THE EFFECT OF EPINEPHRINE ON

AVOIDANCE BEHAVIOR

APPROVED:

[Signature]
Major Professor

[Signature]
Minor Professor

[Signature]
Dean of the School of Education

[Signature]
Dean of the Graduate School
THE EFFECT OF EPINEPHRINE ON
AVOIDANCE BEHAVIOR

THESIS

Presented to the Graduate Council of the
North Texas State University in Partial
Fulfillment of the Requirements

For the Degree of

MASTER OF SCIENCE

By

Paul Henry Beer, B. S.
Denton, Texas
January, 1966
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>LIST OF TABLES</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>LIST OF ILLUSTRATIONS</td>
<td></td>
</tr>
</tbody>
</table>

## Chapter

<table>
<thead>
<tr>
<th>Chapter</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>II. METHOD</td>
<td>10</td>
</tr>
<tr>
<td>III. RESULTS</td>
<td>15</td>
</tr>
<tr>
<td>IV. DISCUSSION</td>
<td>20</td>
</tr>
<tr>
<td>V. SUMMARY AND RECOMMENDATIONS</td>
<td>25</td>
</tr>
</tbody>
</table>

BIBLIOGRAPHY | 27 |
**LIST OF TABLES**

<table>
<thead>
<tr>
<th>Table</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. <strong>Summary of Analysis of Variance for the 3 X 3 Latin Square Design</strong></td>
<td>17</td>
</tr>
<tr>
<td>II. <strong>Summary of Analysis of Variance for the Three Treatment Conditions of Epinephrine, Normotensin, and Placebo.</strong></td>
<td>18</td>
</tr>
<tr>
<td>III. <strong>Means and Standard Deviations for Treatment Effects</strong></td>
<td>19</td>
</tr>
<tr>
<td>IV. <strong>t Values for Test of Significance of Treatment Groups.</strong></td>
<td>19</td>
</tr>
</tbody>
</table>
### List of Illustrations

<table>
<thead>
<tr>
<th>Figure</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Injection Ratio for Experimental Drugs and Placebo</td>
<td>12</td>
</tr>
<tr>
<td>2. Phase 1 Training Trials Without Shock With Mean Running Times Measured in Seconds</td>
<td>15</td>
</tr>
<tr>
<td>3. Phase 2 Training Trials With Mean Distance Measured in Centimeters</td>
<td>16</td>
</tr>
</tbody>
</table>
CHAPTER I

INTRODUCTION

The behavior of the human organism is made exceedingly complex by the interrelationship of psychological and physiological concomitants of emotion. For many years man has been interested in his emotions and those of his fellow man.

In 1919 John B. Watson stated that there were three "primary" emotions which could be observed in the human infant. These were fear, rage, and love or sex. He added that the other emotions were learned or acquired during the organism's development. However, in 1927 Sherman (8, 9) challenged this theory and noticed that Watson's observers could not discriminate among the supposedly separate emotions. He therefore concluded that Watson's description of "primary" emotions was biased and inaccurate.

Previously McDougall (7) had developed an idea which suggested an association between particular emotions and particular instinctive reactions. Thus, the emotion of fear was associated with the instinct of flight, and the emotion of anger or rage with the instinct of fighting or attack. McDougall used the term emotion to mean the tendency to feel, and an instinct was the tendency to act when in the presence
of a certain object in the environment. Other investigators noticed that the emotions also had their bodily "expression" which might involve strong muscular activity as in fear or anger. These findings suggested the importance of physiological correlates of emotions.

Prior to 1890 thoughts concerning the "coarser" emotions were that the mental perceptions of some event excited the mental affection called emotion; this latter state of mind gave rise to the bodily expression. In other words, emotions were present first and gave rise to physiological states. William James postulated a contrary theory which stated that "... the bodily changes follow directly the perception of the exciting fact, and that our feeling of the same changes as they occur is the emotion" (6, p. 242). He continued by stating that particular perceptions do produce widespread bodily effects by a sort of immediate physical influence which came before the arousal of the emotion or emotional idea. James not only suggested a mere relationship between emotions and physiological states, but he also implied that the physiological influence existed before the emotion and may lead to this emotion.

