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An Investigation of the Extinction of the Freezing Component of the Cer After Frontal Ablation.

Roger Warren Mcintire
Louisiana State University and Agricultural & Mechanical College

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AN INVESTIGATION OF THE EXTINCTION OF THE FREEZING COMPONENT OF THE CER AFTER FRONTAL ABLATION

A Dissertation

Submitted to the Graduate Faculty of the Louisiana State University and Agricultural and Mechanical College in partial fulfillment of the requirements for the degree of Doctor of Philosophy

in

The Department of Psychology

by

Roger Warren McIntire
B. A., Northwestern University, 1958
M. A., Louisiana State University, 1960

August, 1962
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The author is forever indebted to his wife, Caroline, without whose time, effort, and loyalty this manuscript would never have been written.
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1. Schema of the Experimental Environment
2. Bar-pressing and Activity during CER Acquisition
3. The Brain Maps
4. Bar-pressing and Activity on Post-operative Days
5. The Relative Positions of the Lesions
ABSTRACT

A previous research paper has suggested that the instantaneous extinction of the freezing component of any conditioned emotional response (CER) following frontal ablation is due to incompatible hyperactivity resulting from the surgery.

This study was designed to test this hypothesis and one alternative to it which is that the frontal lesion has an effect specifically decremental to the freezing component.

During the acquisitioning of a bar-pressing habit, a conditioning paradigm (buzzer and shock) was introduced during training sessions. Thus, the subject exhibited both conditioned suppression of bar-pressing and the behavior syndrome of the CER.

Two different lesions were then performed; one was a bilateral lesion in the most anterior area of the frontal area, and the other was a lateral lesion slightly posterior to this.

The most significant findings were that: (1) only the response strength of the freezing component of the conditioned syndrome of behaviors was reduced by the operations, and (2) this effect was found only in the group with the laterally located lesion.
INTRODUCTION

Statement of the Problem

The purpose of this study is to present evidence concerning the effect of two types of frontal ablations on retention of previously acquisitioned instrumental and emotional responses in the white rat.

Recent Literature

Several studies of the last decade have attempted to determine the effect of frontal ablation on anxiety. In these studies, the measurement of anxiety was the strength of a response which in some way was anticipatory of a noxious stimulus such as freezing or defecating. This response was considered to be motivated by anxiety or fear. Some previous studies have suggested that any decrement in this response indicated a decrement in this motivation (Kahn, 1953; Lichenstein, 1950; Streb & Smith, 1955).

The assumption here that a decrement in the strength of one response has come about because of a general decrement in motivation and not because of a physical assault on a response mechanism has been supported in some additional recent studies where several response measures
showed a decrement. These responses were conditioned to stimuli paired with a noxious stimulus. Hunt (1956) demonstrated a combination of responses called the CER (conditioned emotional response) which included freezing and defecation as responses learned to a conditioned stimulus. Streb and Smith (1955) report that the frontal ablation resulted in a decrement in the CER and conclude that frontal ablation reduces a general motivation, anxiety.

Maher and McIntire (1960), in a partial replication of the Streb and Smith study, found that although frontal ablation diminished the freezing response, the defecation component of the CER remained unaffected. They point out that to continue to hold to the hypothesis that frontal ablation abolishes or reduces some single motivational state called anxiety is not possible in the light of their data unless one either postulates two or more motivational states or seeks some explanation of the specific decrement in freezing.

One of the alternatives to this latter possibility, which is presented by Maher and McIntire, is that a general increase in motility (hyperactivity) following frontal ablation is accompanied by a decrement in response strength of any discriminative response not compatible with hyperactivity and would, of course, include freezing. This effect has been demonstrated in the bar-pressing of monkeys (French, 1959), in a rat's failure to reduce speed at the
end of a runway (Epstein & Morgan, 1943), and in the frequency of errors in a discrimination problem with rats (Maher, 1955).

However, if the hyperactivity notion is to be adequately investigated, data concerning its relationship to the specific area damaged must be considered. Richter and Hawkes (1939), measuring activity in a revolving drum, found that activity increases in frontal animals. Beach (1941) found that only 75% of his frontal animals became hyperactive. The inconsistency of the effect lead Zubeck and DeLorenzo (1952) to hypothesize that the placement of the lesion within the frontal area was an effective variable. Three different lesions were performed in the Zubeck-DeLorenzo study: (1) frontal-lateral; (2) frontal-dorsal, and (3) all cortical area anterior to level four. Results were inconsistent within the experimental groups except in the group receiving the extensive frontal lesion. All of these animals were hyperactive post-operatively. Thus, there is the question: Damage to which area brings about the decrement in freezing? This is in addition to the question concerning the nature of the decrement.

