EMG) recordings were taken from the lateral surface of the right gastrocnemius. Analysis of the EMG records for each S at a sensitivity of 150 micro-volts/centimetre showed that within 15 minutes of the curare injection all skeletal muscle activity ceased. The first minute signs of muscle activity only appeared one hour after the end of training. DiCara and Miller concluded:

> In the present experiment we have shown that changes in heart rate can be achieved by instrumental training techniques when the animals are so deeply curarized that muscle action potentials are completely abolished (DiCara and Miller, 1968, p. 11).

This series of experiments by Miller et al. has provided convincing evidence of operant conditioning of changes in heart rate in curarized rats using both brain stimulation and escape from, or avoidance of, electric shock as the reinforcement for the heart rate changes. However there is a great need for independent replications of these experiments.

Operant conditioning procedures were not specifically used in the final two sets of experiments I would like to present in this Chapter. However the procedures and results used in these experiments are relevant to the present discussion.

Hnatiow and Lang (1965) reported the results of an attempt to train human Ss to reduce the variability of their heart rates. No aversive stimuli were used in this experiment and S was simply instructed to try to keep his heart rate as steady as possible while observing a visual display which was synchronized with his heart rate. Ss were instructed to try to keep the pointer in this display in the target area in the center of a scale, and the only reinforcement used was the S's perception of his success or failure. Control Ss watched a display which was unrelated to their own heart rate variability and which merely simulated cardiac changes. In experimental Ss the total time spent within the target area was significantly greater during display than it was during non-display periods. In control Ss these differences were non-significant. In experimental Ss the amount of time spent on target increased progressively throughout training. Control Ss showed no such improvement. For the experimental Ss no significant correlations were obtained between measures of heart rate variability and any measure of respiration change.

Lang, Sroufe, and Hastings (1967) used a procedure similar to that of the earlier experiment. Five minute display (feedback) periods alternated with five minute non-display periods. Pairs of experimental and yoked control Ss were run. Both Ss were instructed to attempt to keep the pointer in the center of the dial. The position of the pointer was determined by the heart rate variability of the experimental S, and so the yoked control S received feedback which was determined by his

partner's, rather than by his own, heart rate variability. The experimental Ss learned to significantly reduce their heart rate variability, whereas the control Ss did not show any significant reduction in heart rate variability. Lang, Sroufe, and Hastings found no evidence that the control of heart rate variability transferred to conditions of non-feedback. In the successful Ss no differences were observed between display and non-display periods in respiration rate, variability, or the interaction of these two. The few Ss who clearly seemed to be manipulating their respiration showed no greater improvement in the task than did Ss showing very stable respiration during the display periods.

Brener and Hothersall (1966) gave Ss auditory feedback of their heart rates - a low frequency tone being associated with low heart rates and a high frequency tone with high heart rates. When one signal lamp was on S was instructed to attempt to produce only the high tones and to inhibit the low ones; when the second signal lamp was on S was instructed to attempt to produce only the low tones and to inhibit the high ones. No indication was given to the Ss that the tones were associated with their heart rates. The signal lamps alternated. The results showed that the Ss displayed high heart rates during one period, and low heart rates during the other, with the difference between the mean heart rates in the final two periods being statistically significant. The results of this experiment provided clear evidence that under conditions of augmented sensory feedback Ss can learn to control their heart rates. Changes in respiration were also observed during the two

signal periods, but although the changes in heart rate were very similar in different Ss, the changes in respiration were very dissimilar. The verbal reports of the Ss as to how they had achieved control over the tones were also very variable.

In a second experiment Brener and Hothersall (1967) used two sessions. In the first session a procedure identical to that of the first experiment was used. The second session was divided into four phases, the first of which was the same as that of the first session. In the second phase a technique of respiration control similar to that of Wood and Obrist (1964) was used: S was instructed to pace his respiration by a signal lamp which flashed at a rate which he found comfortable. Ss were given training in pacing their respiration and after a little practice they were able to breathe very regularly under these conditions. In the third phase of the experiment the conditions of controlled respiration were maintained but S was instructed to attempt to control the tones as he had done before, but without changing his breathing. In the final phase of the experiment respiration control was removed. Once again good heart rate control was demonstrated both with and without respiration control. The results of these two experiments offer firm evidence of heart rate control under conditions of augmented feedback. The results of the second experiment strongly suggest that respiratory changes are not a prerequisite for such control, i.e., that heart rate can be controlled independently of changes in respiration.

The results of the independent and contemporaneous experiments of Lang *et al.* (1965, 1967) and Brener and Hothersall (1966, 1967) show that under conditions of augmented feedback human *Ss* can learn to exercise control over both heart rate variability and heart rate itself. These results also suggest that this control is independent of changes in respiration.

CHAPTER II

OPERANT CONDITIONING OF AUTONOMIC RESPONSES OTHER THAN HEART RATE

In the late 1950s Kimmel and his co-workers began a series of experiments on operant conditioning of the galvanic skin response (GSR). The results of these experiments have been reviewed in some detail by Kimmel (1967) and so they will only be briefly presented in this chapter. Kimmel and Hill (1960) made either pleasant or unpleasant odors contingent on a criterion reduction in skin resistance in two experimental groups. Two groups of yoked control Ss were also used. No differences in rate of emission of the GSR were found between the groups reinforced with the different odors, or between the experimental and control groups. In extinction the experimental Ss showed an increase in response frequency and the yoked control Ss a decrease. Kimmel and Hill were unable to account for this result. This experiment had a number of unsatisfactory features. Due to difficulties in presenting and expelling the different odors there was a variable delay between the emission of the response and the presentation of the reinforcement. Secondly there was no independent demonstration of the reinforcing properties of the stimuli used as 'reinforcers' in this experiment. Without a demonstration that the pleasant and unpleasant odors could in fact function as positive and negative reinforcers, it could be claimed that the results reported by Kimmel and Hill (1960) demonstrate that the odors they used do not function as reinforcers, rather than the more obvious claim that the GSR can not be conditioned as an operant.

Fowler and Kimmel (1962) used the presentation of a dim light as a reinforcer - S being seated in a dark sound-proof room. It is important to note that the S was not instructed to attempt in any way to turn the light on as often as he could. Fowler and Kimmel simply assumed that for a S in a dark room the presentation of such a dim light would be reinforcing. In view of the difficulties which have been encountered in using light onset as a reinforcer in studies using animals (Hurwitz, 1956; Lockard, 1963; Kiernan, 1964), an independent demonstration of the reinforcing properties of the light would have been valuable. They found in this experiment that whereas the yoked control Ss showed a steady decrease in rate of emission of GSRs, the experimental Ss did not show a decrease. This resulted in a statistically significant difference between the two groups in the numbers of GSRs they emitted during the last two minutes of acquisition and the first two minutes of extinction. Kimmel and Kimmel (1963) replicated this study and according to Kimmel (1967) their results:

> . . . provided more convincing evidence of operant conditioning of an autonomically mediated response than had been obtained previously. Not only did the instrumental Ss make significantly more responses than their yoked controls during reinforcement and extinction, but the instrumental Ss' response frequency during acquisition and most of extinction was higher than it had been during the last 5 minutes of habituation prior to reinforcement (Kimmel, 1967, p. 338).

Johnson (1963) used verbal reinforcement (That's good! That's fine!) in an investigation of the effects of response contingent reinforcement upon the rate of emission of the GSR. Five experimental Ss read through a set of cards containing nonsense syllables and

whenever they emitted a GSR they received a verbal reinforcement. Three control Ss received the same number of reinforcements but they were randomly presented. The experimental group of Ss showed a significantly greater rate of emission of GSRs than did the control Ss on three of the four conditioning days. Johnson concluded that verbal reinforcement can modify the rate of emission of the GSR.

Greene (1966) used a light as the reinforcer for GSRs. During 'reinforcement' the yoked control Ss showed a decline in the rate of emission of GSRs, whereas in general the experimental Ss did not. This differential effect resulted in a statistically significant difference in the number of GSRs emitted by the two groups in acquisition. Rice (1966) presented visual reinforcement to his Ss only when a GSR occurred in the absence of observed EMG responses. Significant differences between experimental and control Ss in rate of emission of the GSR were found under these conditions.

Shapiro, Crider and Tursky (1964) presented experimental Ss with a tone whenever they emitted a criterion electrodermal response (EDR)[1]. Control Ss received the same number of tones as their experimental partners but at times of no skin potential changes. The experimental Ss maintained a mean rate of responding at or above their resting level,

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[1]The distinction between the galvanic skin response (GSR) and the electrodermal response (EDR) is based upon the technique of measurement which is used. A GSR is recorded by passing a small current between two electrodes on the skin and measuring the resistance between them with a sensitive galvanometer. The electrodermal response (EDR) is measured by determining the voltage potential across two electrodes on the skin. Electrical activity of the sweat glands underlies both the GSR and the EDR (Sidowski, 1966, p. 173).

while the rate of responding in the control Ss declined over time. This resulted in a statistically significant difference between the groups in numbers of EDRs. Crider, Shapiro and Tursky (1964) reported the results of a second experiment in which an intra-S control procedure was used. Each S received a period of contingent reinforcement of EDRs and a period of non-contingent reinforcement, within a single session. During the conditioning periods Ss emitted more EDRs than they did during the periods of non-contingent reinforcement. Shapiro and Crider (1967) used monetary reinforcement of EDRs. Under different stimulus conditions they obtained differences in response rate consonant with changes in the scheduling of reinforcement. Birk, Crider, Shapiro and Tursky (1966) administered an immobilizing dose of d-tubocurarine to a volunteer prior to a reinforcement session in which EDRs were reinforced. Although S was unable to lift his arms or move his body he was able to breathe without difficulty throughout the session. Under these conditions an increased rate of emission of the EDR was found when each response produced a tone. They concluded that curarization had no effect on the conditioning phenomenon which their previous experiments had demonstrated.

Gavalas (1967) investigated the possibility that operant reinforcement could increase the rate of emission of the GSR. Eight human Ss were used and each S was run for ten consecutive days - each daily session being ten minutes in length. The first two days were used to determine the S's operant level for emission of the GSR; on the third day

the reinforcer was introduced and was presented randomly for ten minutes in order to measure the S's reaction to it; reinforcement of GSRs began during the second half of the third day's session and continued for the next four days; the last three days were extinction sessions. Control Ss followed the same procedure except that they received random, non-contingent reinforcement during the four and a half days of conditioning. Criterion GSRs were reinforced during conditioning by a flash of light and the comment 'That's good' or 'That's fine'. During the experiment all Ss performed a distracting task - pronouncing nonsense syllables. Experimental and control Ss differed significantly on three of the four days of conditioning and on the last day of extinction in rate of emission of GSRs. No significant differences were found during adaptation or during the period when the reinforcer was first introduced during the third day.

In a second experiment Gavalas matched Ss on the basis of their rate of emission of GSRs during adaptation. One member of each pair was assigned to the experimental group, and one member to the yoked control group. Six of the nine experimental Ss showed a clear increase in rate of emission of the GSR as compared with their yoked controls, while three of the experimental Ss did not show any such increase. However the group differences between the experimental and yoked control Ss in rate of emission of the GSR were significant on the last day of conditioning and the second day of extinction.

Higgins(1967) made either a positive reinforcer, a visual stimulus, or a negative reinforcer, an electric shock, contingent on GSRs in two groups of rats. Statistically significant differences were found in the

number of GSRs given by these two groups and Higgins concluded that the GSR can be modified using instrumental conditioning procedures.

There have been two recent reports of attempts to suppress GSRs using operant techniques. Senter and Hummel (1965) gave electric shocks to human Ss whenever they emitted a GSR. A decreased rate of GSR emission was observed in these Ss. Johnson and Schwartz (1967) confirmed this finding in an experiment in which a loud, and presumably aversive, tone was presented following spontaneous GSRs. A significant suppression effect was found and this effect persisted in extinction.

The results which have been presented thus far give the impression that a clear and unequivocal affirmative answer can be given to the question 'Can the GSR be conditioned as an operant?' In order to correct this impression some negative results will now be presented.

Mandler, Preven and Kuhlman (1962) made a visual stimulus contingent upon GSRs emitted during reinforcement periods. Ss subsequently received a sum of money proportional to the number of times the light had been switched on. They used an intra-S control procedure in which evidence of conditioning was sought in comparisons of response rates during stimulus periods when the contingencies were and were not in effect. Although more GSRs were emitted during the S^D periods, evidence for operant conditioning of the GSR, there was a general decrease in the rate of emission of this response throughout the session. Mandler et al. concluded that their results showed no evidence of operant conditioning of the GSR. This conclusion is directly opposed to the

conclusion of Kimmel et al., and is somewhat disturbing since Mandler et al. used what would appear to be a stronger reinforcer than the one used by Kimmel and Kimmel (1963). A light signalling money would be expected on a priori grounds to be a stronger reinforcer than a light alone. In support of Mandler et al. Mednick (1964) reported negative results from an attempt to condition the GSR as an operant.

Stern, Boles and Dionis (1966) gave Ss nickels whenever they emitted a spontaneous GSR, and failed to obtain operant conditioning of this response. Stern (1967) reported negative results in a study in which an attempt was made to condition spontaneous GSRs as operants. Four groups of sixteen human Ss were used. For the Ss in Group 1 a light was switched on as soon as they emitted a GSR; the Ss in Group 2 were yoked directly to those in Group 1 and received identical and simultaneous reinforcements. The Ss in Group 3 were indirectly yoked to Group 1 receiving the same number of reinforcements per minute, but only when S was not giving a GSR. Group 4 received no reinforcement. The GSR data showed no separation of these four groups in terms of number of GSRs during either conditioning or extinction, and there was no systematic change in rate of responding over time.

Kimmel and Kimmel (1968) reported the failure of an attempt to condition the GSR as an operant. They used their standard procedure for reinforcing spontaneous GSRs, except that a bright, red light rather than the previously used dim, white one was used as the reinforcer. The experimental Ss reinforced for spontaneous GSRs showed no tendency

towards increased responding during conditioning. In attempting to account for these unexpected results they could only suggest that possibly the red light was an aversive stimulus for the S. Support for this suggestion came from results reported by Kimmel and Kimmel (1968) for a group of Ss for whom the reinforcement for spontaneous GSRs was switching off the red light. In conditioning this group showed a systematic tendency toward increased responding, and persistence of the responding in extinction. Although these results do support the suggestion that the red light was aversive to the S it must be pointed out that initially they felt that changing from the dim white light used in their earlier experiments to the bright red light used in this one would be insignificant, and so the red light was not obviously aversive. It will be most interesting to see whether Kimmel is able to replicate his earlier findings of operant conditioning of the GSR, using the dim light as the reinforcer, in later studies. This latest finding of Kimmel and Kimmel is regarded as strongly supporting the suggestion, which was made earlier in this chapter, that an independent demonstration of the reinforcing properties of the stimuli used as 'reinforcers' in a number of Kimmel et al.'s experiments would have been very valuable. Post hoc designation of the stimuli as either positive or aversive, this designation being made on the basis of the results of attempts to use the stimuli to reinforce GSRs, seems completely unsatisfactory.

Why certain investigators have consistently reported negative results from attempts to operantly condition the GSR, while certain other investigators have reported positive results from such attempts,

is not clear at the present time. However it can safely be said that the question of whether or not the GSR can be conditioned as an operant is far from being completely answered.

Avoidance conditioning procedures have also been used in a number of attempts to operantly condition the GSR. The first of these was by Mowrer (1938) who reported his failure to condition the GSR in an experiment in which shock avoidance was contingent on a change in skin resistance. Kimmel and Baxter (1964) used a CS - UCS interval of five seconds, and if S made a criterion GSR during this period he avoided the electric shock. Yoked control Ss were used. The experimental Ss were found to emit significantly larger GSRs than did the controls, but the actual number of criterion GSRs did not increase, nor did the number of successful avoidances. Two replications of this experiment were reported by Kimmel, Sternthal and Strub (1966) in which a more objective criterion for the GSR was used. The differences between the experimental and yoked control groups were not significant in either of these replications, although the direction of these differences was the same as that of the earlier study, i.e., the experimental Ss made more and larger, but not significantly so, GSRs than did the control Ss. Grings (1965) found that avoidance Ss made slightly, but non-significantly, more GSRs than did control Ss. Grings and Carlin (1966) used both a punishment and an avoidance paradigm in an attempt to condition the GSR. The avoidance Ss were found to increase the number of GSRs they emitted, although the average magnitude of these responses was reduced. The punished Ss emitted fewer GSRs and the responses which they did emit were reduced in size.

Van Twywer and Kimmel (1966) attempted to condition the GSR as an avoidance response. An important feature of this experiment was that only GSRs which did not occur within five seconds of an EMG response, or a respiration irregularity, were reinforced. Although there may be difficulties in formulating a universally acceptable definition of exactly what constitutes an 'EMG response' or a 'respiration irregularity' the use of these measures does constitute an improvement of their experimental procedures. Significant differences in the numbers of GSRs emitted by the experimental and yoked control Ss were found. Similar results have been obtained by Kimmel and Sternthal (1967) who were able to successfully condition the GSR as an avoidance response. They also reported that the GSRs were not accompanied by any systematic changes in EMG frequency, respiration rate or respiration irregularities.

Since in these experiments both positive and negative results have been obtained it is difficult to formulate a conclusion as to whether or not the GSR can be conditioned as an operant. In addition it is difficult to generalize from the GSR to autonomic responses in general since the GSR can not be regarded as a representative autonomic response. The post-ganglionic fibers which innervate the sweat glands are cholinergic, unlike most post-ganglionic fibers of the sympathetic division of the autonomic nervous system (A.N.S.) which are adrenergic (Thompson, 1967, p. 198). Keele and Neil (1961) have pointed out that the GSR, unlike other autonomic responses, does not have any clear biological utility. In addition the GSR seems to function independently of the rest of the A.N.S. Shapiro, Crider and Tursky (1964), for example,

report operant conditioning of the GSR without any correlated changes in other autonomic activities - heart rate and skin potentials. So there is a need both for further experiments on the GSR as well as experiments on operant conditioning of other autonomic responses.

Very few experiments have been performed on operant conditioning of autonomic responses other than heart rate and the GSR. The first attempt to condition vasoconstriction as an operant was made by Skinner and Delabarre. In describing this experiment Skinner (1938) writes:

> In collaboration with Dr. E. B. Delabarre I have attempted to condition vasoconstriction of the arm in human subjects by making a positive reinforcer dependent upon constriction. The experiments have so far yielded no conclusive results, but there are many clinical observations that seem to indicate conditioning of this sort[2] (Skinner, 1938, p. 112).

An experiment by the Russian worker Lisina, as quoted by Razran (1961) demonstrates operant conditioning of a vasomotor response. Lisina observed that while the unconditioned vasomotor reaction to electric shock was constriction, occasionally Ss showed a vasodilatory response prior to the onset of the shock. An experimental

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[2]The results of this experiment, together with those on operant conditioning of the GSR reported by Mowrer (1938), are the 'evidence' to which Kimble (1961) refers in his statement:

> Thus, for autonomically mediated behavior, the evidence points unequivocally to the conclusion that such responses can be modified by classical, but not instrumental, training methods (Kimble, 1961, p. 100).

The evidence available to Kimble hardly seems to have justified such an unequivocal conclusion. Subsequent evidence has of course cast considerable doubt upon the validity of Kimble's conclusion.

procedure was used in which if a vasodilation occurred prior to shock onset, the shock was not presented, i.e., an avoidance conditioning paradigm was used with vasodilation as the effective response. Lisina ran this procedure for some time and found no increase in the frequency of dilatory responses. Visual feedback of vasomotor activity was then introduced by allowing the Ss to watch their plethysmographic records. Under the feedback conditions it was observed that vasodilation increased rapidly in frequency with a concomitant decrease in the number of shocks which the Ss received. The results of this experiment are of importance in indicating that vasodilation can be conditioned as an operant, and secondly in indicating the crucial role played by feedback in operant conditioning of an autonomic response.

Fromer (1963) conducted a study of instrumental conditioning of vasomotor responses in the rabbit. The results of this experiment failed to provide strong evidence of conditioning of the response. Snyder and Noble (1968) presented a light to human Ss on the peak amplitude of each vasoconstriction in the right index finger which was not preceded by bodily movement. Ss were told that the light would indicate correct responses. Two experimental groups were run as were two control groups, which received an equal number of presentations of the lights, but these presentations were contingent on vasomotor stability. Experimental Ss showed a significant increase in the number of vasoconstrictions during conditioning, while the control Ss did not show any such increase. In the experimental Ss conditioned vaso-

constrictions were independent of gross bodily movement, muscle tension in the finger and forearm, heart rate and respiratory irregularities.