In later studies, using Watson's theory as a basis, investigators began to study the emotions from a physiological standpoint. Some of these studies were concerned with the effects which bodily secretions have on behavior. It was consequently discovered that some glands of the human
body do not deliver their secretion(s) to a specific area via a duct or tube. Instead, they are ductless glands which empty their products directly into the blood stream and are therefore called endocrine glands.

The adrenal is one such gland. It is composed of two parts, the cortex and the medulla. The major active secretion of the adrenal medulla is epinephrine, sometimes called adrenalin. This endocrine hormone was the first to be isolated in a crystalline form and was therefore the first to be used in studying its effects on emotions.

While investigating the physiological effects that epinephrine had on an organism, Cannon (1) described the emergency behavior of an animal in terms of physiology and emotions. He suggested that physiologically this behavior was a result of an oversecretion of epinephrine. In regard to emotions he showed that the organism was made ready to respond to the stressful situation by either an angry, aggressive "fighting" pattern of behavior, or by a "flight" response that was nonaggressive and fearful. This theory implied that the anger and fear of the "fight-flight" reaction are admixed, inseparable, and always associated with the secretion of epinephrine.

Wolff and his co-workers (10) noticed that in psychotic patients different physiological reaction patterns occurred in various organs with different emotional reactions. This was in disagreement with Cannon's theory which suggested
that anger and fear of the "fight-flight" response were inseparable.

Other investigators have also challenged Cannon's theory. Funkenstein et al. (3) cite work done by Ax and Greenblatt in which they studied the same subjects in an anger-inducing situation and in a fear-inducing situation. They found a different physiological pattern when the subjects reported their emotions as "anger" than when they reported it as "fear." In these experiments the anger was directed outward toward the experimenter. It is therefore suggested that Cannon's theory did not account for the fact that these two behavioral reactions, anger and fear, might have different physiological origins.

Carrying this conclusion one step further, Funkenstein (3) conducted a study using psychotic patients. He wanted to test the assumption that the physiological basis of both anger directed toward the self and fear would be similar. To do this he constructed a stressful situation and based his findings on the subjective feelings reported by each subject. His results confirmed the hypotheses that: (a) The changes in physiological patterns of his subjects who reported anger directed outward from the self differed from those who reported feelings of anger directed toward the self; (b) The physiological patterns of those subjects who reported anger directed outward away from the self differed from those who reported feelings classified as anxiety; (c) The changes in
the physiological patterns of those subjects who reported their feelings as anger directed toward the self were similar to those feelings reported as anxiety. The data suggested that the physiological basis or cause of "anger directed toward the self" and of fear or anxiety are similar. It was also implied from the data that "anger out" was associated with nor-epinephrine-like substances, and anxiety with epinephrine-like substances.

In postulating the "fight-flight" theory of behavior, Cannon felt that the physiological basis of fear and anger was conjunctive and inseparable and was caused by an excessive secretion of epinephrine. He could not always explain the changes of behavior on the basis of epinephrine alone, and further investigation revealed other substances which seemed to underlie these changes. These substances were called Sympathin E and Sympathin I. Funkenstein stated, "Recent physiological experiments have shown that Sympathin E and nor-epinephrine are probably the same substances" (4, p. 412).

Funkenstein (3) further cites other studies which indicate that the "fight-flight" reaction can be separated into components. He related that von Euler has evidence suggesting that aggressive animals have an excessive amount of nor-epinephrine in their adrenals, while animals which are easily frightened have an excessive amount of epinephrine.
In 1955 Funkenstein again tested the hypothesis that anger directed outward from the self is associated with the secretion of nor-epinephrine while anxiety or fear is associated with the secretion of epinephrine. He noted the reactions of subjects when they were injected with epinephrine and nor-epinephrine and quantitatively determined epinephrine-like and nor-epinephrine-like reactions. The mecholyl test was interpreted as an indicator of the nor-epinephrine/epinephrine ratio. The data confirmed the hypothesis.

An observation of Funkenstein and Meade (2) was that an elevated blood pressure was one of the responses to psychological stress. At least two different mechanisms were found to account for the rise. These two mechanisms were similar physiologically to those produced by the intravenous injection of epinephrine and nor-epinephrine. Another finding was that the blood pressure reaction following mecholyl seemed a reliable indicator of the type of blood pressure increase which was present in a given case. Further verification was sought in another series of experiments.