Is the reduction in the freezing response of the CER after frontal ablation due to its incompatibility with hyperactivity or is it that the frontal ablation is doing some selective damage to the freezing response mechanism itself such as removing its motivation (anxiety)? In the
latter case, other behaviors ought to show no decrement. In the former, other behaviors ought to also be interfered with by hyperactivity. Thus the purpose of this proposed study can now be more specifically stated: to give evidence concerning: (1) the two possible explanations of the effect of frontal lesions on the freezing component of the CER, and (2) which areas within the frontal area are involved.

Rationale

The rationale involved in the following procedure section is that if the interference effect of hyperactivity is responsible for the decrement in freezing, then a decrement in other motor activities ought to occur as well, whether motivated by anxiety or not. On the other hand, if the frontal ablation is doing something selective to the freezing response, such as removing its particular motivation, then, with this removed, other learned motor activity ought to resume. These two explanations are operationally illustrated in Table 1.

Some other measurable response must be made available to S as well as the CER. The illustrated operations supply such a situation by presenting the CER training in an environment in which S may continuously press a bar for food (operations 1 and 2). Thus, when the conditioned stimulus comes on in the first phase of a CER-training trial, S freezes (CER) and stops pressing the bar (conditioned
### Operational Illustration of the Rationale

<table>
<thead>
<tr>
<th>Column 1</th>
<th>Column 2</th>
<th>Column 3</th>
</tr>
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<tbody>
<tr>
<td>Operation</td>
<td>Alternative One (interfering hyperactivity)</td>
<td>Alternative Two (selective damage to freezing)</td>
</tr>
<tr>
<td>1</td>
<td>Bar-pressing acquisition</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Conditioned suppression and CER acquisition</td>
<td></td>
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<tr>
<td>3</td>
<td>Ablation</td>
<td></td>
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<tr>
<td>4</td>
<td>Observe decrease in freezing during CS, but no resumption of bar-pressing</td>
<td>Observe decrease in freezing during CS, and resumption of bar-pressing</td>
</tr>
</tbody>
</table>
suppression). Now if the frontal ablation (operation 3) removes the anxiety for freezing, then the bar-pressing should resume (operation 4, col. 1 of Table 1). If, however, the produced general hyperactivity is interfering with freezing, then bar-pressing should not resume because hyperactivity should also interfere with it (operation 4, col. 2 of Table 1). Therefore these are the two alternatives for this study.

Alternative One

As a result of any frontal lesion, the freezing alone will be eliminated: bar-pressing can now return when the CS (buzzer) is presented.

Alternative Two

As a result of any frontal lesion, not only will freezing be eliminated, but generalized hyperactivity will occur: bar-pressing cannot now return when the CS (buzzer) is presented.
METHOD

Apparatus. A Model A-101 Skinner-box supplied by the Scientific Prototype Manufacturing Co. was used to carry out all training (see Figure 1). The bar was adjusted so that the minimal amount of vertical pressure necessary to activate the micro-switch attached was 25 gm. A brass food cup was situated to the left of the bar in the corner. A tube connected the cup to an electrically operated feeder outside the Skinner-box.

The walls to either side were made of plexiglass. Mounted outside the wall to the right of the bar were two shielded, photo-sensitive resistors, 1 in. from each end and 1 in. above the floor. Directly opposite these resistors and just outside the other wall were two light sources focussed on the resistors. The entire apparatus thus far described was placed in a soundproof box which contained a buzzer. When activated, the buzzer raised the noise level 1 db. inside. The door to the soundproof box contained a one-way mirror which was 2 in. from the left plexiglass wall when the apparatus was closed. An electric fan mounted over a hole in the top provided a masking white noise.

The photocells activated a counter giving a measure of the activity of S by recording the number of times S broke
FIG. 1 SCHEMA OF EXPERIMENTAL ENVIRONMENT
the light beams in the Skinner-box. All recording and programing equipment was outside the soundproof box.

The recording of bar-presses and presentation of rewards was controlled by a Foringer programer which could be wired for any ratio schedule. Also a Foringer shock scrambler was used when shock was delivered to the grid floor of the Skinner-box.