DiCara and Miller (1968) reported a dramatic demonstration of operant conditioning of a vasomotor response. Rats were curarized and given a series of training trials signaled by the onset of a tone. During the tone Ss could obtain positively reinforcing brain stimulation by making specified vasomotor responses. Six rats were reinforced for relatively greater vasodilation in the right ear, and six for relatively greater dilation in the left ear. Ss reinforced for right ear vasodilation showed significant increases in dilation of the right ear, and significant decreases in vasodilation of the left ear, with the difference between the two ears in number of dilations being highly significant at the end of training. Ss reinforced for left ear dilation showed a significant increase in number of dilations of the left ear, but did not show a significant change in vasomotor activity in the right ear. Analyses of heart rate, vasomotor activity of the tail, and temperature did not show any differences between the two groups, or within the groups during training. So the conditioned response was highly specific.[3]

Plumlee (1968) implanted rhesus monkeys with chronic intra-arterial catheters. For four monkeys a discriminated avoidance procedure was used with a ten second tone terminating with a ten ma. shock, unless

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[3]DiCara and Miller have also been able to modify vasomotor responses in the tail of the rat using an operant conditioning procedure. A report of this experiment will appear in Commun. Behav. Biol. later this year.

the monkey's diastolic blood pressure rose to high levels and remained high for one second, in which case the tone terminated and the shock was not presented. All four monkeys acquired the discriminated avoidance response, showing diastolic elevations in blood pressure of up to 60 mm. of mercury. No changes in blood pressure were observed in response to a neutral tone which had never been paired with shock. For four other monkeys a similar procedure was used except that the avoidance response was a decrease in systolic blood pressure. All four monkeys showed drops in blood pressure, but the stimulus control over this response was not as good as was the case with the blood pressure increases.

Results such as these are extremely dramatic and if replicated they must surely have important implications for the medical treatment of a number of disorders of the cardiovascular system, for psychology in general, and particularly for the treatment of psychosomatic disorders.

In his 1961 address to the New York Academy of Sciences Neal Miller stated:

> I believe it is important to direct research towards this neglected problem in order to find out whether the two branches of the nervous system obey different laws. If they obey the same laws a dog rewarded whenever he salivates strongly should secrete more saliva than one given the same average number of rewards at times when he is salivating least (Miller, 1961, p. 834-835).

Miller and Carmona (1967) reported the results of an experiment in which the procedure suggested in this quotation was used. They attempted to change the rate of spontaneous salivation of mildly thirsty dogs by

using water to reinforce some of them when they increased their rate of salivation and others when they decreased their rate. The dogs increased their rate of salivation when reinforced for doing so, and the Ss reinforced for decreases in rate of salivation actually decreased their rates. The differences between the two groups were statistically significant. Miller and Carmona concluded that:

> The results clearly show that the autonomically innervated visceral response of salivation can be modified by an instrumental training procedure (Miller and Carmona, 1967, p. 4).

Brown and Katz (1967) reported a similar set of results from an experiment on operant conditioning of the human parotid salivary response. One group of Ss received ten cents each time a spontaneous increase in salivary rate occurred. A second group received ten cents in the absence of salivation or for minimal salivation. The number of salivations increased in the group reinforced for salivating but the increase did not attain statistical significance due to the marked inter-S variability. No significant changes in salivation were observed in the second group. They concluded that:

> This study indicates that salivary secretion in the human subject may be brought under stimulus control by means of operant reward techniques without evidence of mediation by swallowing (Brown and Katz, 1967, p. 160).

The results of these two experiments indicate that another autonomic response - salivation - is subject to modification and control through the use of operant conditioning techniques.

The final experiment which will be described in this chapter is that of Miller and Banuazizi (1968) who conducted a study to

determine whether curarized rats could learn yet another type of autonomically mediated response - the contraction or relaxation of the large intestine. While deeply curarized three Ss were reinforced with brain stimulation for intestinal relaxation, and four Ss were reinforced for contraction. Both groups of rats showed progressive changes in intestinal motility in the appropriate direction. Although the brain stimulation initially had an effect on intestinal motility this effect soon disappeared. In addition since the brain stimulation was used to reinforce different intestinal responses the results of this experiment can not be explained as solely due to the unconditioned effects of the brain stimulation.

CHAPTER III

METHOD

Subjects

The Ss used in these experiments were male, albino rats of the Sprague-Dawley strain purchased from Sprague-Dawley, Inc., of Madison, Wisconsin. The rats weighed from 200 to 250 grams at the time of delivery and were housed, six per cage, in large wire mesh cages in the colony room of the Department of Psychology of the University of Tennessee. Purina chow pellets and tap water were available in the home cages throughout the experiment. Supplementary cabbage and meat were given once a week. The colony room was maintained at a temperature of between 70° and 76° F., and was kept on a twelve hour light dark cycle changing at 6:30 A.M. and 6:30 P.M.

Implantation Procedure

For seven days before the operation the rats were housed in individual cages. Although the rats were not found to be particularly wild in order to further tame them and to accustom them to being handled by E each rat was handled and petted briefly on each of the seven days prior to the operation. In general the handling technique developed by Weininger, McClelland and Arima (1954) was used.

Twenty four hours before the operation all food was removed from the rat's living cage, although water continued to be available on an ad lib. basis. This deprivation procedure was adopted in

accordance with the suggestion of Routtenberg (1968) that food deprivation for a period of 24 hours before the administration of pento-barbital anesthesia increases the number of rats reaching surgical levels of anesthetisation, decreases the latency of induction of anesthesia, and lengthens the duration of the anesthesia. My own observations of the reactions of rats to pentobarbital anesthesia with and without food deprivation have confirmed Routtenberg's observations.

On the day of the implantation operation the rat was given an intraperitoneal (IP) injection of sodium pentobarbital (Nembutal)[1] at a dosage of 50/mg/kg. Once a surgical level of anesthetisation had been attained, the rat was mounted in a David Kopff stereotaxic instrument, and the implantation operation was begun. In general the procedure described by Miller, Coons, Lewis and Jensen (1961) was employed except that a commercially manufactured bipolar electrode[2] was used, and, following a suggestion by Goldstein (1967), the screws used to anchor the electrode to the skull were located on the lateral surface of the skull. The bipolar electrode was aimed at the medial forebrain bundle in the posterior portion of the lateral hypothalamus. (Krieg stereotaxic coordinates 1.5 mm. posterior to Bregma, 8.5 mm.

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[1]The sodium pentobarbital used in these operations was manufactured and supplied by Abbott Laboratories, North Chicago, Illinois.

[2]The bipolar electrodes were manufactured by Plastic Products Company, Roanoke, Virginia.

ventral to the point of entry, and 1.7 mm. left of the midline. After the operation the rat was returned to his individual cage, and for the next ten days a careful record was kept of his weight. Most rats were found to lose weight following the operation, usually from ten to twenty grams, and then to slowly return to their pre-operative weights. If a rat continued to lose weight he was given supplementary meat.

Testing the Effect of the Brain Stimulation

Ten to fourteen days after the implantation operation each rat was tested in a modified Skinner box in order to assess the effects of the brain stimulation. The dimensions of this box were six by eight by twelve inches, and it was constructed of Plexiglass. A lever was mounted on one wall of this box three inches above the level of the floor. Each lever pressing response made by the rat produced half a second of 60 cycles per second, ac stimulation, which was delivered from a Grass SD5 Stimulator. Fluctuations in brain resistance were minimized by a 100K potentiometer in series with the rat, and the current intensity was measured with an ac microammeter. Initial shaping was used and once the rat was pressing the lever consistently the intensity of the current was adjusted, between the limits of 20 to 90 microamps, so as to find the current intensity which yielded the fastest rate of responding without any gross motor side effects. Once this current intensity had been found it was used on all subsequent Skinner box sessions and was also the intensity of brain stimulation used to reinforce changes in heart rate

during the curare conditioning sessions.[3] Prior to the first curare session each rat was given two self-stimulation sessions in the Skinner box, with 500 responses being followed, on a continuous schedule of reinforcement (CRF), by the brain stimulation during each session. Only those rats which responded consistently during both these sessions were given curare conditioning sessions. Between each curare session each rat was tested in the Skinner box with at least 200 responses being reinforced with CRF brain stimulation. This procedure was adopted in order to ensure that curarization had not had any effect on the positively reinforcing properties of the brain stimulation. All of the rats which self-stimulated on the initial Skinner box sessions also self-stimulated on all of the later Skinner box sessions and so it seems safe to conclude that the brain stimulation continued to act as a positive reinforcer throughout the experiment. In two of the rats a small decrease of some ten microamps in the intensity of the brain stimulation which yielded the best self-stimulation rates was found between the first and last Skinner box sessions. In these rats the intensity of the brain stimulation used to reinforce heart rate changes during the curare conditioning sessions was adjusted to match the intensity used during the last Skinner box session. It is interesting that Olds (1958) has also reported that the intensity threshold for rewarding brain stimulation may decline during the course of an experiment.

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[3]The two exceptions to this general rule are discussed later on this page.

Curare Conditioning Sessions

At the beginning of each curare conditioning session the rat was given an IP injection of 12 units/kg., of d-tubocurarine chloride.[4] This dosage was found to be sufficient to maintain the rats used in this experiment in a completely immobile state for over two hours. During the curare conditioning session the rat lay on a small hammock in a sound shielded box which in turn was inside an electrically shielded enclosure. The hammock was tilted so that the rat's head was slightly lower than the rest of his body. This was done to allow saliva to run out of the rat's mouth. Once paralysis began, usually within two minutes of the injection, the rat was fitted with a mask made of moulded nylon which fitted over the animal's nose and respiration was begun. The mask was connected via plastic tubing to a small animal respirator (E and M Instrument Co., Model V5KG). Respirator settings of a 1:1 Inspiration/Expiration ratio, and a respiration rate of 70 breaths per minute were used. During the first five minutes following curarization the rat's heart rate was monitored and adjustments made in respiration rate and volume so as to ensure a stable and regular heart rate. Once such a heart rate had been found respiration rate and volume were fixed and no further adjustments were made during the remainder of the experimental session.

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[4]The d-tubocurarine chloride used in this experiment was manufactured and supplied by Eli Lilly and Company, Indianapolis.

The electrocardiogram (EKG) electrodes were Grass EKG silver disc electrodes. They were clipped to shaved areas of the skin lateral to the chest, and to the rear of the stomach. The shaved area of the skin was liberally coated with EKG Sol electrode paste. The EKG was recorded by means of a Grass Model 7 Polygraph. The amplified EKG signal was taken from one of the polygraph's J6 output jacks and fed through a pulse shaping circuit which converted the EKG wave form to a square wave pulse of fixed duration. This pulse provided the input to the BRS solid state control and analysis circuits. A second output from the polygraph was used to drive a cardiotachometer. A third channel on the polygraph was used for a marker pen which recorded the onset and offset of the feedback stimulus, of the brain stimulation, and of the Time Out.

An electrode lead was connected to the bipolar electrode to allow delivery of the brain stimulation. This lead was connected through a relay to a SD5 Stimulator. Signal lamps for the delivery of visual stimuli, and a speaker for auditory stimuli were located respectively in the roof and the door of the sound-shielded box.

Pairs of rats were operated upon and tested at one time. This was done since only two rats could be given their series of curare conditioning sessions at one time. One rat in each pair was assigned on the basis of a coin toss to one of the experimental groups, and the second rat was assigned to the second group. This assignment of the rats to the different groups was done before the first curare conditioning session in order to avoid the possibility of experimenter bias.

The curare conditioning session began with an adaptation phase which lasted for approximately thirty minutes. During this time the rat's heart rate was monitored and recorded and an inter heart-beat interval (IBI) selected so that approximately 50 percent of the IBIs emitted by the rat would be longer than this criterion IBI, and approximately 50 percent would be shorter. Following this adaptation phase the conditioning phase of the session was begun. The conditioning also lasted for approximately thirty minutes. During conditioning the rats in Group 1 were reinforced for increases in heart rate, and the rats in Group 2 for decreases. The procedure in conditioning for a rat being reinforced for heart rate decreases has been notated in Figure 1.

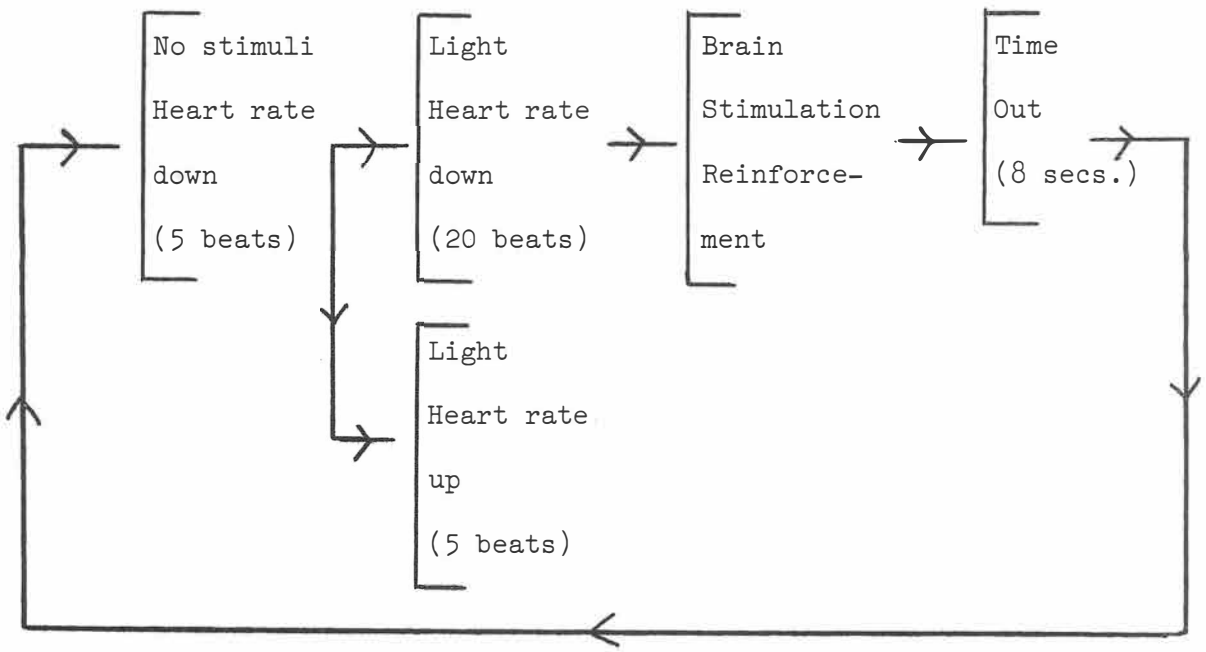

Figure 1. The procedure used in conditioning for a rat being reinforced for heart rate decreases.

At the beginning of the conditioning trial no stimuli were present but the rat's heart rate was monitored and recorded. If the first five IBIs emitted by the rat met the criterion which had been selected on the basis of the rat's adaptation heart rate, a light in the experimental chamber was switched on. If the rat's heart rate did not meet the criterion no stimuli were presented, and this condition continued until a sample of five IBIs was emitted which did meet the criterion. Once the light was switched on, provided that the rat's IBIs continued to meet the criterion, it remained on. When twenty, successive, additional, criterion IBIs occurred, the light terminated with the delivery to the rat of half a second of brain stimulation at the same current intensity as was used in the Skinner box. The brain stimulation was followed by an eight second Time Out period during which none of the stimuli were presented and none of the contingencies were in effect. This Time Out period was introduced to allow the rat's heart rate to recover from the effects of the brain stimulation, since this stimulation was found to elicit a clear decrease in heart rate. Malmo (1961) and Holdstock (1967) have reported a similar slowing of heart rate in rats following septal self-stimulation. At the end of the Time Out period the contingencies were reinstated and the procedure repeated until the end of the conditioning phase of the session. If, in the presence of the light, the rat's heart rate increased the light was switched off and the procedure recycled. If it was found that the rat was not meeting the criterion, and so was not receiving any brain stimulation reinforcements, the criterion was

made less stringent and was kept at this value until it was felt that a change to a more stringent criterion value could be made. If, on the other hand, it was found that the rat was easily meeting the criterion, the criterion was shifted to a more stringent value thus making it more difficult for the rat to obtain the brain stimulation reinforcements. Changes in the criterion were accomplished by shifting it in the appropriate direction by .01 seconds. The procedure for a rat being reinforced for heart rate increases would, of course, be the opposite to the procedure which has been described, i.e., an increase in heart rate for five beats would initially switch the light on and if maintained for an additional twenty beats would lead to the delivery of the brain stimulation. An extinction phase, during which neither the feedback stimulus or the brain stimulation were presented, followed the conditioning phase of the session. This extinction phase lasted for approximately thirty minutes. During the adaptation, conditioning and extinction phases of the curare session the numbers of heart beats in successive one minute periods were recorded. During conditioning the numbers of brain stimulation reinforcements and the criterion changes were also recorded.

Each rat was given four curare conditioning sessions separated by at least three days.

CHAPTER IV

RESULTS AND DISCUSSION

The numbers of heart beats for each of the twelve rats used in the first experiment during successive one minute periods of the adaptation, conditioning, and extinction phases of the four curare conditioning sessions are shown in Tables I to VIII of Appendix A. In addition criterion changes and numbers of brain stimulation reinforcements during conditioning periods are shown in these tables.

In order to present the changes in heart rate which occurred in the two experimental groups within each of the four curare conditioning sessions mean heart rates were calculated for each of the twelve rats during four blocks of adaptation periods, four blocks of conditioning periods, and four blocks of extinction periods. Since there was some variation[1] in the lengths of the three phases of the different curare sessions these means were not based upon equal numbers of one minute periods. Consequently the following procedure was adopted in calculating these means. The number of one minute periods in any phase of the curare session (N) was divided by four, and means calculated for four blocks of N/4 periods. These means for the first and second curare sessions are shown in Tables 1 and 2 and are presented graphically in Figure 2.

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[1]The minimum and maximum lengths of the adaptation, conditioning and extinction phases of the curare conditioning sessions were 20 and 31 minutes for adaptation, 20 and 31 minutes for conditioning, and 20 and 33 minutes for extinction. The average lengths of the adaptation, conditioning and extinction phases were respectively 25, 27, and 26 minutes.