Additional studies yielded results which showed that students who responded to a stressful situation by anger directed outward away from the self had physiological reactions similar to those produced by an injection of nor-epinephrine. However, those who responded with depression
or anxiety had physiological reactions similar to those of epinephrine.

Ax later questioned if the physiology was specific for the emotion or the individual. In Funkenstein's summary (4) the results showed that the physiology was specific for the emotion and not for the individual. Ax also found that when the subject was angry at others, his behavior was similar to that seen after an injection of nor-epinephrine. But when the subject became frightened, it was more similar to that caused by an infusion of epinephrine.

Still other investigators have suggested that nor-epinephrine and epinephrine are secreted by different cells in the adrenal medulla, and when these cells are stimulated, they secrete their respective hormones. Some physiologists have also found that these different cells have neural representation in the hypothalamus. Hess et al. (5) found that by stimulating the different areas of the hypothalamus, aggressive behavior or nonaggressive behavior could be elicited in an animal. It was concluded that through a specific behavior such as fear or anger, different areas of the hypothalamus were stimulated. This action in turn stimulated the different cells of the adrenal medulla which resulted in the secretion of epinephrine and nor-epinephrine, respectively. From the experimental evidence cited above, it was assumed that epinephrine was a major physiological factor in the emotion fear.
It was the purpose of the present study to compare the effect of intraperitoneal injections of the following drugs on a conditioned approach-avoidance response in mice. These drugs were epinephrine and Normotensin, an epinephrine neutralizing hormone. Normotensin was used to see what effects the neutralization of epinephrine would have in the experimental situation. The avoidance conflict created in this study was used to measure fear or "anger directed toward the self." This measure was used on the assumption that a fear reaction to an aversive situation would lead to avoidance behavior. Funkenstein (3) has implied that "anger in" or fear is associated with epinephrine-like substances.

Having made the assumptions that epinephrine will elicit a fear response and that Normotensin will neutralize epinephrine, the following hypotheses were proposed:

A. Subjects receiving an injection of epinephrine will show a significantly greater fear response than those subjects receiving an injection of a placebo.

B. Subjects receiving an injection of epinephrine will show a significantly greater fear response than those subjects receiving an injection of Normotensin.
CHAPTER BIBLIOGRAPHY


CHAPTER II

METHOD

Subjects

A total of thirty male, naive, black mice from the colony C57BL/6J at the Jackson Memorial Laboratories were the subjects for this study. This strain was selected because of its sensitivity to behavioral studies. All animals were between thirty-five and fifty-five days old at the time of testing, and all subjects were maintained on ad libitum food and water.

Apparatus

A three compartment linear maze was used. The maze was a plywood box, 100 centimeters long, 6.3 centimeters wide, and 7.5 centimeters deep. It consisted of a 10 centimeter long start box at one end, and at the opposite end a goal box 10 centimeters long. A divided copper grid, 7.5 centimeters x 6.3 centimeters, through which shock was provided by an RCA forty-five volt dry cell battery, was placed directly in front of the entrance to the goal box. A hinged screenwire top covered the maze. The start box and the goal box were each separated from the runway by a guillotine door. These doors were operated manually from the outside. The
length of the maze from the start box door to the goal box door was calibrated and marked in centimeters, thus allowing for measurement of the distance which each subject ran.

Procedure

Preliminary training consisted of familiarizing all mice with the maze. Each subject was allowed to explore the maze for five minutes each day for two days.

There were two phases to the training. In the first phase all mice were trained to an approach response. The subjects were on a twenty-one hour food deprivation which served as the motivation for action. Each subject received a .4 gram food reward in the goal box. Each animal was given two trials per day for three days and one trial on the fourth day, a total of seven trials. This criterion was established in a pilot study using nine mice. Running speed for each subject was recorded after each trial.

In the second phase all mice were trained to shock avoidance. As the animal approached the goal, he passed across the divided copper grid and was shocked with forty-five volts. A water-soluble jelly with salt added was applied to the grid to increase electrical conductivity. Each subject was given one trial a day for five days, a total of five trials. This criterion was also established in the above mentioned pilot study.