A four channel event recorder was employed to record the following two dependent and two independent variables simultaneously as a function of time: (1) bar-presses; (2) activity (the two dependent variables), and (3) buzzer presentations, and (4) shock (the two independent variables.)

Controlling the recorder, buzzer, and shock was a programer situated in a sound deadening box in an adjacent room. This equipment consisted of a time switch, and several delay relays constructed in order that E could set the program so that at any predetermined time the buzzer would sound and 60 sec. later the buzzer would go off and, instantaneously, shock would come on for 2 sec.

Subjects. Twelve 110-day-old male albino rats of the Sprague-Dawley strain were used and weighed from 350 to 365 gm. at the beginning of the experiment.

Response Measures. In addition to bar-pressing and activity, three other measures were recorded on each S each
day during the study: (1) weight; (2) water consumption; (3) defecation. Each S was weighed just before the day's training session. His weight was recorded and he was fed just after each day's training session, either 8, 9, or 10 gm. of ground chow, depending on whether his weight was under, equal to, or over his 80% body weight. The amount of water S had consumed in the 24 hrs. previous to the day's training session was noted by weighing his water bottle.

Two measures of defecation were kept: (1) in the home cage, and (2) during daily training, before, during, and after the buzzer presentation if it was to be presented that day. S's activity during the training session was measured each day as described in the preceding apparatus section. The number of bar-presses was noted after each day's training session. Both activity and bar-pressing were recorded as a function of time, before, during, and after the conditioned suppression training trial.

Procedure. The first 12 days of procedure consisted of bar-pressing acquisition (operation 1, Table 1). On Day 1, after having been reduced to 80% body weight over a seven-day period, and after 23 hrs. of deprivation, S was placed in the apparatus. He was allowed to explore the apparatus for 20 min. during which time E activated the feeder just as S approached the food cup. When, in E's judgment, this stimulus quickened S's approach, the feeder was activated
when $S$ was not approaching the food cup. This procedure continued through the 20 min. period, resulting in an immediate investigation of the food cup by $S$ every time $E$ activated the feeder.

On Day 2, the procedure was the same as on Day 1, except that $E$ now activated the feeder only when $S$ to some degree approximated the response of pressing the bar. The criterion of degree of approximation was continually raised until $S$ actually pressed the bar, thereby providing his own reward on an FR-1 schedule. On Day 3, $S$s were raised to a VR-5 schedule at the end of the first 5 min. of the 20 min. session. On Day 4, $S$s were raised to a VR-15 schedule at the end of the first 5 min. of the 20 min. session. On Days 5-12, 30 min. sessions were given while $S$s continued acquisition of the VR-15 schedule in order that $S$ would not satiate during the longer session as his acquisition continued.

On Day 13, the acquisition of conditioned suppression and CER behaviors was begun (operation 2, Table 1). At some time during the bar-pressing session between min. 5-25, according to a table of random numbers, the programer was activated which presented the buzzer, but on this day no shock was presented.

On Days 14-24, the procedure was the same as on Day 13 except that a 20 ma. shock was presented for the 2 sec. following the buzzer. On Days 25-26, the shock was in-
creased to 35 ma.

Following the last day's training session Ss were matched into three groups on bar-pressing and activity during the presentation of the buzzer on the preceding 3 days. The groups were: (1) Group A (frontal), Ss ablated in the most anterior area of the frontal lobes; (2) Group L (lateral-frontal), Ss ablated in the most lateral area of the frontal lobes, and (3) Group S (sham), Ss on which all operative procedure was performed except the skull was not entered.

The ablations (operation 3, Table 1) were performed using a compound general anesthetic which was administered to each S nine hrs. after his training session on Day 28. While S was held in an especially fitted oral-nasal clamp, an incision was made along the mid-line of the scalp from a level just posterior to the orbit of the eye to the posterior level of the auditory meatus. After clearing away underlying tissue, the skull was entered by drilling a hole in each of the frontal bones. The meninges were cut and cortical tissue removed with a small canula (1/32 in. diameter at the tip) connected to a mechanical aspirator which developed 22 lbs. vacuum pressure.