Table 1

Mean heart rates in beats per minute during four blocks of adaptation, conditioning and extinction periods

	Session 1											
Rat No.	Adap.	A	A	A	Cond.	C	C	C	Ext.	E	E	E
		Group 1:			Reinforced for heart rate increases							
2 C	487	488	487	487	486	481	474	472	463	453	458	458
6 B	441	425	428	412	428	431	437	439	420	412	404	414
7 C	503	492	482	472	481	483	487	488	491	496	494	489
10 C	524	479	474	486	471	493	497	487	482	490	495	493
4 D	486	465	471	467	464	453	455	448	462	442	442	453
5 D	392	388	379	370	350	347	348	357	361	353	346	342
Totals	2833	2737	2721	2694	2680	2688	2698	2691	2679	2646	2639	2649
Means	472	456	454	449	447	448	450	449	447	441	440	442
		Group 2:			Reinforced for heart rate decreases							
1 B	538	532	532	529	510	505	499	501	488	478	471	489
6 C	483	478	482	480	465	453	452	457	449	452	458	453
8 C	324	346	401	420	386	382	371	370	363	368	371	374
9 C	562	556	541	533	519	519	518	528	534	535	540	549
10 D	482	478	479	479	474	464	456	466	457	459	467	470
Totals	2839	2837	2876	2880	2795	2760	2734	2738	2717	2718	2735	2755
Means	473	473	479	480	466	460	456	456	453	453	456	459

Table 2

Mean heart rates in beats per minute during four blocks of adaptation, conditioning and extinction periods

	Session 2											
Rat No.	Adap.	A	A	A	Cond.	C	C	C	Ext.	E	E	E
		Group 1: Reinforced for heart rate increases										
2 C	494	498	497	498	488	485	487	486	479	478	475	471
6 B	457	476	465	464	479	497	490	488	482	478	479	470
7 C	414	415	405	409	413	420	427	437	438	442	443	445
10 C	512	483	475	471	474	481	480	486	464	441	431	432
4 D	474	455	454	453	464	481	490	485	478	479	478	475
5 D	383	377	375	364	369	378	396	408	398	385	380	381
Totals	2734	2704	2671	2659	2687	2742	2770	2790	2739	2703	2686	2674
Means	456	451	445	443	448	457	462	465	457	451	448	446
	Group 2: Reinforced for heart rate decreases											
1 B	473	459	442	456	449	432	430	433	444	453	459	462
6 C	411	414	413	410	405	401	395	397	404	408	409	416
8 C	391	384	382	379	363	354	351	337	340	354	359	372
9 C	534	528	523	518	516	510	496	481	485	490	492	491
9 D	455	439	411	366	356	342	335	331	329	330	331	331
10 D	426	407	414	410	408	402	398	395	398	405	408	415
Totals	2690	2631	2585	2539	2497	2441	2405	2374	2400	2440	2458	2487
Means	448	439	431	423	416	407	401	396	400	407	410	415

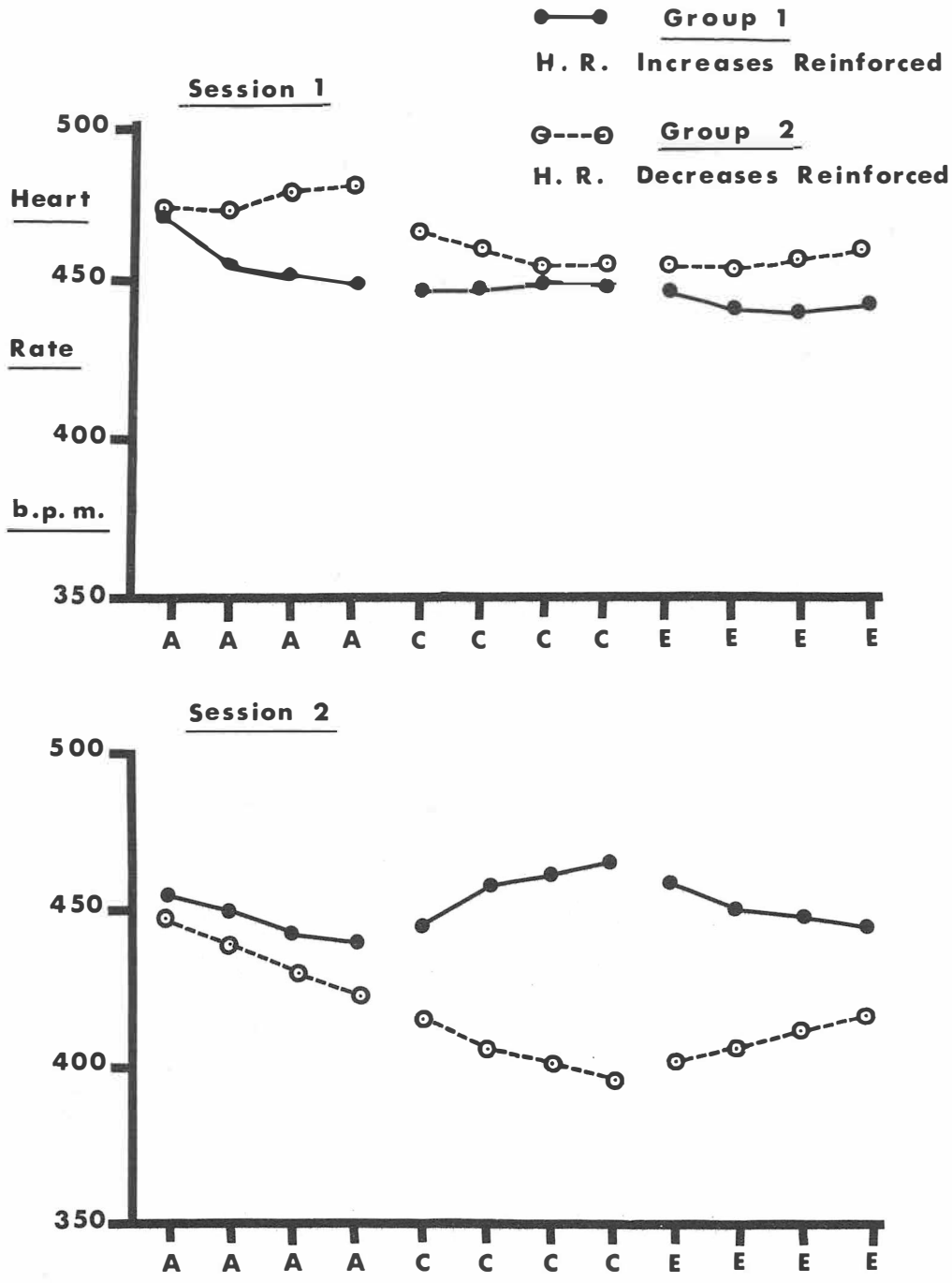

Figure 2. Group mean heart rates for four blocks of adaptation, conditioning and extinction periods during the first and second curare sessions.

During the conditioning phase of the first curare session the group reinforced for decreases in heart rate - Group 2 - showed a small decrease in heart rate resulting in a difference of 24 beats per minute (b.p.m.) between the means for the last block of adaptation periods and the mean for the last block of conditioning periods; and a difference of 10 beats per minute between the means for the first and last blocks of conditioning periods. The small decrease in heart rate shown by this group might be regarded as evidence of operant conditioning of a decrease in heart rate in curarized rats. However the group reinforced for heart rate increases - Group 1 - showed no difference in heart rate between the means for the last block of adaptation periods and the last block of conditioning periods, and a difference of only 2 b.p.m. between the means for the first and last blocks of conditioning periods. This result appears contrary to what might have been expected on the basis of the principles of operant conditioning. On the other hand in the present research project we have consistently found that within a curare session, in the absence of any experimental contingencies, there is an over-all decrease in heart rate, and so the fact that the heart rate of the group reinforced for heart rate increases did not in fact decrease might be regarded as evidence, although admittedly weak evidence, of an operant conditioning effect. However this argument could equally well be used to account for the small decrease in heart rate shown by Group 2 during the first curare conditioning session and so it seems most realistic to conclude that the results of this experiment show little if any evidence of operant conditioning of changes in heart rate during the first curare conditioning session.

Trowill (1967) reported a mean increase, during a curare conditioning session, of 18 b.p.m. in a group of 19 rats reinforced with brain stimulation for increases in heart rate, and a mean decrease of 19 b.p.m. in a group of 17 rats reinforced for decreases in heart rate. Trowill's results for the decrease group are very similar to those of the experiment reported in this dissertation - a decrease of 19 b.p.m. during conditioning as compared with the decrease of 24 b.p.m. in the present experiment. However he was much more successful in conditioning increases in heart rate. Trowill used a training period of ten minutes for nine of the rats, and a period of thirty minutes for the remaining Ss, whereas the average length of the conditioning phase of the first session in my experiment was 29 minutes. So at least for three quarters of Trowill's Ss the length of the training period was close to that of the conditioning phase of the first session used in the present experiment. The most important difference between the two experiments is that in my experiment the group to be reinforced for increases in heart rate had a much higher mean heart rate during adaptation (456 b.p.m.), than was the case in Trowill's experiment. His increase group had a pre-training mean heart rate of 401 b.p.m. Wilder's (1956) Law of Initial Values states that the magnitude of the response to an experimental stimulus or condition is related to the pre-stimulus level of the response system. Since there was a large difference in the pre-conditioning heart rates of the two increase groups used in these experiments this law provides an explanation of the difference in the effectiveness of the conditioning procedures in producing increases in heart rate.

The results of the present experiment also contrast with those of Miller and DiCara (1967). Miller and DiCara were able to condition both large increases and decreases in heart rate in two groups of curarized rats reinforced with brain stimulation for changes in heart rate. The result of this differential effect was a large difference of 194 b.p.m. in the mean heart rates of their two groups at the end of one curare conditioning session.

One possible explanation of the difference in the results of the two experiments is that Miller and DiCara used a ninety minute training period, whereas in the present experiment the conditioning phase of the first curare session only lasted for approximately thirty minutes. However even after thirty minutes Miller and DiCara were able to condition a difference of approximately 100 b.p.m.[2] in the mean heart rates of their two groups. Although it seems possible that if the conditioning phase of the first curare session had been increased in length in the present experiment a larger difference in the heart rates of the two groups would have been conditioned, since similar procedures were used in the two experiments, an explanation of the clear difference in their results must be sought.

At the beginning of conditioning in the Miller and DiCara experiment, there was a difference of 22 b.p.m. in the mean heart rates of their two groups, with the group which was to be reinforced for

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[2]This difference has been taken from Figure 1 on page 14 in Miller and DiCara (1967).

increases in heart rate showing the higher average heart rate; in the present experiment, on the other hand, there was a difference of 34 b.p.m. in the mean heart rates of the two groups at the beginning of conditioning, with the group which was to be reinforced for increases in heart rate showing the lower average heart rate. Since rats were randomly assigned to the experimental groups in both experiments this difference was due to chance. In view of this difference in the initial heart rates of the groups used in these experiments it seems that an assessment of the conditioning effects in terms of the changes from these initial levels which were conditioned, is more satisfactory than a comparison of the four groups in terms of heart rates at the end of conditioning. Table 3 shows the changes in heart rate which were actually conditioned in the two experiments. Table 3 clearly shows how much more successful Miller and DiCara were in conditioning changes in heart rate than was the present experimenter. The results reported by Miller and DiCara represent a dramatic demonstration of operant conditioning of both increases and decreases in heart rate in curarized rats with brain stimulation reinforcement. Although there was some evidence of operant conditioning of a decrease in heart rate during the first curare session in the present experiment there was no evidence of operant conditioning of an increase in heart rate. I have not been able to find a satisfactory explanation of the large difference in the results of these two experiments in which similar procedures were used.

A similar position obtains with respect to the results of experiments in our laboratory on avoidance conditioning of changes in

Table 3

A comparison of the conditioned changes in heart rate in two experiments using curarized rats and brain stimulation reinforcement

Experiment	Group	H.R. at the beginning of conditioning	H.R. after 30 mins. of conditioning	Change in Heart rate
Miller and DiCara (1967)	Increase	422 b.p.m.	460 b.p.m.	(460 - 422) = 38 b.p.m.
	Decrease	400 b.p.m.	360 b.p.m.	(400 - 360) = 40 b.p.m.
Present Experiment	Increase	449 b.p.m.	449 b.p.m.	(449 - 449) = 0 b.p.m.
	Decrease	480 b.p.m.	456 b.p.m.	(480 - 456) + 24 b.p.m.

heart rate.[3] In these experiments much smaller heart rate changes were conditioned than was the case in an experiment reported by DiCara and Miller (1968) in which changes in heart rate were conditioned using an avoidance conditioning procedure. Why Miller and his co-workers have been consistently more successful in operantly conditioning changes in heart rate than we have been is, at the present time, not clear.

Tables 2 (p. 51), 4 and 5, and Figures 2 (p. 52) and 3 show that on the second, third and fourth curare conditioning sessions of the present experiment, there were clear differences in the heart rates of the two groups, with the group reinforced for increases in heart rate showing a higher heart rate than did the group reinforced for heart rate decreases. Figures 2 and 3 clearly show the difference between the results for the three later sessions and the first session. Although there was at best little evidence of operant conditioning of changes in heart rate during the first curare session, there was clear evidence of such a result during the later sessions. Table 6 presents the changes in heart rate which were conditioned in the two experimental groups during the later curare conditioning sessions. Table 6 shows that during the later curare sessions the group reinforced for increases in heart rate showed a clear heart rate increase, and the group reinforced for decreases in heart rate showed a clear heart rate decrease. Figures 2 and 3 show that the difference in the heart rates of the two groups tended to increase during conditioning on the later curare sessions.

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[3]These unpublished experiments were performed during 1967 by Brener and Goesling.

Table 4

Mean heart rates in beats per minute during four blocks of adaptation, conditioning and extinction periods

Rat No.	Adap.	A	A	A	Cond.	C	C	C	Ext.	E	E	E
					Session 3							
		Group 1: Reinforced for heart rate increases										
2 C	491	491	488	487	482	490	495	497	500	501	500	495
6 B	501	465	464	463	484	499	513	537	519	486	473	469
7 C	506	504	480	470	480	481	496	506	507	506	498	488
10 C	479	450	435	428	411	428	464	473	477	467	456	452
4 D	472	443	453	455	471	481	488	480	474	454	467	456
5 D	374	371	377	376	377	386	401	413	408	402	393	384
Totals	2823	2724	2697	2679	2705	2765	2857	2906	2885	2816	2787	2744
Means	471	454	449	446	451	461	476	484	481	469	464	457
		Group 2: Reinforced for heart rate decreases										
1 B	492	478	472	464	418	423	418	404	385	379	375	383
6 C	448	459	454	458	452	438	443	434	440	449	455	468
8 C	481	451	420	412	408	409	407	390	413	415	431	432
9 C	529	528	529	524	508	503	499	508	504	500	508	521
9 D	432	429	427	426	415	403	398	394	389	392	392	389
10 D	456	447	458	449	449	435	426	416	414	420	435	456
Totals	2838	2792	2760	2733	2650	2611	2591	2546	2545	2555	2596	2649
Means	473	465	460	455	442	435	432	424	424	426	433	441

Table 5

Mean heart rates in beats per minute during four blocks of adaptation, conditioning and extinction periods

	Session 4											
Rat No.	Adap.	A	A	A	Cond.	C	C	C	Ext.	E	E	E
		Group 1: Reinforced for heart rate increases										
2 C	482	473	440	463	465	472	481	479	474	472	466	460
6 B	477	473	466	466	477	487	476	485	458	439	443	437
7 C	415	437	462	469	463	472	476	484	473	472	468	462
10 C	470	463	469	468	465	469	473	482	485	489	477	469
4 D	456	449	433	429	410	417	428	434	426	417	417	423
5 D	411	397	403	408	413	416	416	415	402	390	393	393
Totals	2711	2692	2673	2703	2693	2733	2750	2779	2718	2679	2664	2644
Means	450	449	445	450	449	456	458	463	453	447	444	441
	Group 2: Reinforced for heart rate decreases											
1 B	518	515	509	493	479	451	419	420	466	472	476	474
6 C	469	481	493	493	467	486	487	484	506	510	501	501
8 C	420	415	410	414	395	387	379	374	381	388	386	386
9 C	519	515	515	511	503	494	483	481	485	490	491	497
9 D	369	358	344	331	323	323	318	313	316	312	308	307
10 D	474	476	475	476	464	458	458	450	452	450	456	461
Totals	2769	2760	2746	2718	2631	2599	2544	2522	2606	2622	2618	2626
Means	461	460	458	453	438	433	424	420	434	439	436	438

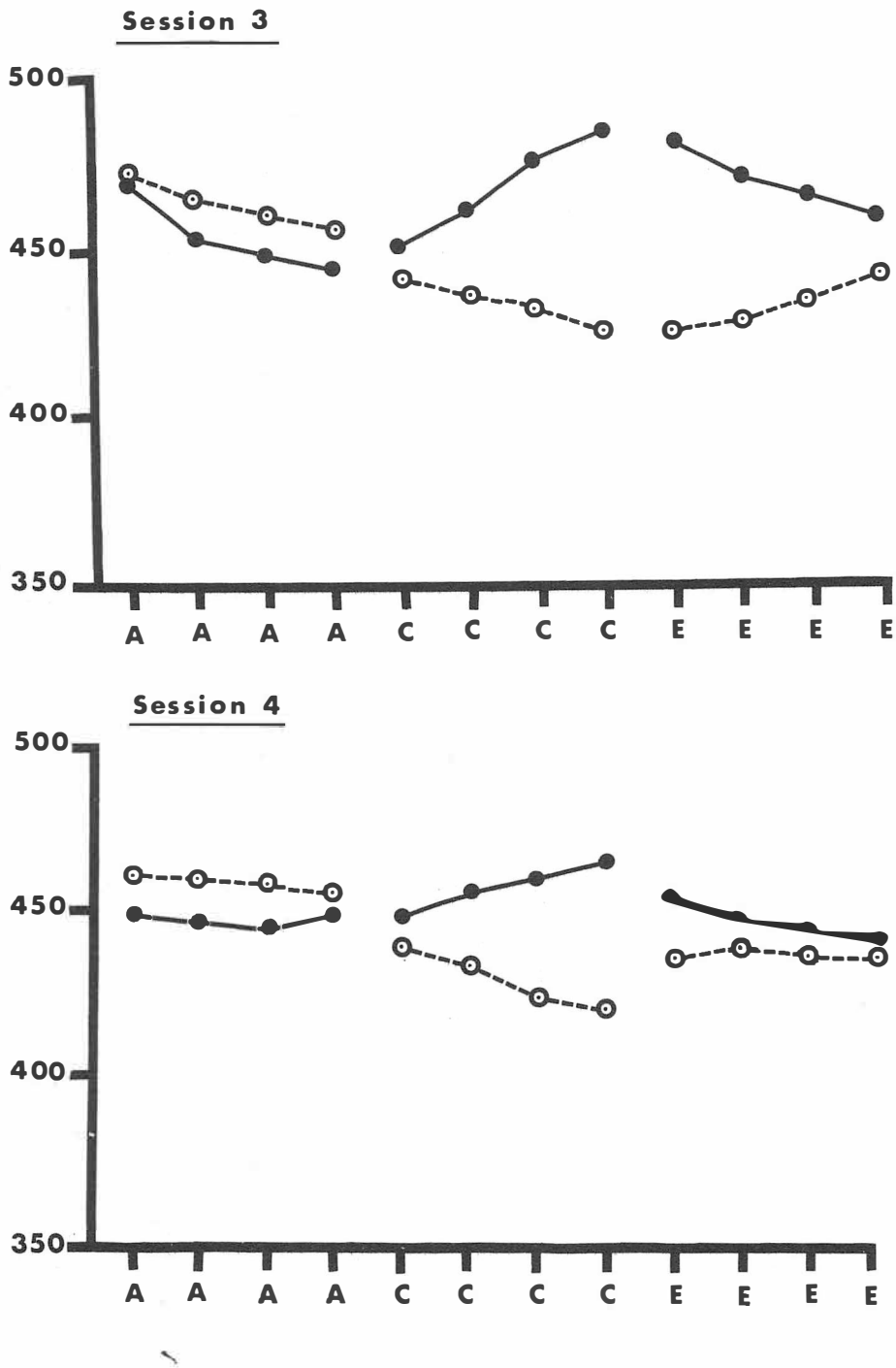

Figure 3. Group mean heart rates for blocks of adaptation, conditioning and extinction periods during the third and fourth curare sessions.

Table 6

Group mean changes in heart rate conditioned during the second, third, and fourth curare sessions

Session	Group	Mean heart rate for last block of adaptation periods	Mean heart rate for last block of conditioning periods	Change in heart rate (Cond. mean-Adap. mean)
2	1 H.R. Incr.	443 b.p.m.	465 b.p.m.	+ 22 b.p.m.
	2 H.R. Decr.	423 b.p.m.	396 b.p.m.	- 27 b.p.m.
3	1 H.R. Incr.	446 b.p.m.	484 b.p.m.	+ 38 b.p.m.
	2 H.R. Decr.	455 b.p.m.	424 b.p.m.	- 31 b.p.m.
4	1 H.R. Incr.	450 b.p.m.	463 b.p.m.	+ 13 b.p.m,
	2 H.R. Decr.	453 b.p.m.	420 b.p.m.	- 33 b.p.m.

In the statistical analysis of the results of this experiment differences were computed between each rat's heart rate during the last block of adaptation periods and the last block of conditioning periods for each session. The results of an analysis of variance performed on this data are shown in Table 7. The treatments effect was found to be highly significant, $F(1, 10) = 47.86$, $p < .001$, indicating that in terms of this measure the two experimental groups showed significantly different changes in heart rate. The sessions effect was found to be non-significant, $F(3, 30) = 2.89$, $p > .05$, but the interaction between treatments and sessions was found to be significant, $F(3, 30) = 5.14$, $p < .01$. This significant interaction appears to have been due to the fact that the group reinforced for heart rate increases only showed clear evidence of operant conditioning of an increase in heart rate during the later curare conditioning sessions.