After the approach-avoidance training was completed, each animal was randomly assigned to one of three groups of
ten subjects each. This was done to test the effects of the order of presentation.

Injections

All injections were made according to the ratio suggested by Barnes et al. (1), milligram per kilogram of body weight. Figure 1 graphically illustrates this ratio for both drugs and placebo.

![Figure 1](image.png)

Fig. 1—Injection ratio for experimental drugs and placebo.

The injections were done with a one cubic centimeter tuberculin syringe and needle. The drugs used were a 1:1000 solution of adrenalin chloride in mammalian Ringer's solution, Normotensin, and a placebo of mammalian Ringer's solution. All subjects served under all three experimental conditions of injection; these drugs were epinephrine (Parke-Davis Laboratories), Normotensin (Marcen Laboratories), and a placebo. All animals were injected intraperitoneally allowing
for a relatively slow onset of the drug effects (1).

A 3 × 3 Latin Square design was used to test for order effects (2), and permitted each subject to serve as his own control. The order of injection for Group 1 was as follows: epinephrine, Normotensin, placebo. The order for Group 2 was placebo, epinephrine, Normotensin; while the order for Group 3 was Normotensin, placebo, epinephrine.

The experimental sequences of time, temperature, and place were held constant, and every effort was made to keep the interexperimental intervals identical for all animals. A two day interval between injections was allowed to permit complete drug dissipation.

A standard procedure was followed for all injections (1). The experimental drugs and placebo were injected intraperitoneally over a period of thirty seconds. Both drugs and placebo were injected in the same volume relationship. The subject remained in the start box for five minutes after injection to allow for the drug to take effect.

In the test trials, after each subject was injected and had stayed in the start box for five minutes, the start box door was opened, and the animal ran the maze. The point at which the subject stopped was marked and this distance from the start box door was recorded in centimeters. The subject was taken from the maze at the point where he first stopped.
CHAPTER BIBLIOGRAPHY


In an attempt to measure the effects which certain drugs have on avoidance behavior, thirty male, black mice were administered epinephrine, Normotensin, and a placebo.

Figure 2 represents the mean running time of all mice for seven trials in the first phase. It can be seen that with each successive trial there is a decrease in the running time.
Figure 3 represents the mean distance run in centimeters by all mice for five trials in the second phase of training.

![Graph](image)

**Fig. 3**—Second phase training trials with distance measured in centimeters.

It is also shown in Figure 3 that there was a decrease in the distance run in the first four trials. However, there was an increase of 5.3 centimeters in the fifth trial, suggesting that this response was not yet stable.

Heterogeneity of variance was tested by an $F_{\text{max}}$ test and found significant. Since there was heterogeneity of variance, a logarithmic transformation was done to correct for this variance; the statistical analyses were done on the transformed data. Even after the transformation there was still heterogeneity, but the only significant difference was
between Group 1, non-injection, and Group 2, epinephrine. This result was due to the low variability in Group 1 since all of the mice were reaching the upper limit of measurement in the maze. Most of the subjects ran the full length of the maze. This low variability appeared to be a function of the length of the runway rather than any individual differences, and a longer runway would probably have allowed for greater variability.

Table I shows a 3 X 3 Latin Square design which was used to test for the effects of order of presentation. This analysis was done on the transformed data.

**TABLE I**

**SUMMARY OF ANALYSIS OF VARIANCE FOR THE 3 X 3 LATIN SQUARE DESIGN**

<table>
<thead>
<tr>
<th>Source of Variability</th>
<th>Sum SQ</th>
<th>df</th>
<th>Mean SQ</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups</td>
<td>.0690</td>
<td>2</td>
<td>.0345</td>
<td>NS</td>
</tr>
<tr>
<td>Order</td>
<td>.0312</td>
<td>2</td>
<td>.0156</td>
<td>NS</td>
</tr>
<tr>
<td>Drugs</td>
<td>4.8885</td>
<td>2</td>
<td>2.4442</td>
<td>34.0176</td>
</tr>
<tr>
<td>Residual</td>
<td>3.0215</td>
<td>2</td>
<td>1.5101</td>
<td>21.5113</td>
</tr>
<tr>
<td>Within</td>
<td>1.2642</td>
<td>18</td>
<td>.0702</td>
<td>NS</td>
</tr>
</tbody>
</table>

Since the order effect was not significant, treatment effects were analyzed by a single classification analysis of
variance with repeated measures. This analysis is shown in Table II.