The skull was closed with a plaster of Paris-alcohol mixture and the scalp sutured. The operation procedure took approximately 20 min. and S showed no anesthetic effects by the end of five hrs.
On Days 29-38, the training session procedure was exactly as on Day 13, that is, the buzzer was presented but no shock. On Days 39-41, extinction of bar-pressing began by the elimination of pellets from the feeder.
RESULTS

CER and Conditioned Suppression Measures. Presented in Figure 2 are the mean bar-presses and the mean number of interruptions of the photocells (activity) for the min. before, during, and after the buzzer presentation. These data are grouped for four-day periods during the CER acquisition phases.

The curve representing bar-pressing for Days 1-4 of the CER acquisition shows the unconditioned response of suppression of bar-pressing to the shock (post-CS interval). The curve representing Days 5-8 shows this effect as even more pronounced with the conditioned suppression (during the CS-US interval) equal to it. As the acquisition of the conditioned suppression continues, the unconditioned cessation of bar-pressing to the shock is insignificantly different in amplitude from the initial level on Days 1-4.

In contrast, the activity data during the CER acquisition phase (right-hand graph in Figure 2) shows an unconditioned response is a burst of activity in the post-CS interval which showed no change throughout this acquisition phase. The simple analysis of variance showed a significant drop in activity across days during the CS-US interval (p < .01) and demonstrates the acquisition of the freezing
FIG. 2  BAR PRESSING AND ACTIVITY DURING ACQUISITION

MEAN BAR PRESSES

MEAN PHOTOCCELL INTERRUPTIONS
response during this period.

There are two distinct differences to be noted between the bar-pressing and activity in these data. First, even in this grouped data, acquisition of the conditioned suppression of bar-pressing is virtually complete by Days 5-8, while the effect of the CER acquisition phase is not noted in activity until Days 9-12. Secondly, the acquisition of freezing is not classical conditioning since the unconditioned response to the shock is a burst of activity not freezing. Freezing "comes in" much like an instrumental response.

Since this cessation of activity certainly cannot be classified as instrumental, then it seems that the most plausible explanation is that it is an unconditioned response to fear that had become conditioned, classically, to the buzzer. It did not appear to the buzzer early in the acquisition series data because it was naturally incompatible with the burst of activity directly elicited by the shock. Thus, a syndrome of behaviors were acquisitioned to the CS. The first of these was conditioned suppression of bar-pressing, followed later, in acquisition, by freezing.

Figure 3 shows the lesions made in the two experimental groups. Group A lesions, presented in the left-hand column, show one irregularity, and that is represented in the third brain map in that column. This lesion does invade lateral areas as well as the intended anterior ones, but these in-
FIG. 3 THE BRAIN MAPS
vaded lateral areas are anterior to level three while the damaged lateral area in Group L is, in every case, posterior to this level. In spite of this deviation, the statement is true that in all Ss in Group A the anterior-frontal area was ablated and no area beyond level three was damaged while in all Group L Ss the anterior-frontal area was left undamaged and a partial ablation of lateral tissue just posterior to level three was performed.

Figure 4 presents, by days, the activity and bar-pressing behavior for the buzzer presentation interval during post-operative days. These data are presented for each experimental group. Group S, the sham-operated group, is not represented because it was insignificantly different from Group A which, therefore, is the better control condition.

On the first and second post-operative days, cessation of bar-pressing was practically complete during the buzzer and no differences between groups are significant. The activity for these data also present no significant differences between groups. However, on the third and fourth post-operative days, the activity of Group L (Ss with a laterally located lesion) showed a significant increase during the buzzer as indicated by the significant main effect of the analysis of variance presented in Table 2. The freezing component of the CER was completely obliterated without any corresponding increase in bar-pressing for this
FIG. 4 BAR-PRESSING AND ACTIVITY ON POST-OPERATIVE DAYS
period. This phenomenon occurred from the third post-operative day through the eighth.

On the ninth and tenth post-operative days, the significant differences between the two experimental groups on activity disappeared as indicated by the t's presented in Table 2. Presumably this was due to classical extinction in Group A since no shock was presented post-operatively.

**Total Bar-pressing.** During acquisition, total number of bar-presses reached approximately 2,000. The post-operative data show a significant recovery from the operation with Group S (the sham-operated animals) consistently higher, but statistically this is not significant. Extinction data showed no differences between experimental groups.