Figures 2 (p. 52) and 3 (p. 61) show evidence of an extinction effect in the results of this experiment in that during the second, third and fourth curare sessions the curves of the two experimental groups which tended to diverge during conditioning converged during extinction. In order to illustrate this extinction effect differences were computed for each session between the mean heart rates of the two groups during the final block of conditioning periods, and the final block of extinction periods. Table 8 shows these differences. Table 8 shows that although there were differences on the second, third and fourth curare sessions between the mean heart rates of the two groups during the last block of conditioning

Table 7

Summary of the Analysis of Variance on the difference in heart rate between the last block of adaptation and the last block of conditioning periods for four curare sessions

Source	df	Sum of squares	Mean Square	F	Significance level (p)
Between Ss	11	31,645	-	-	-
Treatments (A)	1	26,180	26,180	47.86	$p < .001$
Ss within groups Error A	10	5,465	546.5	-	-
Within Ss	36	11,146	-	-	-
Sessions (B)	3	1,784	594.7	2.89	ns
A x B	3	3,180	1,060	5.14	$p < .01$
B x Ss within groups. Error B	30	6,182	206.1	-	-

Table 8

Differences between the group mean heart rates for the last block of conditioning periods and the last block of extinction periods

Difference between the group means for the last block of conditioning periods.	(449-456) = -7 b.p.m.	(465-396) = +69 b.p.m.	(484-424) =+60 b.p.m.	(463-420) = +43 b.p.m.
Difference between the group means for the last block of extinction periods.	(442-459) =-17 b.p.m.	(446-415) = +31 b.p.m.	(457-441) =+16 b.p.m.	(441-438) = + 3 b.p.m.

periods, these differences were not maintained in extinction. The difference in the mean heart rates of the two groups became progressively smaller over the last three curare sessions. So in this experiment a heart rate change in the direction being conditioned occurred while reinforcement was being delivered, but the changed heart rate was not sustained once the reinforcement was no longer contingent on the change in heart rate. This result is important since it shows that one other phenomenon of operant conditioning can be demonstrated when the operant response is a heart rate change.

The results of this experiment show that both increases and decreases in heart rate can be conditioned when brain stimulation is used as the reinforcement for the heart rate change. Since these heart rate changes were conditioned in completely paralyzed rats the possibility that the heart rate changes were mediated by some type of skeletal response has been at least markedly reduced.[4] The results of this experiment extend those reported by Trowill (1967) and Miller and DiCara (1967) in showing the importance of extent of conditioning in experiments on operant conditioning of changes in heart rate, and they also provide evidence of an extinction effect.

Since the results of this first experiment provided a clear demonstration that both increases and decreases in heart rate can be

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[4] The implications of these results will be discussed in detail in Chapter V of this dissertation.

conditioned when brain stimulation is used as the reinforcer with curarized rats, it was decided to perform a second experiment in which an attempt was made to condition both increases and decreases in heart rate in individual rats. Since this experiment was a pilot study its procedures and results will be presented and discussed in Appendix B of this dissertation.

CHAPTER V

SUMMARY AND CONCLUSIONS

The aim of the first experiment reported in this dissertation was to investigate the possibility that both increases and decreases in heart rate can be conditioned in curarized rats when brain stimulation is used as the reinforcement for the heart rate changes. Curarized rats were used in an attempt to reduce the possibility of skeletal mediation of the conditioned heart rate changes. Brain stimulation was used as the reinforcer because such stimulation has been found to yield powerful reinforcing effects in many experiments in which more typical operant responses have been used. This stimulation could also be made contingent on the heart rate changes in a reliable and controlled manner. Two groups of six rats each were used in this experiment one of which was reinforced during the four curare conditioning sessions for increases in heart rate, and one for heart rate decreases. Little if any evidence of operant conditioning of either an increase or a decrease in heart rate was found in these groups during the first curare conditioning session. This result contrasts with the reports of Trowill (1967) and Miller and DiCara (1967). A number of possible explanations of the difference in the results of these experiments were considered.

During the second, third and fourth curare conditioning sessions clear differences in heart rate were found between the two groups, with the group reinforced for heart rate increases showing a clear increase in heart rate during conditioning, and the group reinforced for heart

rate decreases showing a clear decrease in heart rate during conditioning. An analysis of variance showed a statistically significant difference between the two groups during the later curare conditioning sessions, and a statistically significant interaction between treatments and sessions. The results for the last three curare sessions confirm and extend the findings of Trowill (1967) and Miller and DiCara (1967) and clearly show that both increases and decreases in heart rate can be conditioned in curarized rats when brain stimulation is used as the reinforcement for the heart rate changes. In addition there was some evidence of an extinction effect during the later curare sessions in that the heart rates of the two groups which tended to diverge during the conditioning phase of the session tended to converge during the extinction phase.

Since the results of this first experiment provided a clear demonstration of operant conditioning of both heart rate increases and heart rate decreases in different groups an attempt was made to condition both increases and decreases in heart rate in the same rat. Two different procedures for this reversal heart rate conditioning were used with three rats. All three rats showed good acquisition of the initial heart rate change, thus replicating the results of the first experiment, but no evidence of acquisition of the heart rate reversal. Since Ss differ considerably in their cardiac behavior and in their reactions to stimuli a demonstration of operant conditioning of both increases and decreases in heart rate in the same S would represent a convincing

demonstration of the phenomenon of operant conditioning of changes in heart rate. However such a demonstration is not provided by the results for the three rats given a series of reversal conditioning trials. It must be remembered however that many investigators have reported failures to establish reversal conditioning with more conventional responses, and so difficulty in establishing heart rate reversals may be due to the nature of the reversal conditioning procedure, rather than due to the characteristics of heart rate as an operant response.

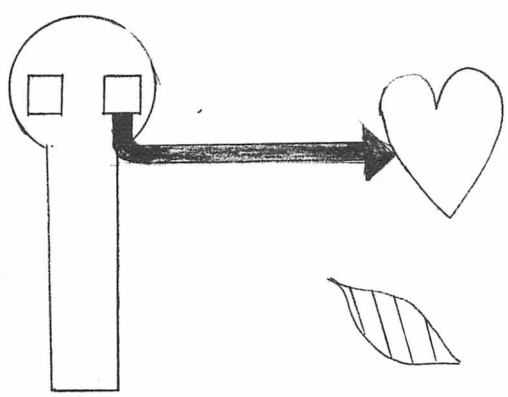
Since one of the most important features of this research was the use of curarized rats as experimental Ss, I would like to end this dissertation with some notes on the limitations inherent in the use of such Ss. It must be pointed out that when these experiments were planned I was not aware of a number of these limitations, and I only became aware of them as a result of my experience in conducting this research.

The most obvious starting point for research on conditioning of changes in heart rate is the question originally posed by Shearn (1961) 'Does the heart learn?' Since operant conditioning procedures have been found to lead to the modification and control of a variety of responses, one widely used procedure has been to make some type of reinforcement contingent on a change in heart rate, in a typical operant conditioning paradigm:

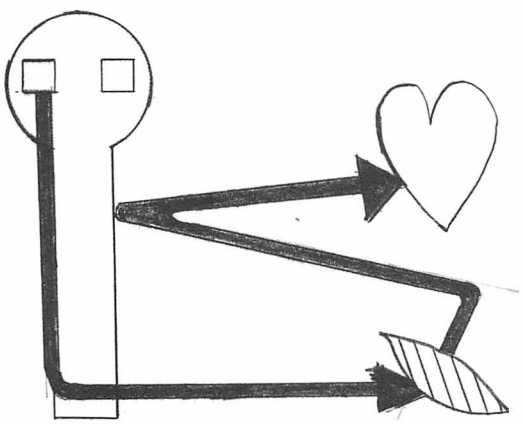
$$\text{Response}_{(\text{Heart rate change})} \longrightarrow \text{Reinforcement}$$

Under such conditions let us assume that a clear increase in the frequency of occurrence of the heart rate change is observed. It might be concluded

that such results demonstrate that changes in heart rate can be operantly conditioned, and that a clear and unequivocal affirmative answer can be given to the question 'Does the heart learn?' However such a conclusion would certainly be challenged by proponents of the mediation or artifact hypothesis who would argue that in such an experiment some type of skeletal response could actually have been conditioned, and that it was these skeletal changes which mediated the heart rate changes which were observed. It might be further argued, as Smith (1954) suggested, that only skeletal responses can be conditioned and that 'conditioned' heart rate responses are no more than artifacts elicited by feedback from the skeletal responses. These different positions can be made clearer with reference to a series of models which were independently proposed by Black (1967) and Brener (1967). The claim that heart rate changes can be conditioned using an operant conditioning procedure is presumably based on the assumption that activity in some neural center directly controls heart rate and produces the heart rate change which has been conditioned.



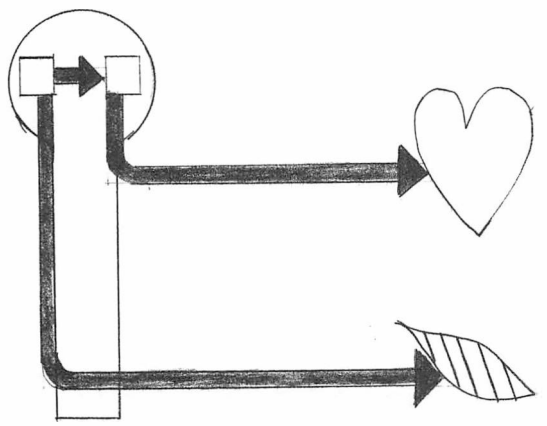
A mediation theorist however would challenge this type of explanation of the results and would propose that neural centers controlling some type of skeletal response had been activated, and that feedback from these skeletal responses produced the heart rate change. The following diagram illustrates this type of argument:



This model shows that if for example respiratory and heart rate changes were conditioned simultaneously, then the heart rate changes which were observed could have been the result of feedback from the respiratory changes.

Curarized rats were used in the experiments reported in this dissertation in an attempt to reduce, if not completely eliminate, the possibility of skeletal changes occurring during conditioning, and so to study operant conditioning of heart rate changes uncontaminated

by skeletal changes. With reference to the above model curarization might be regarded as a procedure which breaks the link between the neural control center and the skeletal response. If this model is accepted curarization is a useful and valuable procedure to adopt in experiments on operant conditioning of autonomic responses. However a third model might be proposed. This model is shown in the following diagram:



According to this model in order for a heart rate change to be mediated by a skeletal response it is not necessary for the skeletal response to actually occur, but only for what Black (1967) has termed the 'central movement process' underlying the skeletal response to occur. So even in a completely paralyzed S, where there is no possibility of skeletal movement, it is still possible for a heart rate response to be mediated by a 'central movement process'. Black (1967) writes:

> When an operantly conditioned heart rate response occurs the result of the conditioning process is to activate neural centers controlling movements, and to produce a change in heart rate (Black, 1967, p. 10).

Smith (1967) has proposed a similar position:

> Thus, it (Smith's 1954 paper 'Conditioning as an Artifact') insists that the somatic response and the autonomic response must be pictured, in the general sense, as arising coordinately from a common source (Smith, 1967, p. 101).

Obrist and Webb (1967) have adopted a similar position. They state:

> However, these studies (studies in which curare has been used) also appear to be inconclusive because the technique evaluates only the extent to which cardiac events are influenced by the actual occurrence of peripheral somatic-motor events. What is not evaluated is the possibility that both responses have a common origin and means of control with the central nervous system . . ..In this case, neither response 'causes' the other; rather they might be regarded as concomitants, or as simply different aspects of the same response process (Brackets mine. Obrist and Webb, 1967, p. 8).

Curare blocks only the peripheral manifestations of the 'central movement processes' or activity in some neural 'common source', but has no effect on the processes or neural sources themselves. So results from curarized Ss have little relevance to this type of mediation argument. Until suggestions such as those of Black and Smith are made more specific, for example what exactly is a 'central movement process', and what is the specific anatomical location of the 'common source', it is difficult to see how this mediation argument can ever be either supported or refuted. The study of mediation becomes extremely difficult when central processes are proposed as the basis of the mediation.

In the history of Psychology there are a number of examples of the misdirection of research due to the posing of questions which lack

any possibility of empirical answer. The history of such questions as:

Is intelligence the result of heredity or environment?

and

What is learned?

shows that initially such questions appear simple and direct, but as more and more 'crucial' experiments designed to answer such questions are performed, the unanswerable nature of the question becomes apparent. After all of the research which has been done on the development of intelligence who, for example, would be prepared to state that intelligence is solely the result of heredity or environment? I suspect that the question, Can the heart be conditioned in the absence of skeletal responding?, is of a similar genre. Originally the use of curarized Ss seemed to offer a way of answering this question but difficulties of interpretation and analysis of the experimental results soon became apparent. In addition it is felt that too great a concern with problems of mediation may be unwise. The heart normally interacts with a number of other systems and so it must be remembered that the curarized S is a highly abnormal preparation, and so the generality of the results which are obtained using curarized Ss may not be very great. In the future it would seem more profitable, rather than continuing a seemingly never ending attempt to eliminate or reduce possible mediating factors, to direct research to the development of procedures which would facilitate the development of conditioned cardiac responses. A second promising approach would be to record skeletal concomitants - for example, respiration - of cardiac conditioning. This respiration data

could then be used in transfer equations similar to those published by Clynes (1960) which allow the prediction of heart rate from a knowledge of respiration. The difference between the heart rate predicted on the basis of the respiration change, and the actual change in heart rate, would allow an assessment of the effectiveness of the operant conditioning procedure.

In conclusion it is felt that curarization is a useful tool, and that the experiments reported in this dissertation represent a successful use of this procedure. But it must be remembered that it is only a tool and is not an end in itself.

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APPENDIXES

APPENDIX A

Tables I to VIII showing heart rates for the two experimental groups during successive one minute periods of adaptation, conditioning, and extinction for the four curare conditioning sessions. Criterion changes and number of brain stimulation reinforcements during conditioning periods.

TABLE I

Heart rate during successive one minute periods of adaptation, conditioning and extinction. Criterion changes and number of brain stimulation reinforcements during conditioning periods

Group 1: Heart rate increases reinforced
Session 1

Rat Number 2 C	6 B	7 C	10 C	4 D	5 D
491	442	505	528	492	394
491	442	505	531	490	394
481	441	505	525	484	394
481	441	504	523	483	394
487	439	501	513	489	389
489	439	500	494	487	389
489	430	497	496	474	389
488	430	497	484	482	393
486	417	491	460	482	393
488	417	490	463	468	393
487	428	489	462	452	389
487	428	489	470	454	388
488	427	486	479	462	383
488	430	480	484	457	379
486	429	483	474	455	378
486	427	482	478	467	380
488	427	480	484	480	380
488	426	481	491	475	377
488	426	478	489	473	381
488	415	473	487	472	379
487	411	471	.67 480 0	475	381
487	408	468	.68 466 0	471	377
487	405	472	.69 460 0	471	378
487	411	473	.70 466 3	471	378
487	411	472	.69 485 6	471	378
.66 488 0	.74 417 0	.71 480 1	.68 486 6	466	378
.67 486 2	424 1	480 1	.67 491 6	465	371
486 2	426 2	481 1	.65 496 4	466	358
486 2	431 3	481 1	496 4	462	359
486 2	430 2	482 1	496 5	467	361
487 1	429 5	482 1	.64 502 0	.62 475 0	.87 354 0
.68 484 3	437 6	481 1	501 0	473 0	.88 348 0
482 2	432 5	483 1	.65 494 2	.63 467 0	.89 356 1
480 3	432 1	483 1	.66 494 5	.64 453 0	355 1

TABLE I (continued)

Rat Number 2 C		6 B		7 C		10 C		4 D		5 D	
480	3	427	1	484	4	.65 492	0	464	2	.90 343	4
480	3	428	2	482	5	496	2	455	2	.91 342	4
478	0	431	3	483	0	495	2	463	0	352	4
478	5	434	6	483	2	.66 482	0	462	4	350	3
474	5	.73 435	5	.68 485	1	.67 482	3	457	2	353	5
473	6	430	1	482	2	.66 484	3	454	1	354	5
473	6	.74 423	1	487	1	484	1	.67 446	2	345	1
473	6	432	5	488	6	459		452	6	344	1
473	5	.73 435	4	489	4	~~457~~		.66 454	4	341	0
473	4	440	6	487	4	489		449	1	.92 341	0
472	0	.72 440	4	487	4	494		461	6	.93 339	1
.69 473	4	442	2	489	3	495		.65 460	4	343	3
473	4	442	4	488	2	497		451	1	351	6
473	4	442	3	493	4	493		461	5	340	4
.68 473	4	441	3	.67 487	2	493		.64 457	0	355	6
470	0	435	1	.68 486	2	488		.65 450	0	354	6
465		.73 431	3	490	2	486		454	0	354	3
461		434	3	486		487		.66 452	4	351	1
460		442	3	485		491		448	1	.94 351	6
458		.72 445	6	491		495		451	2	360	6
470		443	4	493		497		446	0	.93 361	1
466		430		494		497		.67 446	5	360	0
460		424		498		497		445		.94 358	5
456		418		497		495		447	5	356	4
453		418		491		496		450	2	357	2
449		418		495		494		457		373	
451		417		502		494		462		370	
449		413		495		493		464		366	
455		413		495		492		467		354	
455		417		496		491		459		350	
459		414		495		490		452		351	
455		414		494				437		349	
459		416		492				432		348	
459		412		497				438		350	
459		401		492				444		349	
457		397		490				444		353	
453		394		491				445		366	
463		390		489				441		352	
459		411		489				441		349	

TABLE I (continued)

Rat Number 2 C	6 B	7 C	10 C	4 D	5 D
462	415	488		442	345
458	412	487		439	347
457	412			442	342
455	417			447	339
	421			450	347
	423			450	349
	423			452	344
	409			460	338
	407			458	340
	405				341
	405				338

TABLE II

Heart rate during successive one minute periods of adaptation, conditioning and extinction. Criterion changes and number of brain stimulation reinforcements during conditioning periods

Group 1: Heart rate decreases reinforced
Session 1

Rat Number 1 B			6 C			8 C			9 C			9 D			10 D		
	540			479			311			567			455			480	
	540			481			318			563			457			478	
	539			481			329			560			450			481	
	535			485			330			562			444			484	
	536			485			333			561			449			484	
	532			485			339			561			451			484	
	533			482			341			562			447			482	
	533			482			348			561			449			482	
	533			477			351			555			452			478	
	529			477			353			551			450			477	
	533			475			387			550			442			478	
	533			475			392			559			444			472	
	531			479			404			559			446			479	
	533			482			409			554			444			481	
	529			482			414			554			442			482	
	531			483			419			545			446			483	
	531			483			418			537			441			482	
	527			484			420			549			437			477	
	529			484			423			526			441			479	
	527			481			422			538			439			478	
.61	512	4		481		.95	402	0		536			438			476	
	512	4		480		.90	381	0		536			441			475	
	510	3		480		.85	379	1		529			442			473	
	508	4		477			382	2		533			447			475	
	508	4		479			386	4		533			440			479	
	510	4	.70	468	0		386	2		538			437			481	
	510	4		470	3		386	2		536			439			482	
	510	3		467	5		387	3		529			438			484	
	507	4		466	4		386	5	.60	526	2		435			480	
	508	3	.69	470	2	.86	380	2		520	2		437			477	
.64	506	0	.71	457	3		383	2		520	4	.70	444	6	.69	480	0
	500	2	.72	457	3		380	3		512	4		442	6	.68	478	3
	510	3	.70	462	1		382	3		517	5	.71	448	4		472	6
.63	496	4		458	5	.87	378	1		519	0		447	5	.69	478	0

TABLE II (continued)

Rat Number												
1 B		6 C			8 C		9 C		9 D		10 D	
502	2	.72	449	4	378	1	520	0	.70	432	5	475	1
498	4		450	2	.88	363	5	518	1		436	4	469	4
498	4	.71	459	0	370	0	519	0		440	2	.70	468	2
498	4		450	5	.89	372	0	516	2		440	3	469	2
498	3	.72	452	1	372	3	523	0		439	5	.69	470	3
500	2	.71	454	3	.88	371	1	521	0		439	5	.70	467	6
500	4	.72	450	3	.87	373	1	518	1	.73	436	5	.71	465	4
501	3	.71	457	0	377	5	514	3		437	3	463	3		
501	3	.70	452	0	367	5	508	6		440	0	455	6		
501	2		455	5	.88	375	4	522	0		436	6	.72	457	3
499	4		454	6	373	2	525	0		434	6	462	0		
503	1		455	5	368	4	526	0	.74	434	2	.70	462	1	
503	1		456	6	.89	366	5	528	0	.72	440	2	458	5	
501	3	.71	453	3	.90	366	2	528	0		439	5	.71	454	5
501	1		454	1		364		527	0		437	3	.72	452	2
500	3		446	2		361		529	0		443	1		452	2
497			445	0		360		531	0	.74	439	3	.71	455	2
491			459	0		362		532			436	4		454	2
487		.70	456	5		364		532			424	5		449	4
485			460	6		366		532			418	5		459	1
485			460	5		368		534			406	5		473	0
484		.71	459	1		368		535			401	5		474	0
482		.70	461	1		368		537			405	6		478	0
484			451	6		370		536			397	6		483	0
479			452	5		368		534			434	4		464	4
476			452	3		367		534			443	0		444	1
471			441			369		535			424			461	
476			448			371		536			428			458	
477			452			371		537			428			457	
478			451			370		538			428			454	
476			452			373		538			424			455	
473			450			373		539			424			457	
469			450			374		540			424			459	
460			453			375		541			427			460	
466			453			373		543			428			458	
478			456			375		541			425			457	
498			455			374		541			423			458	
498			457			375		540			427			459	
494			457					540			428			460	
494			458					540			427			460	