### TABLE II

**SUMMARY OF ANALYSIS OF VARIANCE FOR THE THREE TREATMENT CONDITIONS OF EPINEPHRINE, NORMOTENSIN, AND PLACEBO**

<table>
<thead>
<tr>
<th>Source of Variability</th>
<th>Sum SQ</th>
<th>df</th>
<th>Mean SQ</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Subjects</td>
<td>.5776</td>
<td>29</td>
<td>.0199</td>
<td>NS</td>
</tr>
<tr>
<td>Within Subjects</td>
<td>22.6442</td>
<td>90</td>
<td>.2516</td>
<td>NS</td>
</tr>
<tr>
<td>Drugs</td>
<td>21.0216</td>
<td>3</td>
<td>7.0072</td>
<td>376.73</td>
</tr>
<tr>
<td>Residual</td>
<td>1.6225</td>
<td>87</td>
<td>.0182</td>
<td>NS</td>
</tr>
<tr>
<td>Total</td>
<td>23.2220</td>
<td>119</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

Because a significant F value was found, further analysis was done by multiple t tests to determine which treatment effects were significant. Table III contains the means and standard deviations which were used in the t tests.
TABLE III
MEANS AND STANDARD DEVIATIONS FOR TREATMENT EFFECTS

<table>
<thead>
<tr>
<th>Source of Variability</th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-injection</td>
<td>1.8907</td>
<td>.0616</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>.8133</td>
<td>.1959</td>
</tr>
<tr>
<td>Normotensin</td>
<td>1.5129</td>
<td>.1062</td>
</tr>
<tr>
<td>Placebo</td>
<td>1.7764</td>
<td>.1408</td>
</tr>
</tbody>
</table>

Table IV contains the t values which were obtained for Group 1, non-injection; Group 2, epinephrine; Group 3, Normotensin; Group 4, placebo. This data revealed that all groups were significantly different from each other at a level greater than .01; therefore, all the conditions appeared to have differential effects.

TABLE IV
t VALUES FOR TEST OF SIGNIFICANCE OF TREATMENT GROUPS

<table>
<thead>
<tr>
<th>Groups</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>...</td>
<td>27.79</td>
<td>17.77</td>
<td>4.26</td>
</tr>
<tr>
<td>2</td>
<td>...</td>
<td>...</td>
<td>18.70</td>
<td>19.81</td>
</tr>
<tr>
<td>3</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>8.21</td>
</tr>
<tr>
<td>4</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>
CHAPTER IV

DISCUSSION

The results in Table IV, as presented in Chapter III, showed that each group, non-injection, epinephrine, Normotensin, and placebo, was significantly different from the others at a level greater than .01. Consequently, the hypothesis that subjects receiving an injection of epinephrine will show a significantly greater fear response than those subjects receiving an injection of a placebo was confirmed. This observation is compatible with Funkenstein's theory (1) which contends that epinephrine is a major physiological cause for the emotion fear. Thus, this theory would propose that a subject receiving an injection of epinephrine would show a greater fear response in a stressful situation than would be shown without this injection. In the present study, subjects were trained to respond to an avoidance situation. Those subjects which had received an injection of epinephrine were significantly more fearful (or showed a greater avoidance response) than those subjects receiving an injection of a placebo.

The function of the placebo, as used here, was to control any confounding effects caused by the injection procedure.
The results are in agreement with studies done by Kosman and Gerard (2), Sines and Keefe (3), and Wurtman and his co-workers (4), who have reported that epinephrine suppresses the output of a learned response.

Funkenstein states that the emotions of fear and anger of the "fight-flight" response have been separated on both physiological and psychological levels by accounting for the direction of the emotion. As shown in Chapter I, epinephrine is proposed to underlie the emotion fear which Funkenstein has shown to be aggression directed toward the self. Therefore, the stress situation of the present study was constructed so as to allow each subject to show an inwardly aggressive response which was interpreted as fear.