TABLE II (continued)

Rat Number 1 B	6 C	8 C	9 C	9 D	10 D
482	461		540	426	461
480	457			428	462
	455			428	462
	452			429	462
	453			427	463
	451			425	467
	452			427	472
				431	474
				432	473
				428	471
				428	469
				427	471
				428	469
				430	473
				432	469
				432	466
				430	470

TABLE III

Heart rate during successive one minute periods of adaptation, conditioning and extinction. Criterion changes and number of brain stimulation reinforcements during conditioning periods

Group 1: Heart rate increases reinforced
Session 2

Rat Number												
2 C			6 B			7 C			10 C			
	492			450			417			526		
	493			455			413			521		
	494			455			415			518		
	496			461			414			516		
	496			461			413			510		
	496			461			410			479		
	497			468			412			489		
	500			468			413			494		
	498			483			417			486		
	501			483			418			476		
	498			482			417			475		
	494			473			417			479		
	494			459			416			486		
	497			459			396			483		
	500			459			394			473		
	497			471			401			477		
	497			471			411			461		
	498			471			414			467		
	502			471			408			476		
	496			459			409			476		
.65	496	0		460			406			472		
	496	0		460			406			462		
	496	0		468		.78	413	0		468		
.66	484	0		465		.80	408	0		471		
	486	0	.70	467	2	.82	410	4		474		
.68	480	0		469	4		412	3		476		
	476	0	.69	477	3		415	5	.68	471	0	
.69	480	0	.68	483	5		417	5	.69	470	0	
	482	1	.67	488	2	.83	417	5		473	2	
	484	3	.66	481	0		418	4		475	2	
	488	5	.67	491	3	.84	421	5		476	6	
.68	487	4	.66	493	3		419	5		478	0	
	487	4		494	2	.81	420	5	.68	479	1	
.67	489	4	.65	498	2		426	6		479	3	

4 D				5 D								
	485				382							
	486				387							
	480				383							
	474				387							
	463				389							
	470				379							
	461				376							
	458				376							
	456				378							
	450				382							
	447				375							
	454				375							
	459				379							
	456				376							
	457				381							
	455				380							
	454				374							
	456				376							
	455				373							
	457				373							
	455				372							
	450				372							
	447				365							
	451				363							
	457				363							
	452				366							
	450				363							
	452				367							
	447				363							
	456				363							
	455			.89	359	1						
.72	462	1		.93	364	2						
	465	4			372	6						
.71	462	1			376	6						

TABLE III (continued)

Rat Number 2 C		6 B		7 C		10 C		4 D		5 D	
484	0	500	0	.79 423	5	481	3	466	2	.91 371	6
488	3	498	1	426	5	481	3	464	2	.90 370	6
488	3	497	0	.76 427	4	483	4	460	0	372	5
485	2	499	5	423	5	482	2	468	4	374	6
487	2	490	5	430	3	479	2	469	3	.89 378	5
487	2	486	5	431	3	481	3	483	5	374	2
487	2	.67 487	5	430	1	.67 484	3	.69 481	5	374	2
486	1	.66 489	4	434	1	484	2	478	1	378	3
487	3	493	2	433	2	466	3	476	2	381	4
490	3	491	2	435	5	488	3	486	5	381	5
490	3	.65 495	2	.75 436	2	489	3	491	6	385	5
487	1	.66 499	5	441	4	486	3	.68 489	5	388	6
483	0	.65 497	0	438	2	486	4	492	6	.88 393	6
487	3	.66 500	4	439	4	486	3	.67 494	4	390	4
484	1	.68 475	0	433		485	3	492	0	391	4
480	0	.69 471	1	442		485	3	.68 493	6	401	3
480		483	4	442		479		488	6	397	4
479		.68 494	5	437		474		.67 488	0	403	5
479		486	5	437		469		483	0	404	6
480		486		438		463		.68 483	1	.87 404	6
480		484		438		460		484	0	409	6
475		483		437		452		483	0	406	3
478		481		444		452		485	1	.86 407	4
483		480		447		447		487	3	409	3
476		475		445		449		484	0	409	3
475		478		445		441		488	4	408	5
477		477		445		441		486	2	408	5
477		479		443		435		480		408	
475		480		441		439		479		406	
471		476		441		437		476		396	
478		478		445		430		477		396	
475		478		445		432		475		394	
476		479		447		434		478		394	
474		478		441		430		474		390	
474		479		441		430		474		387	
473		480		446		430		479		385	
472		478		448		432		487		384	

TABLE III (continued)

Rat Number 2 C	6 B	7 C	10 C	4 D	5 D
471	480		434	482	387
470	478		433	478	387
470	478		433	473	382
471	476		431	486	383
470	472		432	477	383
	470		432	475	381
	468		432	480	378
	464			478	380
	461			475	380
				476	379
				482	379
				472	378
				473	383
				470	384
				477	383
					384
					382
					375
					378
					381

TABLE IV

Heart rate during successive one minute periods of adaptation, conditioning and extinction. Criterion changes and number of brain stimulation reinforcements during conditioning periods

Group 2: Heart rate decreases reinforced Session 2												
Rat Number	1 B		6 C		8 C		9 C		9 D		10 D	
	490		402		395		536		463		440	
	478		412		399		534		457		428	
	468		413		393		535		457		420	
	466		413		387		534		458		420	
	463		413		383		533		455		425	
	462		413		385		532		448		431	
	461		415		385		531		447		417	
	466		414		382		527		445		413	
	460		414		384		525		451		410	
	448		413		382		526		446		408	
	437		413		380		525		437		408	
	434		415		384		523		437		404	
	429		413		382		522		431		405	
	448		414		382		521		427		402	
	461		409		383		522		425		412	
	472		409		381		519		416		412	
	459		408		377		519		411		414	
	455		410		381		518		409		415	
	445		410		377		517		412		416	
	447		412		379		518		409		414	
.73	456	0	411		.90	367	1	516		403		413
.71	461	1	.83	411	1	367	1	.63	515	0	400	412
.70	449	1	409	2	365	2	.61	516	2	399	411	
	449	1	.84	404	4	362	2	.63	517	2	400	411
	449	1	.85	402	0	.89	359	0	518	0	386	409
	442	2	.83	403	3	356	4	.62	515	2	350	409
	438	4	398	2	356	4	514	2	350	410		
	433	3	.82	403	1	.87	354	3	.63	506	2	346
	432	2	402	4	354	3	509	0	348	410		
	432	2	400	5	354	3	.62	511	0	347	408	
	432	2	398	5	352	5	.64	511	4	.89	352	0
	432	2	.83	399	0	355	4	501	0	.90	364	0
	430	6	402	3	355	4	499	0	363	2	409	2
.71	432	0	396	5	.86	356	5	.63	496	4	.91	359

TABLE IV (continued)

Rat Number												
1 B			6 C			8 C			9 C			9 D			10 D		
430	2		.85 392	4		354	4	.64	492	4		354	5		407	3
430	2		.84 397	0		354	3		494	2	.93	353	3		407	3
430	2		.82 399	2	.85	344	6		486	3		346	3		404	5
430	2		393	4		341	6	.65	481	5	.94	344	4	.85	402	3
428	4		392	5		344	3		479	5		344	0		403	0
.72 436	3		.83 393	4		351	0	.66	479	1		345	0	.84	403	3
426	6		398	3		337	6		482	2	.93	344	4		402	6
.74 432	0		.82 401	1	.84	334	4		482			342	5		397	4
433	0		395	5		334	4		488			340	6	.83	401	0
433	0		.83 397	1		331	6		487		.94	338	4	.82	403	3
433	0		.82 400	5		330	6		483			338	2		402	4
.72 432	2		395	4		334			485			334	4		400	4
432	2		402			337			487			335	3		398	5
434	1		404			341			490			336	2	.83	398	3
439			404			343			491			333	5		401	0
447			405			343			488			333	5		396	5
447			406			348			489			334	4		396	4
445			407			350			491			335	3		391	5
445			408			356			491			332	4	.84	397	0
443			408			357			493			333	5	.82	397	4
443			409			360			493		.95	332	0		394	5
447			409			359			490			334	0		389	6
451			408			358			492		.94	332	4	.83	398	3
451			407			362			492			328	6	.82	398	2
452			409			357			492		.95	329	2		393	5
455			408			359			491			327	5		392	3
456			409			369			491			329			393	
456			412			374			490			331			402	
456			410			373			490			327			401	
457			416			373			491			327			404	
458			415			369			491			329			396	
459			416									330			392	
460			417									330			403	
462			420									327			405	
461												329			406	
461												331			406	
460												332			408	
460												330			403	

TABLE IV (continued)

Rat Number 1 B	6 C	8 C	9 C	9 D	10 D
461				327	402
461				331	402
464				328	407
464				330	409
				333	411
				333	414
				332	415
				330	417
				328	411
				330	414
				330	413
				334	415
				333	418
				333	
				328	
				328	
				328	
				330	

TABLE V

Heart rate during successive one minute periods of adaptation, conditioning and extinction. Criterion changes and number of brain stimulation reinforcements during conditioning periods

Group 1: Heart rate increases reinforced
Session 3

Rat Number 2 C	6 B	7 C	10 C	4 D	5 D
487	526	502	482	486	380
487	516	505	477	476	376
497	498	509	472	475	373
493	484	508	483	474	375
490	483	505	474	465	373
491	467	504	460	465	371
489	466	503	460	460	371
493	464	504	448	444	368
494	468	506	442	451	367
488	459	503	441	457	365
492	466	501	439	451	369
488	465	500	436	433	375
486	461	472	435	424	376
486	460	469	432	439	374
489	466	459	432	448	373
492	469	459	426	460	376
487	463	467	426	464	376
484	462	475	429	451	378
488	462	478	430	450	377
484	460	473	428	455	379
.66 489 0	.68 472 0	.69 485 2	.78 411 0	454	380
.68 481 0	473 3	.68 487 4	.79 408 0	445	378
482 0	483 3	476 0	403 1	445	380
482 0	.67 489 3	.69 469 1	.78 419 4	451	380
482 0	493 3	481 3	406 0	452	376
476	493 5	.70 475 0	418 4	460	381
.70 487 6	494 0	477 4	.77 420 4	462	375
489 6	.66 498 2	.69 481 6	418 2	457	371
489 6	499 2	484 5	430 5	459	369
489 6	.65 502 3	.67 488 3	.76 423 3	.68 458 0	.93 375 6
.69 490 5	501 2	491 6	434 4	.70 467 5	.92 376 4
.67 494 3	497 0	494 1	445 6	.69 477 6	382 6
494 3	506 3	495 1	.74 462 5	.68 482 5	386 6
.68 496 4	.64 512 3	497 5	.72 465 7	.67 475 0	373 6

TABLE V (continued)

Rat Number 2 C	6 B	7 C	10 C	4 D	5 D
495 1	513 4	.66 500 2	.68 468 3	.69 468 2	372 6
495 1	518 3	.67 500 2	.69 466 1	473 5	.89 377 5
495 1	508 0	500 6	.70 462 1	.68 478 3	377 1
494 0	517 4	.66 507 5	460 1	.67 481 5	382 6
.67 496 5	.63 521 5	.65 505 0	461 2	.66 482 0	388 6
496 5	527 6	.66 509 6	465 5	.67 483 4	.88 385 0
.66 497 3	531 6	.65 507 3	.69 473 5	482 4	.89 388 4
497 3	.62 536 4	.66 506 6	.68 474 3	.66 480 0	390 6
498 3	539 3	506	.67 479 3	482 0	.88 394 6
497 3	541 5	506	480 0	480 0	390 3
495 2	540 3	508	.68 478 5	.67 483 4	398 6
498 6	546 2	508	476	.66 489 6	401 6
498 6	537	505	479	491 6	.87 400 1
500	527	506	480	.65 493 4	404 6
500	521	506	475	.64 489 0	404 6
501	505	506	477	.65 488 0	.86 408 2
498	505	506	475	485 0	407 2
499	500	508	475	.66 488 4	412 6
501	490	512	475	.65 489 3	410 6
502	482	506	463	.66 480 0	.85 415 4
500	482	504	453	467 0	411 1
502	474	493	462	.68 477 3	417 2
501	473	477	451	477 4	415 5
498	478	480	455	479 5	415 5
496	471	483	460	476	410
504	471	489	457	474	411
502	475	492	452	478	407
500	470	495	458	472	410
498	470		454	470	406
498	468		454	468	406
496	467		452	469	405
496	468		451	452	402
493	469		451	438	402
494			450	443	402
494				463	407
				468	402
				469	399
				470	400
				464	398
				455	396

TABLE V (continued)

Rat Number					
2 C	6 B	7 C	10 C	4 D	5 D
				455	395
				458	394
				454	395
				453	390
				461	390
					388
					385
					381
					389
					384
					382
					384
					386
					381

TABLE VI

Heart rate during successive one minute periods of adaptation, conditioning and extinction. Criterion changes and number of brain stimulation reinforcements during conditioning periods

Group 2: Heart rate decreases reinforced

Session 3

Rat Number 1 B		6 C		8 C		9 C		9 D		10 D	
500		447		485		529		442		462	
498		450		479		525		439		462	
494		442		480		528		435		457	
490		444		478		533		429		453	
486		456		480		532		425		454	
482		438		482		523		429		453	
477		454		484		521		426		451	
473		471		489		530		426		445	
485		468		481		533		427		447	
481		465		431		531		428		452	
478		462		407		533		431		442	
474		459		413		533		429		455	
474		449		429		528		431		456	
470		451		426		523		430		445	
477		448		421		529		430		436	
473		461		419		523		430		446	
469		454		412		521		428		456	
467		463		415		526		428		468	
465		455		417		524		426		472	
462		457		408		524		425		464	
465	.70	462	0	417		.64	496 4	427		453	
464		462	0	410		.66	507 0	427		455	
464		457	1	409		.64	511 1	427		452	
464		454	4	413		.63	519 0	428		450	
.72 453	5	448	6	81.411	0	.62	511 5	423		448	
438	4	448	6	411	0	.63	505 5	423		454	
.73 415	4	.72 436	5	409	0		510 3	424		447	
.76 398	4	439	1	407	0		514 1	427		447	
.78 400	5	439	1	407	0		517 3	431		448	
.79 411	0	436	5	405	1		500 4	427		447	
410	1	436	6	.79 409	0		491 4	426		447	
.78 418	1	.74 439	1	414	0	.66	483 6	.81 426	4	.71 458	0
411	3	439	1	411	1		489 3	420	6	.70 454	4

TABLE VI (continued)

Rat Number												
1 B			6 C			8 C		9 C			9D			10 D	
	403	3		438	3		404	4		495	1	.82	413	5		448	6
	429	0		437	4		406	2	.65	497	2		414	2	.69	445	2
.77	436	1		444	0		408	1	.64	503	5		410	6		442	6
	438	1		445	0		407	1		505	0	.83	411	1	.68	448	3
	427	2		448	0		407	1	.63	505	1		412	0		450	0
	428	2	.72	445	0		407	1		507	5	.82	410	5		441	6
	423	2		434	6		407	2		508	4	.83	405	3		440	6
	421	3	.74	445	0		407	2		510	3		406	6	.67	434	4
	421	3		442	1		407	2		509	4	.84	402	0		433	5
	415	2		437	4		402	3		507	4	.83	402	6	.66	432	0
	416	2		434	2		394	6		510	5	.84	398	5		433	1
.78	401	5	.72	433	6		389	6		506	5		396	6		429	3
.79	412	1		430	6		385	6		509			402	0		426	5
	413	0		430	6		382	6		508		.83	402	3	.65	424	6
.78	410	3		432	5		409			508			398	5	.66	428	1
	408	4		433			407			506			399	5		428	2
.79	397	4		434			410			506			394	5		428	4
.80	401	3		438			415			499		.84	393	6		428	6
.81	396	3		442			415			489			398	0	.65	421	6
	396	4		444			420			497		.83	399	0		423	6
	384			446			415			503		.82	393	5	.66	420	5
	388			442			416			499			394	5		419	6
	382			448			414			500			398	6	.67	415	6
	387			450			415			501		.83	394	1		413	6
	384			451			415			502			391	2		413	6
	385			447			415			501			391	3		409	6
	384			446			418			504			396	6		410	
	379			449			420			502			397	3		411	
	373			450			425			509			388			413	
	378			447			430			510			389			414	
	382			453			430			499			389			414	
	376			456			435			509			390			416	
	382			462			437			516			389			418	
	371			458			440			517			389			415	
	379			450			438			520			389			414	
	375			460			436			526			389			414	
	369			462			430			509			389			414	
	375			468			430			508			393			420	

TABLE VI (continued)

Rat Number 1 B	6 C	8 C	9 C	9 D	10 D
376	468	430	520	393	420
381	471	432	526	393	422
382	469	430	531	393	428
384	469		531	393	428
387				393	428
385				390	432
				391	431
				388	435
				393	435
				394	438
				394	440
				393	444
				392	447
				394	448
				393	455
				393	459
				393	455
				392	460
				390	460
				385	461
				383	461
				380	

TABLE VII

Heart rate during successive one minute periods of adaptation, conditioning and extinction. Criterion changes and number of brain stimulation reinforcements during conditioning periods

Group 1: Heart rate increases reinforced
Session 4

Rat Number 2 C		6 B		7 C		10 C		4 D		5 D	
498		485		399		481		453		431	
486		484		417		481		457		423	
484		481		415		470		451		412	
477		470		424		450		454		410	
479		466		419		469		451		404	
472		476		423		461		463		402	
477		476		438		452		464		395	
476		471		436		458		463		385	
482		472		440		473		464		386	
482		470		446		473		449		397	
479		468		452		470		447		394	
476		468		460		475		445		397	
464		464		466		473		439		407	
450		466		466		465		435		412	
433		466		468		461		428		407	
442		463		469		462		430		393	
450		460		470		467		435		397	
440		468		471		470		429		399	
438		470		469		472		440		401	
435		470		466		469		439		410	
439	.69	471	0	.69	461 0	.66	473 0	434		411	
456	.70	471	3	.70	466 1	.70	466 1	428		407	
456	.69	479	5	.71	466 2		462 0	424		408	
462	.68	481	1		460 1		462 1	434		412	
470	.69	475	1		459 2		468 4	431		401	
468	.68	482	3		463 4	.69	469 2	435		401	
466		480	0	.69	470 4	.70	456 0	436		409	
466		482	0		465 0		456 0	425		409	
.71 467	0	487	1	.70	466 2		465 5	426		412	
461	2	490	3		475 4		458 5	424		409	
463	1	491	3	.71	475 4		475 3	.72 402	0	.72 413	0
469	2	494	5	.69	478 5		479 4	.82 406	4	.76 413	0
457	1	484	1		479 3		477 2	.80 409	4	.80 411	4
473	1	.69 474	0	.68	476 2		472 1	414	3	.79 414	6

TABLE VII (continued)

Rat Number 2 C		6 B		7 C		10 C		4 D		5 D	
	475	2	472	3	476	0	473	1	415	4	414	6
.69	476	1	477	1	.69	476	3	.68	473	0	416	6	412	2
	478	3	475	2		473	2	.69	469	4	416	6	411	0
	478	3	475	2		473	2		475	6	418	1	411	0
	480	3	484	5	.71	478	4	.68	474	2	416	0	414	2
	482	4	486	6		488	6		475	0	416	0	416	4
.68	480	1	488	6		498	6		471	2	418	1	421	5
	483	0	484	4		508	6		472	4	419	2	425	4
	481	1	486	5		418	7		476	3	421	3	421	3
	481	0	486	4		498	6		478	5	427	6	419	2
	479	0	481	4		498	6	.67	481	2	427	6	413	1
	478	4	464			478			482	3	.78	431	3	413	1
.70	478	4	450			470			483	1		431	3	415	1
	473		455			466			483	3		433	4	.80	412	1
	462		459			476			482	1	.77	435	2		412	1
	474		462			474			483	1		435	2		411	0
	479		450			475			485	3		431	0		415	3
	482		443			471			482			431	0		416	4
	473		440			476			485			437	6		418	4
	474		433			469			487			437	6		418	4
	470		430			472			487			435	3		415	
	471		439			471			488			431			411	
	473		441			470			489			429			407	
	472		445			470			489			429			403	
	469		445			469			490			427			395	
	469		443			468			488			423			391	
	472		441			466			487			421			394	
	467		444			466			488			419			394	
	465		437			467			484			421			391	
	464		436			466			468			418			391	
	463		435			464			476			421			386	
	462		429			461			474			415			390	
	461					461			471			417			387	
	460					460			471			419			393	
	461					460			471			411			393	
	461								469			413			396	
	459								469			417			388	

TABLE VII (continued)

Rat Number 2 C	6 B	7 C	10 C	4 D	5 D
458			468	419	392
457			467	417	393
				417	394
				421	396
				417	400
				420	401
				422	393
				424	389
				424	391
				419	388
				425	390
				424	

TABLE VIII

Heart rate during successive one minute periods of adaptation, conditioning and extinction. Criterion changes and number of brain stimulation reinforcements during conditioning periods

Group 2: Heart rate decreases reinforced
Session 4

Rat Number 1 B	6 C	8 C	9 C	9 D	10 D
519	455	421	521	373	468
519	462	421	520	373	462
518	475	419	519	371	468
518	476	418	518	368	477
516	470	416	517	367	482
517	475	418	517	366	480
514	466	421	519	363	481
515	474	412	516	359	478
516	481	407	517	358	475
514	491	412	513	356	473
515	488	406	512	375	469
510	488	409	514	354	475
509	492	413	521	354	481
510	494	412	519	352	478
510	495	416	515	350	482
501	497	420	511	347	477
501	491	413	511	351	477
497	488	413	510	347	482
488	492	408	514	342	475
489	494	.89 397 2	512	341	466
490	494	396 3	516	340	468
490	491	393 4	511	337	471
.67 490 2	489	397 2	507	333	475
482 5	500	392 6	507	333	480
.68 478 5	.68 475 5	.87 390 4	.62 511 5	331	474
476 4	.72 454 3	.86 386 3	.63 502 6	331	474
.69 472 5	.73 455 4	.85 390 0	.64 501 6	329	477
.70 474 0	463 1	384 1	504 2	329	473
472 0	.72 478 0	383 2	.63 504 3	334	475
438 4	.71 477 1	.86 382 3	498 6	330	477
453 5	.69 485 3	381 4	.64 497 5	.99 326 4	.70 475 0
.71 452 4	486 4	379 5	495 5	324 3	474 0
.72 445 4	481 6	.87 376 3	.65 494 5	323 4	.71 468 4

TABLE VIII (continued)

Rat Number												
1 B			6 C			8 C		9 C			9 D			10 D	
.73	443	5		480	5	376	1		497	2	1.00	321	5		470	2
.74	438	4	.70	491	0	375	2		492	6		322	4	.70	458	4
.76	435	4	.70	491	0	373	3	.66	486	6	1.01	320	3	.74	452	2
	424	5		499	4	373	3		484	4	1.00	322	0		454	1
.78	412	4	.68	500	3	375	1		483	5		322	4	.73	460	1
	410	4		478	6	372	3	.67	483	2		323	5	.74	460	2
.80	407	4	.70	483	2	378			482	3		328	4		461	1
.82	410	2	.68	484	3	380			482	3		325	4		458	2
.81	406	2		480	4	382		.66	483	1		316	6		459	3
	406	1		478	4	382			483	1		316	5	.71	454	5
	416	0	.69	478	3	382			482	3		316	3	.72	451	3
.79	420	1	.68	487	2	384			481	3		316	3		466	1
	427	1	.67	489	3	384		.65	480	3		314	6		459	5
.76	433	2	.66	490	3	389			479	4		316	3	.74	452	5
	429	2		485	6	385			479	4		313	7		458	2
	449			484	4	389			482	1		311	3		460	2
	471			503		387			483	1		309	4		458	4
	472			504		391			482			318			460	2
	469			504		387			482			316			454	6
	470			504		386			484			316		.75	454	0
	471			509		383			486			316		.74	452	4
	468			514		384			488			314			451	6
	468			516		386			486			314			450	6
	472			513		387			488			313		.75	448	5
	483			519		388			490			314			449	3
	480			509		391			490			312			449	3
	474			504		387			490			311			448	4
	472			501		389			491			312			454	
	477			510		387			489			310			450	
	476			499		382			491			310			448	
	474			502		385			492			309			454	
	471			502		392			489			308			454	
	474			499					488			309			452	
	471			496					491			308			449	
	477			497					492			306			447	
	474			499					496			307			451	
	474			501					494			306			449	
				502					498			307			451	

TABLE VIII (continued)

Rat Number					
	6 C	8 C	9 C	9 D	10 D
	503		499	307	454
	504		498	306	456
			497	306	455
				308	449
				308	454
				308	461
					462
					464
					464
					462
					461
					457
					456
					462

APPENDIX B

OPERANT CONDITIONING OF BOTH INCREASES AND DECREASES IN HEART RATE IN THE SAME RAT

Since the results of the first experiment reported in this dissertation provided clear evidence of operant conditioning of both increases and decreases in heart rate in groups of curarized rats, a second experiment was performed. In this experiment an attempt was made to condition both increases and decreases in heart rate in the same rat. Two rats were used. The following changes were made in the procedure employed in this second experiment.

1. The rats were trained in the morning, with a curare session never starting earlier than 8:00 A.M. or later than 11:00 A.M. Since rats have clear diurnal activity cycles (Munn, 1950) controlling the time of the curare sessions represented a procedural improvement.

2. Curare sessions were separated by a fixed interval of four days. In contrast to this fixed interval, in the first experiment curare sessions were separated by varying intervals of from three to six days.

3. Following the sessions in the Skinner box in which the reinforcing properties of the brain stimulation were assessed, the rats were given a curare adaptation session during which they were curarized and their heart rates recorded, but the contingencies were not in effect and so neither the feedback stimulus or the brain stimulation was presented. The introduction of this adaptation curare

session provided baseline data against which the conditioned changes in heart rate could be evaluated.

4. The adaptation and extinction phases of the curare conditioning sessions lasted for thirty minutes and the conditioning phase lasted for forty minutes. This procedure eliminated the small variation in the lengths of the different phases of the curare sessions which was present in the first experiment.

Subjects

Two, male Sprague-Dawley rats were used in this experiment. They were housed and maintained under conditions identical to those of the first experiment.

Implantation Procedure

The implantation operation and the procedure used to assess the reinforcing properties of the brain stimulation were identical to those of the first experiment.

Curare Sessions

The procedure used during the curare sessions was similar to that of the first experiment but with the additional controls which have been mentioned. The first curare session was an adaptation session during which the contingencies were not in effect and no stimuli were presented. During this period the rats' heart rates were recorded each minute for one hundred minutes. Four days later the rats were given their second curare session, during which the reinforcement

contingency was in effect. One of the rats was reinforced for increases in heart rate, and one for heart rate decreases. The decision as to the direction of the heart rate changes to be conditioned first in each rat was made on the basis of the toss of a coin. The first thirty minutes of the curare conditioning session was an adaptation phase during which no stimuli were presented. During the next forty minutes a conditioning procedure identical to that of the first experiment was employed. This conditioning phase was followed by a thirty minute extinction phase. During all three phases of the session the rats' heart rates were recorded during successive one minute periods.

The conditioning procedure was used for three curare sessions each of which had thirty minute adaptation and extinction phases, and a forty minute conditioning phase. All curare sessions were separated by four days. The numbers of heart beats for the two rats used in this experiment during successive minutes of the first four curare sessions are shown in Tables XIV to XX of Appendix C. In addition criterion changes and number of brain stimulation reinforcements during conditioning are also shown in these tables. In order to present the changes in heart rate which occurred in the two rats during each of the first four curare sessions mean heart rates were calculated for each of the rats during ten blocks of adaptation periods in the case of the curare adaptation session, and three blocks of adaptation periods, four blocks of conditioning periods, and three blocks of extinction periods during the three curare conditioning sessions. Unlike the first

experiment in this experiment each of these blocks of periods represented ten minutes of the curare session. These mean heart rates are shown in Tables IX and X and are presented graphically in Figures 4 and 5.

TABLE IX

Mean heart rates in beats per minute during blocks of adaptation, conditioning and extinction periods

	Session 1 Adaptation		Session 2 Conditioning	
Rat No.	2 E H.R. Incr. Reinforced	3 E H.R. Decr. Reinforced	2 E H.R. Incr. Reinforced	3 E H.R. Decr. Reinforced
Adap.	511	517	Adap. 521	542
Adap.	524	514	Adap. 515	552
Adap.	522	514	Adap. 510	534
Adap.	520	516	Cond. 490	539
Adap.	520	512	Cond. 496	549
Adap.	515	508	Cond. 495	546
Adap.	509	504	Cond. 498	540
Adap.	507	506	Ext. 500	551
Adap.	505	512	Ext. 495	540
Adap.	505	508	Ext. 494	534

TABLE X

Mean heart rates in beats per minute during blocks of adaptation, conditioning and extinction periods

	Session 3 Conditioning		Session 4 Conditioning	
Rat No.	2 E H.R. Incr. Reinforced	3 E H.R. Decr. Reinforced	2 E H.R. Incr. Reinforced	3 E H.R. Decr. Reinforced
Adap.	510	517	486	474
Adap.	515	512	486	465
Adap.	524	520	491	459
Cond.	547	519	490	453
Cond.	550	522	509	450
Cond.	555	518	504	446
Cond.	554	514	522	436
Ext.	542	513	533	439
Ext.	530	515	517	446
Ext.	527	518	506	448

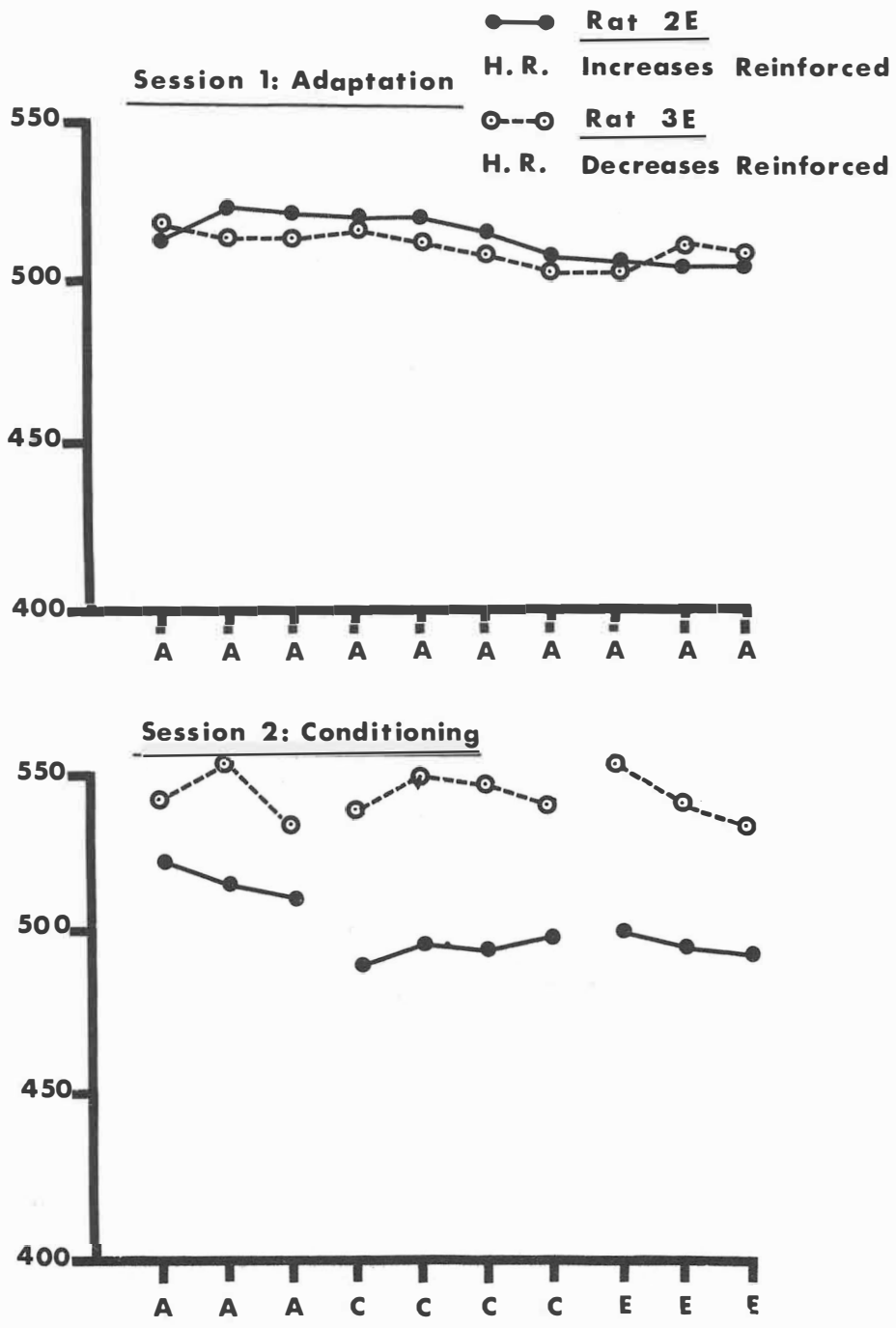

Figure 4. Mean heart rates during ten 10 minute blocks of one minute periods in Session 1, and mean heart rates during three blocks of adaptation periods, four blocks of conditioning periods, and three blocks of extinction periods in Session 2.

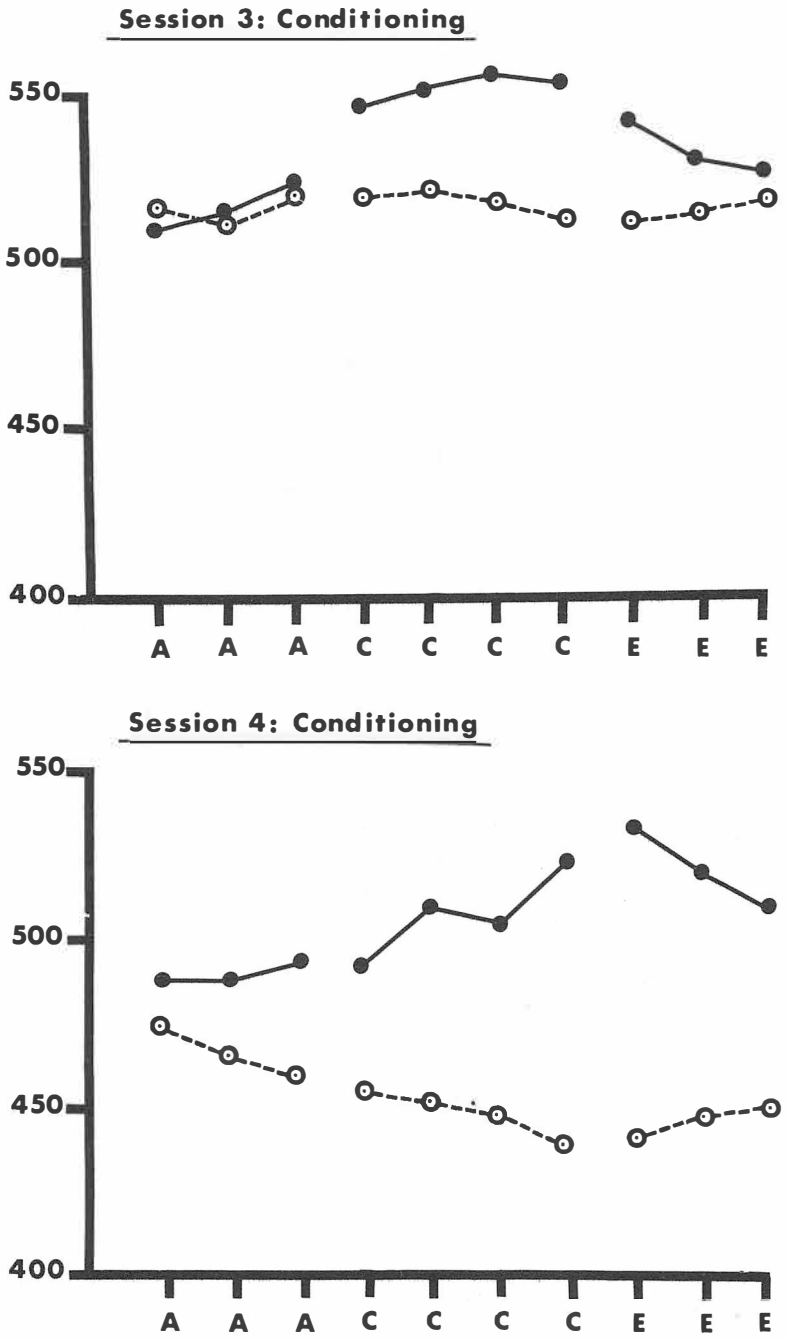

Figure 5. Mean heart rates during three blocks of adaptation periods, four blocks of conditioning periods, and three blocks of extinction periods in Sessions 3 and 4.

During the adaptation curare session both groups showed a small over-all decrease in heart rate. The two rats showed little if any evidence of operant conditioning of either an increase or a decrease in heart rate during the first curare conditioning session. On the third curare session there was some evidence of operant conditioning of changes in heart rate, and on the fourth session clear heart rate changes were conditioned. During the fourth curare session rat number 2 E showed an increase in heart rate of 31 b.p.m. during conditioning, and rat number 3 E a decrease of 23 b.p.m. So the results for these two rats in this second experiment provide additional evidence of operant conditioning of both increases and decreases in heart rate in curarized rats.

The fifth curare session was a reversal conditioning session during which the following procedure was adopted. As usual the session began with a thirty minute adaptation session. This was followed by a twenty minute conditioning phase during which the two rats were reinforced for heart rate changes in the direction which had previously been conditioned. During these conditioning periods rat 2 E showed a clear increase in heart rate, and rat 3 E a decrease. A ten minute extinction period, during which the contingencies were not in effect, followed these conditioning periods. A thirty minute reversal conditioning phase followed these extinction periods. During this reversal phase of the curare session the rat which had previously been reinforced for heart rate increases - 2 E - was reinforced for heart

rate decreases, and the rat which had previously been reinforced for heart rate decreases - 3 E - was reinforced for heart rate increases. During the reversal conditioning periods the feedback stimulus was changed so that criterion IBIs - long IBIs in the case of rat 2 E, and short IBIs in the case of rat 3 E - were followed by a high frequency tone, and non-criterion IBIs were followed by a low frequency tone. It was hypothesized that changing the modality of the feedback stimulus and making it more informative would facilitate acquisition of the heart rate reversal. The reversal conditioning phase of this session lasted for thirty minutes and was followed by a twenty minute extinction phase. The results for the reversal conditioning session are shown in Table XI and are presented graphically in Figure 6.