Having made the assumption that a specific volume of Normotensin will neutralize the same volume of epinephrine, it was also hypothesized that those subjects receiving an injection of epinephrine should show a significantly greater fear response than those subjects receiving an injection of Normotensin. The results presented in Table IV of Chapter III confirm this hypothesis.

Another result observed from the data was that subjects receiving an injection of Normotensin showed a significantly greater fear response than those receiving the placebo. This result is contrary to the assumption that the mere act of injection caused the release of epinephrine and thus stimulated some amount of fear in the animal. Also, if Normotensin
does neutralize epinephrine, then the subject should have shown a greater fear response under the experimental condition of placebo injection. On the other hand, the injection of Normotensin appeared to have a paralyzing effect on the animals. Therefore, this injection may not have allowed the subject to function adequately. However, the exact pharmacological effect that Normotensin had on the experimental animals and the physiological site of action can only be inferred from the data, since there is no available quantified evidence concerning the drug.

Essentially, the preceding study attempted to experimentally validate the theory that epinephrine is the major physiological factor underlying the emotion fear. Nevertheless, there are certain methodological considerations which must be discussed in relation to this experiment.

The injections given in this study were intraperitoneal. This method of administration was chosen for three reasons, which are as follows: (1) the drugs administered were non-irritating; (2) the peritoneum of the abdominal cavity presented a large absorption area; and (3) the technique was simple and could be performed by one person.

One variable which may have affected the results was the "single shot" dose used. Since it was not possible to titrate the drug in this type of injection, an overdose could be administered to a sensitive animal. Most of the absorbed material entered the portal circulation where it
may have been partially metabolized within the liver. This metabolism could have caused a significant change in the structure of the drug and could possibly have changed its physiological action.

The experimental format required all injection to be made intraperitoneally, contrary to intramuscular route suggested for the administration of Normotensin. Therefore, the correct titer of this drug may not have been absorbed.

There was another variable which may have confounded the results. This condition was the volume of fluid injected; volume has been found to be especially important when dealing with small animals such as mice. The physical size of the experimental doses may have produced pressure effects which were manifested as physiological changes. This may be the reason for the previously mentioned paralyzing effect which Normotensin had on the subjects.

Based on this experimental evidence and the assumptions which were made, it was concluded that epinephrine was a major physiological factor underlying the emotion fear. However, this conclusion can only be made if the limitations of this study are considered.
CHAPTER BIBLIOGRAPHY


CHAPTER V

SUMMARY AND RECOMMENDATIONS

Summary

Thirty male, black mice were used in this study. All subjects were trained to shock avoidance and were injected with epinephrine, Normotensin, and placebo. This study was an attempt to test the theory that epinephrine is the cause of the emotion fear.

The hypothesis that subjects receiving an injection of epinephrine would show a significantly greater fear response than those subjects receiving an injection of a placebo was tested. A significant difference was found, and the hypothesis was accepted.

The hypothesis that subjects receiving an injection of epinephrine would show a significantly greater fear response than those subjects receiving an injection of Normotensin was tested. Again a significant difference was found to exist, and the hypothesis was accepted.

Statistical analysis of the data revealed that all groups (non-injection, epinephrine, Normotensin, and placebo) were significantly different from each other. It was also found that subjects receiving an injection of Normotensin
showed a greater fear response than those subjects receiving an injection of the placebo. This was contrary to what would have been predicted. No specific conclusions were made as to the cause of this behavior, except that Normotensin may have undergone some pharmacological change after injection as a result of the method of injection.

Recommendations

Based on the results and conclusion of this investigation, several additional related conditions require further experimentation and exploration.

1. Future investigations directly related to the present study should modify the maze by increasing the length of the runway. This modification should create a variability in the subjects' avoidance response.

2. Future research efforts should increase the number of avoidance trials to allow this response to become more stable. The number of experimental trials should also be increased from two to at least five trials, thereby providing a greater sample of the experimental behavior.

3. Exploratory investigations concerning the drug Normotensin should be conducted before its experimental influence and function can be determined.

4. In further drug experimentation concerning fear, the subject should serve only under one experimental condition of injection, thus eliminating the delay for drug dissipation.
BIBLIOGRAPHY

Books


Articles