Although both rats showed heart rate changes in the direction being conditioned during the first set of conditioning periods of this session, they showed no evidence of acquisition of the reverse heart rate change during the reversal conditioning periods. In fact rat 2 E which was being reinforced for heart rate decreases during the reversal periods, showed an increase in heart rate, while rat 3 E which was being reinforced for heart rate increases during the reversal periods, showed a heart rate decrease. So during the reversal phase of this session both rats continued to show heart rate changes in the direction which had been conditioned during the earlier curare conditioning sessions, in spite of the change in the direction of the heart rate change which produced the brain stimulation reinforcement. It seemed possible that

TABLE XI

Mean heart rates in beats per minute during blocks of adaptation, conditioning and extinction periods

Session 5 Reversal Conditioning			
2 E		3 E	
Adaptation	456	Adaptation	487
Adaptation	458	Adaptation	482
Adaptation	455	Adaptation	464
Cond. H.R. Incr. Reinforced	470	Cond. H.R. Decr. Reinforced	467
Cond. H.R. Incr. Reinforced	486	Cond. H.R. Decr. Reinforced	447
Extinction	467	Extinction	435
Cond. H.R. Decr. Reinforced	479	Cond. H.R. Incr. Reinforced	443
Cond. H.R. Decr. Reinforced	476	Cond. H.R. Incr. Reinforced	443
Cond. H.R. Decr. Reinforced	479	Cond. H.R. Incr. Reinforced	439
Extinction	487	Extinction	452
Extinction	481	Extinction	452

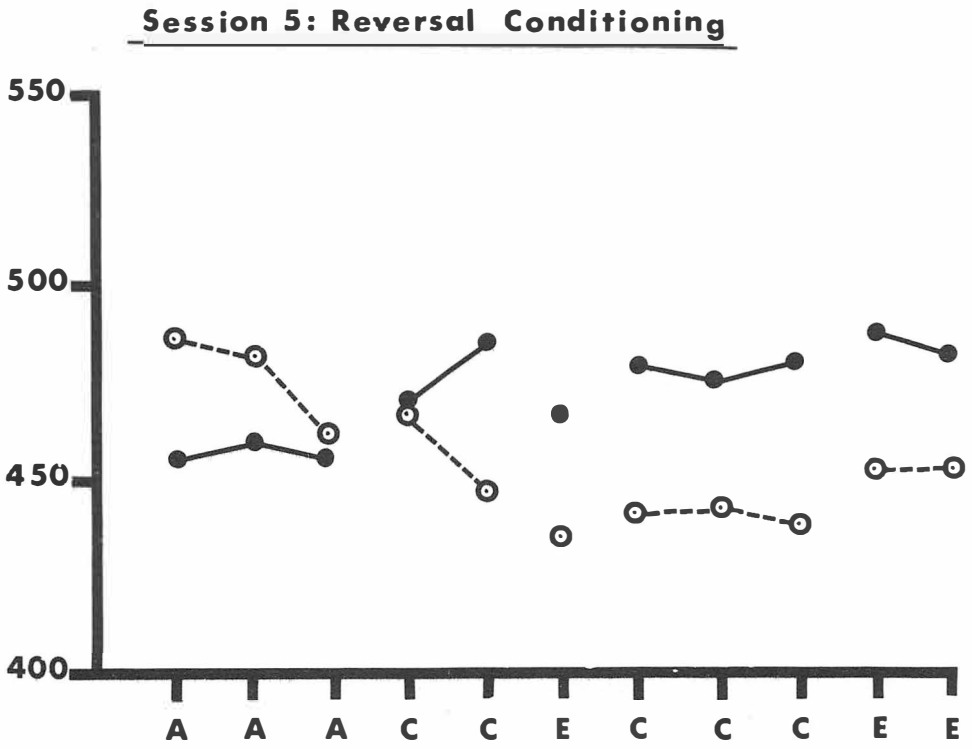

Figure 6. Mean heart rates during three blocks of adaptation periods, two blocks of conditioning periods, one block of extinction periods, three blocks of reversal conditioning periods, and two blocks of extinction periods in Session 5.

further conditioning might lead to acquisition of the heart rate reversal and so two further curare sessions were given during which the reversal conditioning procedure was employed. The results for these two curare reversal conditioning sessions are shown in Table XII and are presented graphically in Figure 7.

The only suggestion of any acquisition of the reversal response was the performance of rat 2 E during the sixth curare session. During the conditioning phase of this session rat 2 E, which was being reinforced for decreases in heart rate showed a heart rate decrease of 14 b.p.m. However during the seventh curare session the rat showed an increase in heart rate of 18 b.p.m. during the reversal conditioning phase. On both of the last two curare sessions rat 3 E showed no evidence of acquisition of the heart rate reversal - an increase in heart rate. On the sixth curare session rat 3 E showed a heart rate decrease of 9 b.p.m., and on the seventh curare session a heart rate decrease of 5 b.p.m. So this rat continued to show during the reversal conditioning curare sessions the heart rate changes which had initially been conditioned.

Although the results for these two rats show good initial acquisition of both a heart rate increase and a heart rate decrease, thus replicating the results of the first experiment, they provide no evidence of any acquisition of the heart rate reversal. However the reversal response which was required in this experiment was a change in heart rate opposite to that which had originally been

TABLE XII

Mean heart rates in beats per minute during blocks of adaptation, conditioning and extinction periods

	Session 6 Reversal Conditioning			Session 7 Reversal Conditioning	
Rat No.	2 E H.R. Decr. Reinforced	3 E H.R. Incr. Reinforced		2 E H.R. Decr. Reinforced	3 E H.R. Incr. Reinforced
Adap.	504	508	Adap.	480	515
Adap.	485	494	Adap.	478	499
Adap.	487	472	Adap.	467	487
Cond.	488	469	Cond.	474	474
Cond.	480	468	Cond.	476	463
Cond.	477	448	Cond.	479	464
Cond.	474	460	Cond.	482	469
Ext.	507	447	Ext.	481	479
Ext.	485	444	Ext.	478	480
Ext.	485	436	Ext.	474	479

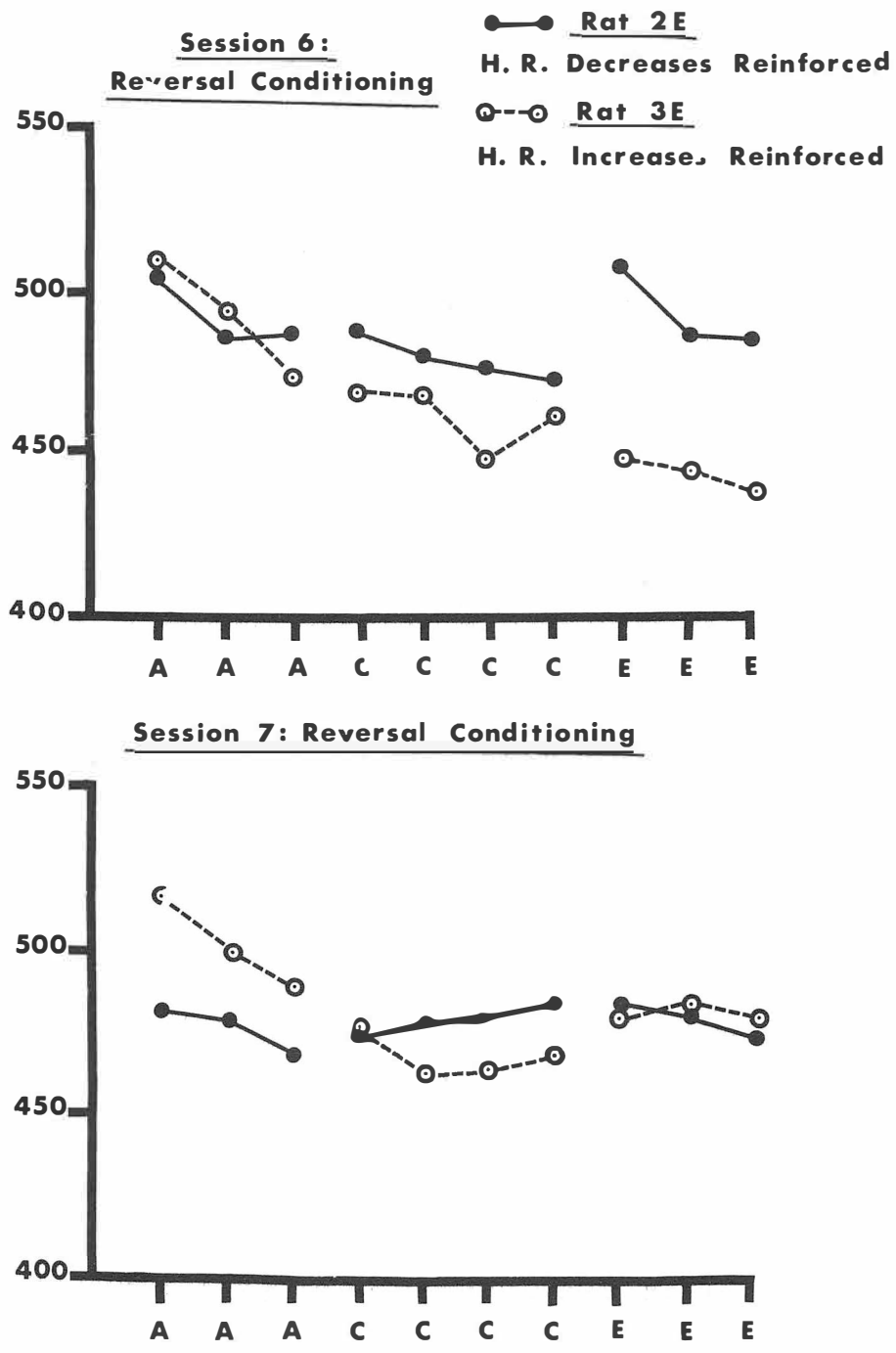

Figure 7. Mean heart rates during three blocks of adaptation periods, four blocks of conditioning periods, and three blocks of extinction periods in Sessions 6 and 7.

conditioned. Difficulty in conditioning such a heart rate reversal was not unexpected since even with a conventional operant response such as lever pressing, it is anticipated that considerable difficulty would be experienced in training a rat which had previously been reinforced for high rates of responding to respond at a lower rate. The results from these two rats do however strongly suggest that considerable difficulty will be experienced in conditioning both increases and decreases in heart rate in the same S.

This suggestion is confirmed by the results from one additional rat in which an attempt was made to condition a heart rate reversal. It was hypothesized that a stimulus which was present during the initial conditioning, but not during the reversal conditioning, would facilitate acquisition of the reversal heart rate response. For this rat - number 5 E - a procedure similar to that of rats 2 E and 3 E was used except that:

1. A light was present during the initial conditioning periods, but not during the reversal conditioning periods. It was hypothesized that making the two sets of conditioning periods more discriminable would facilitate acquisition of the heart rate reversal.

2. During the initial conditioning periods the rat was reinforced for heart rate increases but no feedback stimuli were presented.

3. The fifth and sixth curare sessions were reversal conditioning sessions during which heart rate decreases were reinforced. During these two sessions criterion IBIs were followed by a high frequency tone, and non-criterion IBIs by a low frequency tone.

The results for the adaptation curare session, the second, third and fourth conditioning sessions, and the fifth and sixth reversal conditioning sessions are shown in Tables XXI to XXIII of Appendix D, and are summarized in Table XIII and are presented graphically in Figures 8, 9 and 10.

On the third and fourth curare conditioning sessions this rat showed a clear increase in heart rate during conditioning - an increase of 33 b.p.m. during the conditioning phase of the third session, and an increase of 45 b.p.m. during the conditioning phase of the fourth session. So once again these results show operant conditioning of a heart rate change in a curarized rat. However on the fifth and sixth curare reversal conditioning sessions, during which heart rate decreases were reinforced, this rat continued to show an increase in heart rate. So with this rat, as was the case with rats 2 E and 3 E, there was no evidence of acquisition of the heart rate reversal. Although with extended conditioning it may be possible to condition heart rate reversals the results from these three rats strongly suggest that such heart rate reversals will only be conditioned with great difficulty.

TABLE XIII

Mean heart rates in beats per minute during blocks of adaptation, conditioning and extinction periods

Sessions	1		2	3	4	5	6
Adaptation		H. R. Increases		Reinforced		H.R. Decr.	Reinforced
Adap.	468	Adap.	452	477	461	455	464
Adap.	460	Adap.	462	459	459	453	458
Adap.	463	Adap.	440	445	455	450	456
Adap.	452	Cond.	448	422	442	471	471
Adap.	452	Cond.	447	447	456	466	473
Adap.	456	Cond.	440	453	470	469	477
Adap.	462	Cond.	447	455	487	469	479
Adap.	459	Ext.	453	460	487	467	474
Adap.	455	Ext.	459	463	491	458	465
Adap.	460	Ext.	466	457	482	451	458

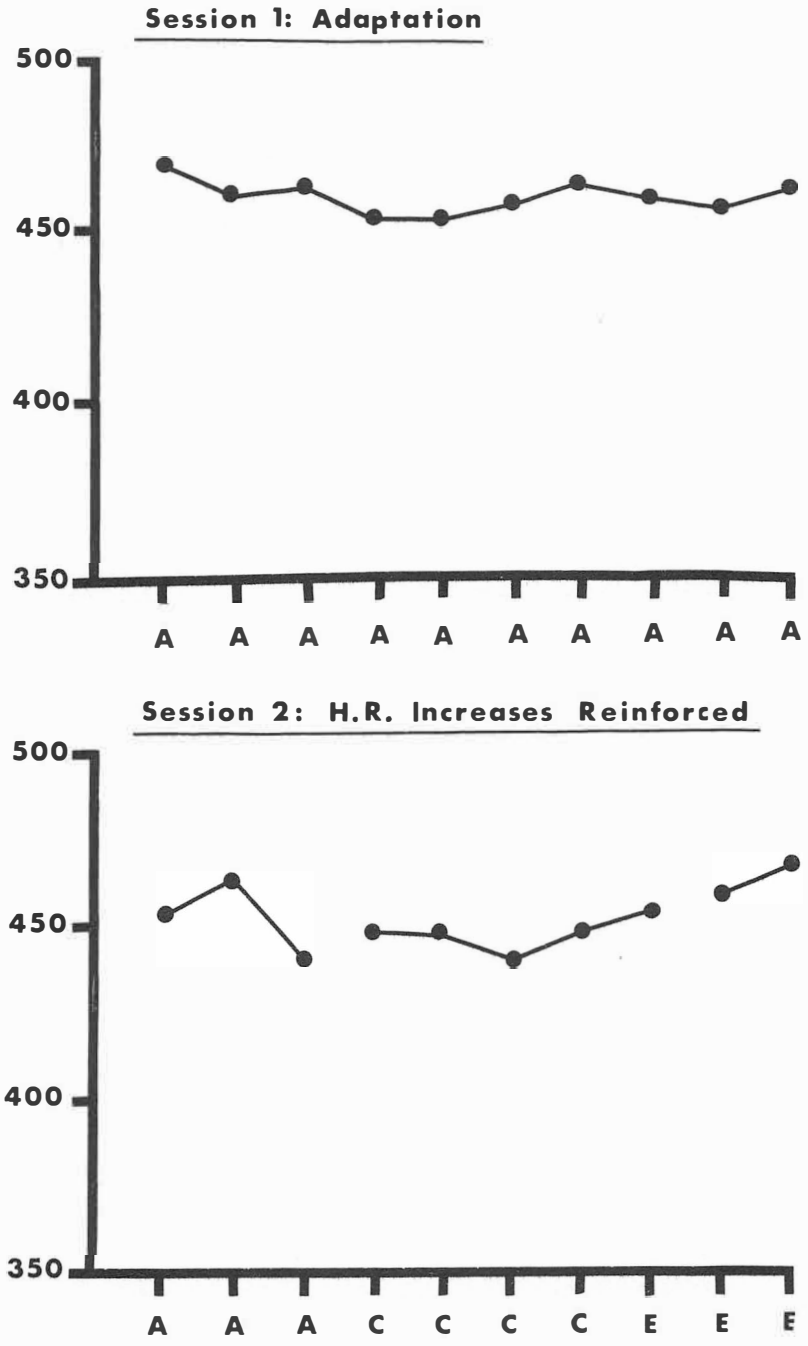

Figure 8. Heart rates during blocks of adaptation, conditioning and extinction periods. Adaptation and Session 2.

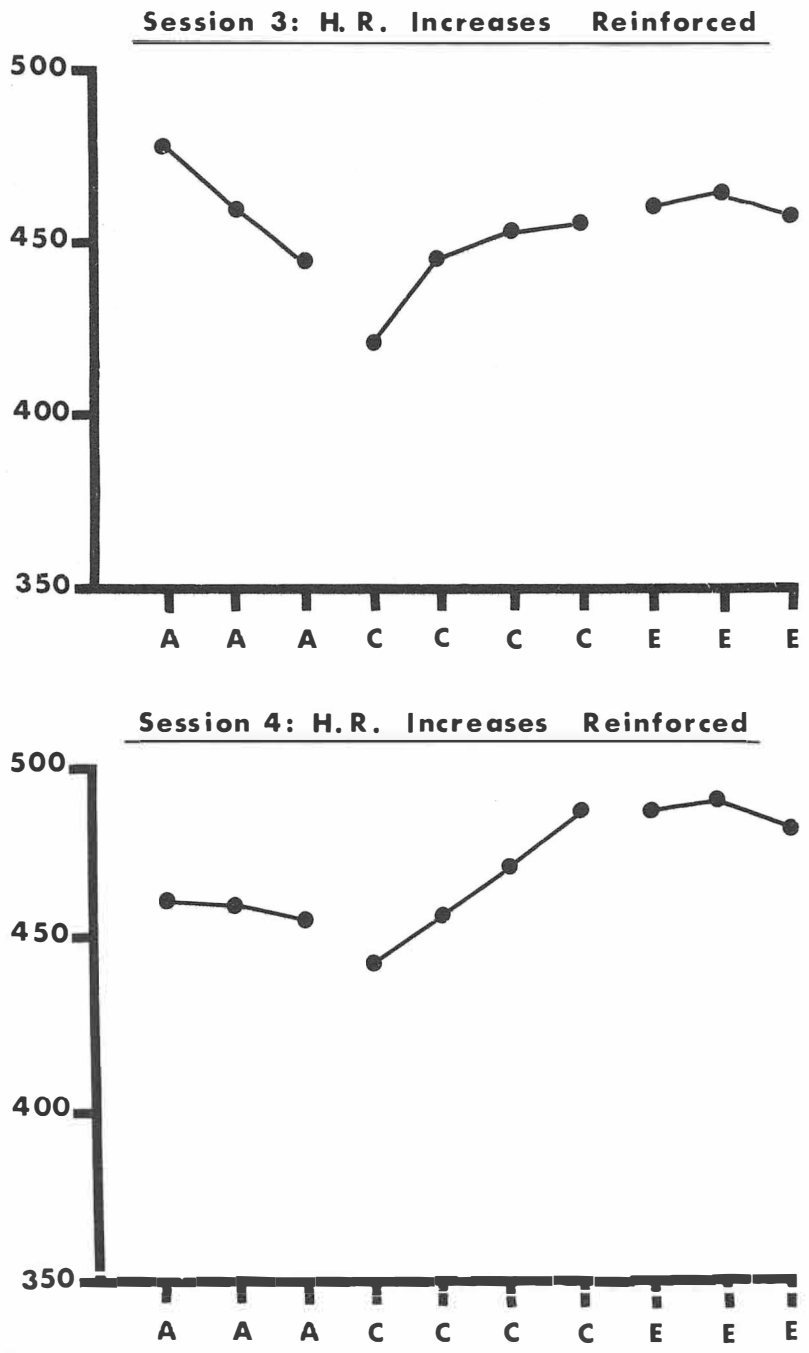

Figure 9. Heart rate during blocks of adaptation, conditioning and extinction periods. Sessions 3 and 4.

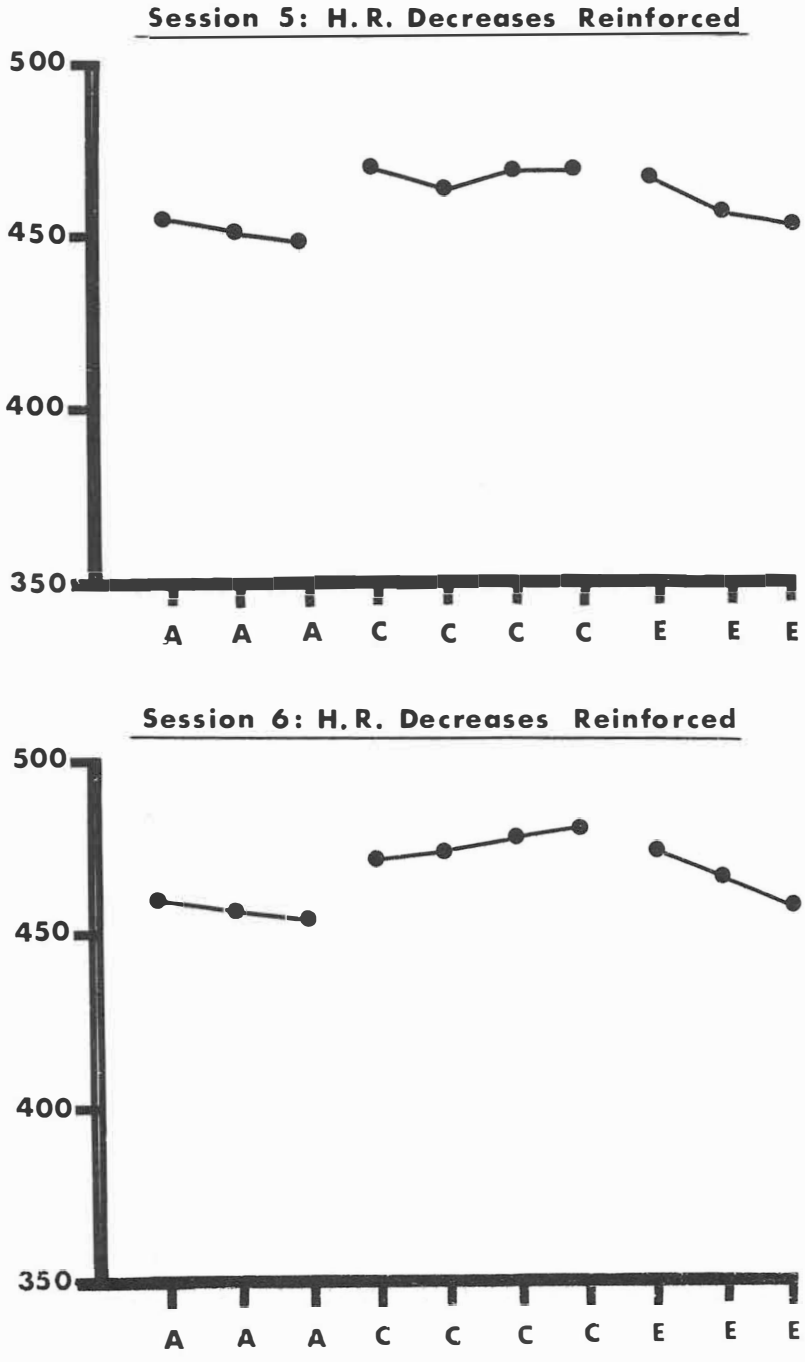

Figure 10. Heart rate during blocks of adaptation, conditioning and extinction periods. Sessions 5 and 6.

APPENDIX C

Tables XIV to XX showing heart rates for rat numbers 2 E and 3 E during successive one minute periods of one adaptation session, three conditioning sessions and three reversal conditioning sessions. Criterion changes and number of brain stimulation reinforcements during conditioning periods.

TABLE XIV

Heart rate during successive one minute periods of Session 1

Rat Number 2 E Adaptation			Rat Number 3 E Adaptation		
558	523	505	524	515	513
556	523	507	527	515	513
553	521	506	523	514	512
541	520	504	529	511	511
523	518	505	507	512	510
489	521	504	518	510	509
473	520	505	513	512	513
481	518	504	511	510	512
503	519	506	512	511	512
523	519	506	507	512	511
524	518	505	511	509	510
523	517	505	517	508	508
523	516	506	523	507	506
522	517	504	519	507	508
523	517	505	511	507	506
522	514	505	509	507	510
524	515	507	505	508	508
527	511	503	511	511	509
527	511	505	513	512	507
526	512	505	517	508	508
524	510		515	505	
522	510		514	504	
522	509		508	503	
526	509		514	502	
528	513		513	505	
524	509		516	505	
522	507		513	504	
519	507		517	504	
518	507		516	505	
516	507		522	506	
519	508		520	510	
517	508		512	504	
515	509		508	503	
518	507		514	504	
522	507		517	502	
520	507		519	504	
519	508		518	505	
521	506		519	507	
523	507		517	509	
524	507		515	510	

TABLE XV

Heart rate during successive one minute periods of Session 2

Rat Number 2 E Conditioning Heart rate increases reinforced			Rat Number 3 E Conditioning Heart rate decreases reinforced		
521	493 0	499	550	.61 539 1	545
521	497 2	497	546	544 0	530
523	499 4	495	542	551 0	525
519	.66 500 1	491	542	543 1	531
521	496 0	496	538	.60 552 3	539
521	494 0	495	534	549 2	549
519	497 0	494	541	552 4	551
523	496 0	491	543	551 4	545
521	.68 496 3	493	542	554 0	539
521	496 3	495	542	554 0	540
517	496 3	496	561	552 2	533
515	495 2	494	565	556 1	537
517	496 3	495	559	554 1	537
515	492 0	496	558	548 3	529
513	494 0	493	554	547 3	533
513	497 4	491	548	.61 542 3	531
515	496 3	493	554	541 1	530
515	494 0	495	553	540 1	534
517	493 0	494	537	540 3	535
513	493 0	491	531	.62 539 2	536
513	.69 493 0		545	539 2	
510	494 1		551	539 2	
507	494 1		555	538 3	
510	496 2		529	538 3	
512	498 2		529	540 0	
508	501 5		538	542 0	
510	.68 501 4		521	539 2	
508	503 5		519	542 3	
512	499 0		525	539 2	
510	499 0		522	540 0	
.64 484 0	499		.68 517 0	552	
484 0	501		.67 521 0	559	
486 0	501		533 0	539	
.66 488 0	503		.64 553 0	542	
488 0	502		.63 544 0	546	
488 0	503		.62 551 1	551	
.69 494 4	503		547 1	559	
496 5	497		546 0	559	
.67 494 1	498		538 2	554	
494 1	496		541 1	554	

TABLE XVI

Heart rate during successive one minute periods of Session 3

Rat Number 2 E Conditioning Heart rate increases reinforced			Rat Number 3 E Conditioning Heart rate decreases reinforced		
514	548 0	545	522	516 3	514
507	.62 546 5	543	527	516 3	516
499	544 5	544	527	522 2	516
501	.61 546 0	535	520	522 0	513
497	548 0	539	510	.64 524 2	513
501	.62 544 2	541	509	527 2	513
501	547 4	541	513	525 4	512
519	549 5	540	517	.65 522 0	513
531	.61 549 0	534	515	522 1	514
531	551 0	527	512	519 3	514
517	.62 548 3	531	515	518 3	514
497	547 4	529	514	520 3	514
499	.61 558 6	531	517	520 1	515
516	558 6	527	514	.64 518 2	515
518	561 4	528	515	521 4	516
525	.60 556 0	526	509	519 5	516
528	.61 555 0	531	504	.65 522 0	516
524	554 4	536	506	.64 519 3	517
519	.60 557 1	531	510	518 6	517
511	.61 554 1	526	519	.65 518 1	515
500	.62 549 3	532	520	521 0	515
507	.61 555 5	526	520	518 2	516
512	553 1	526	514	514 4	517
525	554 1	525	516	.66 514 0	519
527	553 1	526	514	.65 515 2	521
539	552 0	525	521	513 3	521
539	555 4	527	525	.66 516 0	522
531	554 2	527	523	.65 514 3	521
531	554 2		523	513 4	
531	554 2		526	515 3	
.62 538 4	555 3		.70 522 0	515 3	
.61 550 3	553 1		.68 521 0	512 4	
551 1	555 3		.66 518 1	514 4	
548 0	551 0		519 0	512 4	
.62 549 4	551		.65 517 4	510	
.61 547 0	541		519 4	510	

TABLE XVII

Heart rate during successive one minute periods of Session 4

Rat Number 2 E Conditioning Heart rate increases reinforced			Rat Number 3 E Conditioning Heart rate decreases reinforced		
492	499 0	536	482	.77 453 3	434
491	500 0	538	478	453 3	438
488	.68 501 4	536	477	450 3	440
478	.67 503 5	534	476	452 2	443
484	.66 513 5	533	474	451 1	443
484	511 5	535	472	452 2	444
494	.65 512 0	532	473	450 2	442
489	.66 505 0	531	468	.75 450 0	440
482	.67 496 0	527	469	452 1	444
478	.68 503 5	526	468	451 1	443
472	.67 510 5	525	467	450 1	448
478	.66 514 3	521	467	451 1	449
482	516 3	519	465	449 3	449
481	.65 508 0	511	466	446 4	448
486	.66 508 1	511	464	448 1	443
488	.67 503 0	511	466	453 2	444
494	.68 497 0	507	465	450 3	446
494	492 2	507	465	448 2	445
495	.67 497 4	504	462	.76 446 3	446
494	508 3	504	460	447 3	446
499	.66 512 4	504	461	448 3	448
501	509 2	504	463	.77 442 1	448
497	508 0	506	460	441 2	446
498	508 2	509	459	.78 438 3	448
493	514 4	509	458	438 3	446
489	.65 514 2	507	460	436 5	448
486	520 4	509	457	436 5	452
482	.64 520 0	507	457	.79 430 1	454
484	523 0		456	428 2	
480	522 0		457	432 1	
.64 484 0	522 1		.80 454 0	430 3	
.68 494 4	526 5		.74 454 3	432 1	
.67 503 1	529 6		453 5	432 0	
504 4	532 6		.75 454 3	432 0	
.66 505 1	530		.76 453 3	431	
.67 505 1	529		452 5	433	

TABLE XVIII

Heart rate during successive one minute periods of Session 5

Rat Number 2 E Reversal Conditioning			Rat Number 3 E Reversal Conditioning		
464	468 0	473 1	491	460 3	445 1
456	468 0	473 1	489	.74 460 1	445 1
450	484 4	479 0	485	458 4	440 0
452	.71 485 6	485 0	486	455 6	438 0
453	488 6	479 0	484	460 2	441 0
454	486 4	474 2	488	.75 462 1	438 0
458	486 4	476 0	491	458 1	.78 438 1
459	.70 496 4	480 0	486	451 5	440 2
458	.69 491 1	479 0	487	443 5	440 2
458	478 0	480 0	487	.76 437 5	440 2
458	478 0	482 0	493	.80 425 2	438 0
457	484 1	476 1	487	429 2	438 0
457	466	478 0	484	431	440 2
458	464	484 0	489	442	439 1
458	459	484 0	492	437	439 1
461	454	482	484	433	441
464	474	488	480	431	445
459	469	484	474	435	449
454	471	489	468	436	449
452	471	489	464	435	456
454	468	493	455	437	459
454	469	488	456	437	456
454	H.R.Decr.	486	462	H.R.Incr.	460
451	Reinforced	488	466	Reinforced	451
453	.72 479 1	484	473	.68 436 0	456
455	473 0	481	468	436 0	458
454	479 0	480	465	.70 432 0	458
457	479 0	480	468	.71 434 0	455
459	486 0	481	464	.72 432 0	454
459	.71 479 0	481	466	.76 444 4	451
H.R. Incr.	478 1	485	H.R. Decr.	450 2	449
Reinforced	480 2	486	Reinforced	458 5	451
.60 462 0	480 2	483	.80 471 0	455 2	453
.62 464 0	478 4	478	.72 472 2	450 0	448
.63 464 0	478 4	478	475 3	442 0	444
.65 463 0	472 6		475 3	442 0	
.68 468 0	474 0		472 4	447 1	
.72 479 1	470 3		466 4	440 0	
479 2	474 2		.73 462 4	446 1	

TABLE XIX

Heart rate during successive one minute periods of Session 6

Rat Number 2 E Conditioning Heart rate decreases reinforced			Rat Number 3 E Conditioning Heart rate increases reinforced		
513	498 0	499	516	471 3	445
513	493 0	510	512	467 0	446
505	484 0	515	512	.72 464 2	443
506	480 1	525	510	465 5	445
503	482 0	526	509	468 6	446
501	479 2	516	509	.71 471 5	448
499	482 1	506	506	471 5	448
500	481 1	507	505	472 6	450
503	479 1	501	501	.70 474 0	449
496	478 2	487	500	474 0	449
499	476 2	474	504	.71 472 0	452
495	478 0	482	500	467 0	452
491	481 0	479	502	.72 458 0	447
486	479 1	486	498	.73 454 0	444
482	477 2	488	497	.74 453 0	436
483	480 1	485	494	.75 450 3	432
481	484 0	488	492	448 0	436
477	478 1	484	488	441 0	438
477	476 2	482	482	435 0	436
483	476 1	480	482	444 1	436
483	475 2	478	475	442 2	437
489	476 2	482	471	.74 450 5	441
487	474 3	484	470	.73 454 6	442
488	473 3	488	471	462 5	440
490	477 2	488	474	.72 462 2	435
490	472 3	488	474	466 3	431
488	475 2	489	472	462 1	436
486	472 4	490	474	460 1	430
486	474 1		471	458 0	
486	472 3		471	.73 462 6	
.72 495 0	476 3		.70 472 0	458 3	
.71 500 0	476 2		.71 472 4	460 5	
.70 487 1	472 3		472 1	457 3	
485 0	474 3		468 0	452 0	
484 1	476		471 3	450	
477 3	489		472 2	447	

TABLE XX

Heart rate during successive one minute periods of Session 7

Rat Number 2 E Conditioning Heart rate decreases reinforced			Rat Number 3 E Conditioning Heart rate increases reinforced		
472	475 0	486	524	467 1	479
478	475 0	484	524	.72 474 4	481
482	475 0	483	520	467 3	476
490	474 1	479	516	461 0	476
486	.69 474 2	478	515	454 0	479
481	474 2	478	514	463 4	478
480	476 0	479	512	474 6	478
478	476 0	478	511	.71 469 1	478
479	477 0	481	510	470 2	480
474	474 2	478	508	468 0	480
470	474 2	479	505	.72 458 0	478
474	475 2	478	504	.73 455 0	478
479	479 0	481	502	460 2	480
472	481 0	480	501	458 1	482
478	480 0	478	500	455 1	482
482	.68 479 1	476	496	460 2	480
483	479 1	476	499	462 5	480
482	478 2	476	497	461 4	481
485	480 0	477	495	464 6	480
479	.67 480 1	475	494	.72 468 3	478
477	478 3	477	492	467 3	484
474	478 3	474	490	466 3	482
467	481 0	474	490	470 6	483
464	481 0	471	487	.71 470 4	479
461	481 0	471	485	470 4	478
463	484 0	473	485	470 4	476
464	484 0	471	488	470 4	478
466	.66 483 1	473	484	470 4	476
466	482 1		486	470 4	
470	481 2		484	469 2	
.70 473 3	481 2		.70 484 5	469 2	
473 3	483 1		.69 483 0	468 1	
472 4	483 1		.70 480 2	468 1	
474 1	482 2		477	468 1	
474 1	484		.71 474 4	478	
474 1	485		472 2	482	

APPENDIX D

Tables XXI to XXIII showing heart rates for rat number 5 E during successive one minute periods of one adaptation session, three conditioning sessions, and two reversal conditioning sessions. Criterion changes and number of brain stimulation reinforcements during conditioning periods.

TABLE XXI

Rat Number 5 E. Heart rate during successive one minute periods of Sessions 1 and 2

Session 1			Session 2					
	Adaptation			Conditioning Heart rate increases reinforced				
484	457	461	459	.72	452	0	457
487	459	460	452	.74	452	0	457
483	460	460	448		454	2	457
475	458	458	450		448	0	457
471	454	458	450	.75	446	0	452
463	455	458	453	.76	443	0	450
465	452	457	453		447	5	452
463	451	457	449		443	3	454
466	453	451	449		447	4	457
466	456	453	454		444	4	456
466	452	455	458		445	4	454
465	450	456	463		447	6	456
464	449	458	468	.75	450	6	460
462	450	456	463	.74	456	6	464
459	452	456	467		441	3	459
457	454	452	467		438	2	460
452	457	452	464	.78	431	5	460
458	461	452	461		432	3	459
454	459	464	455	.77	440	6	461
458	456	467	449		437	3	459
458	456	464	447		439	5	464
462	456	466	444		446	6	467
464	456	470	441	.76	448	5	468
462	457	469	438		450	7	464
461	457	468	437	.75	455	7	466
461	460	465	438		452	6	466
464	461	464	439	.74	450	0	470
468	464	465	438	.75	447	2	473
466	462		438		447	4	
466	462		438		449	3	
441	462		.60	444	0		447	4	
438	461		.62	440	0		443	1	
444	464		.62	444	0	.76	441	1	
449	467			448			443	3	
455	464		.68	448	0		445		
457	461		.70	448	0		450		

TABLE XXII

Rat Number 5 E. Heart rate during successive one minute periods of Sessions 3 and 4

Session 3 Conditioning Heart rate increases reinforced			Session 4 Conditioning Heart rate increases reinforced		
488	429 3	453	497	444 6	490
489	430 6	458	489	.75 450 6	488
484	431 4	458	472	.74 447 3	490
480	.80 438 6	460	469	444 0	484
479	.78 440 6	463	451	455 5	484
479	.76 441 2	464	442	457 6	483
474	446 6	466	446	.73 456 2	487
469	.74 448 0	463	446	460 2	487
467	450 3	460	448	459 2	494
462	450 2	460	454	459 3	496
459	448 1	462	451	455 0	492
459	448 0	465	458	456 2	491
463	450 2	464	461	455 1	490
468	450 3	464	460	451 0	488
467	450 2	466	460	452 1	490
464	456 4	460	460	454 2	491
461	450 2	461	454	460 6	492
454	455 3	464	458	468 6	487
449	448 0	458	462	476 6	490
441	452 1	458	465	.72 476 6	486
453	453 3	454	461	476 6	487
451	453 3	457	455	.71 477 6	484
454	454 5	459	449	.70 482 7	484
444	454 4	460	453	481 6	481
445	452 3	458	457	.69 483 4	477
443	.73 454 0	453	458	484 4	475
441	457 1	458	453	487 6	477
433	454 0	450	455	486 5	479
442	454 0		453	.68 488 0	
446	.74 453 1		455	486 0	
.68 423 0	456 2		.78 437 4	.69 487 2	
.72 408 0	456 3		435 6	487 4	
.74 408 0	458 4		.77 444 6	.68 492 6	
.78 412 0	457 5		.76 443 6	491 4	
.80 416 0	454		440 5	486	
.81 426 3	456		439 4	489	

TABLE XXIII

Rat Number 5 E. Heart rate during successive one minute periods of Sessions 5 and 6

Session 5				Session 6			
Reversal	Conditioning	Heart rate	decreases reinforced	Reversal	Conditioning	Heart rate	decreases reinforced
	459	468 3	468		465	474 1	475
	459	469 1	469		462	474 1	475
	457	468 3	468		467	475 0	474
	457	470 2	468		465	475 0	474
	454	468 3	464		464	.69 474 2	470
	456	468 3	463		464	474 2	471
	451	471 0	465		465	470 4	473
	453	469 3	466		461	470 4	471
	454	466 3	465		460	476 0	469
	451	.74 462 2	463		462	476 0	468
	453	464 0	460		459	476 0	467
	454	464 0	459		459	472 1	468
	453	465 0	458		458	472 3	464
	454	465 0	456		457	474 2	464
	453	.72 466 0	455		457	.68 479 1	465
	454	466 0	457		458	479 1	464
	451	466 0	454		458	479 1	461
	454	468 0	453		458	476 3	460
	453	.70 471 0	451		456	476 3	459
	454	471 1	454		460	475 4	458
	451	472 1	451		454	478 1	458
	450	472 1	450		456	478 1	457
	449	469 3	451		457	476 3	458
	450	471 1	451		455	478 0	458
	450	.69 468 0	452		458	476 3	458
	450	468 0	451		456	481 0	458
	452	471 0	451		456	481 0	460
	448	467 1	450		456	481 0	456
	450	467 1			455	.67 478 3	
	450	471 0			457	478 3	
.70	475 0	466 3		.71	461 0	480 1	
	475 0	471 0			463 0	478 3	
	471 0	470 0			465 0	480 1	
	473 0	470 0		.70	473 3	478 3	
	471 0	470			473 3	476	
.71	468 4	469			472 4	476	

VITA

David Hothersall was born in Longridge, England on August 25, 1940. He received his early education at Bishop's Court in Freshfield, Lancashire. In 1952 he emigrated with his parents to Johannesburg in the Republic of South Africa and continued his schooling at Christian Brothers' College in Boksburg, matriculating in 1957. In 1959 he entered the University of the Witwatersrand in Johannesburg and graduated in 1961 with a Bachelor of Arts degree with a first class pass in Psychology. He was accepted into the Department of Psychology's honors program and in 1963 received an Honors degree with a first class pass in Psychology. In 1964 he was awarded a Master's degree with a distinction in Psychology.

A post-graduate scholarship awarded by the Council of the University of the Witwatersrand allowed him to return to England and to work with Dr. Jasper Brener in the laboratory of Dr. Harry Hurwitz at Birkbeck College in the University of London. Together with Doctors Brener and Hurwitz he emigrated to the United States of America in 1965 and entered the University of Tennessee as a graduate student in the Department of Psychology in September 1965. In 1966 he was awarded a pre-doctoral research fellowship by the National Institutes of Health.

David Hothersall is married and has one daughter born in Knoxville on September 4, 1967.