**Total Activity.** Activity decreased significantly post-operatively, but the experimental group differences do not reach significance except total activity reached pre-operation levels by the second post-operative day.
TABLE 2
Analysis of Variance of the Post-operative Activity Data

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>P</th>
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<tbody>
<tr>
<td><strong>Between Ss</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Experimental Groups (E. G.)</td>
<td>1</td>
<td>3315.3</td>
<td>53.2</td>
<td>&lt;.01</td>
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<tr>
<td>Error</td>
<td>6</td>
<td>62.3</td>
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<tr>
<td><strong>Within Ss</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Days (D.)</td>
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<td>268.0</td>
<td>2.19</td>
<td></td>
</tr>
<tr>
<td>E. G. X D.</td>
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<td>270.8</td>
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<tr>
<td>Error</td>
<td>54</td>
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<tr>
<td><strong>Total</strong></td>
<td>79</td>
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**Between Groups t's for Individual Days**

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<th>df</th>
<th>t</th>
<th>P</th>
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<tr>
<td>8</td>
<td>6</td>
<td>3.22</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>9</td>
<td>6</td>
<td>.61</td>
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</tr>
<tr>
<td>10</td>
<td>6</td>
<td>.25</td>
<td></td>
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</table>
DISCUSSION

The Loss of Freezing in Group L. Alternative one of the introduction (the specific-effect alternative) cannot claim much support from Group L since these Ss did not return to bar-pressing when freezing disappeared. The lateral lesion, although it was the effective one, left the conditioned suppression unharmed.

Upon first investigation of the data pertaining to the performance of the CER after the operations, it would seem that alternative two of the introduction (hyperactivity interference) was supported. The freezing component of the CER had disappeared, yet bar-pressing had not resumed. One might therefore argue in favor of the "interference alternative" of hyperactivity. Yet bar-pressing outside of the CS-presentation interval was unaffected and no hyperactivity was noted at that time (outside of the CS-presentation interval). If the interference of hyperactivity were to be generally true, then bar-pressing, inside or outside the CS-presentation interval, should have shown a decrement. However, it was only during the CS that activity had increased.

The situation, in brief, is that the lateral-frontal Ss showed renewed activity, but only in the CS interval and no decrement in bar-pressing or increase in activity during
other periods. So it seems that alternative one, referring to a selective effect of the lesion, is inadequate because the affected Ss did not return to bar-pressing during the CS, and alternative two, referring to the interference of hyperactivity, is inadequate because Ss did not show a decrement in bar-pressing outside the CS interval.

A third alternative suggests itself when one considers the manner in which the CER was learned. The acquisition data on the CER indicate that it is the conditioned suppression of bar-pressing that is the most sensitive and the earliest indicator of acquisition during the buzzer-shock series. The third alternative might be that the lateral lesion had a partial decremental effect on the learned behavior to shock and brought about activity where in the later stages of acquisition there was freezing, the last response to be learned to the shock. But this effect was not to the extent of losing the conditioned suppression, the response learned fastest, and therefore the more overlearned response. In other words, the selectivity may be a function of the degree of learning rather than the ablation directly.

The Particular Locus of the Lesion. Since only the lateral lesion produced a loss (of both freezing and bar-pressing to the CER) and no such loss is noted with the anterior lesion, a re-examination of the Maher and McIntire paper seems in order. In that study a very extensive lesion was performed, the extent of which is shown in Figure 5.
FIG. 5 THE RELATIVE POSITIONS OF LESIONS
This lesion invades the locations of both of the lesions done in the present study. It is a frontal lesion in the broad sense, that is, it is in the front part of the brain, but further data from Woolsey and LeMessurier, adapted from Zubeck (1951) (see Figure 5), indicate that motor area I may be included in this extensive lesion, and also included in, what is called here, the lateral lesion. This area is shown in Figure 5 and leaves only a small area in Figure 5 as the true prefrontal area. Although the Maher and McIntire lesion includes this prefrontal area, it also includes part of motor area I. It is in this included area of motor area I that the present study localizes the lesion producing the loss of the freezing component of the CER.

Although it is not hard to see why Maher and McIntire observed this loss, also having included this area in their ablation, the reason as to why damage in this particular motor area should cause a partial decremental effect in freezing is not understood.

Since these areas are mapped on the basis of their capability to initiate a motor response under direct stimulation, paralysis would seem to be a plausible prediction. No paralysis was noted in either the Maher and McIntire study or the present one. This observation is further verified by the fact that no decrement was evident in bar-pressing, a rather complicated motor response, in the present study except during CS presentation. The demonstrated phe-
nomenon is an increase in motor activity, not something easily associated with paralysis.

One explanation as to why this third alternative seems to hold might be that a more subtle type of control has been damaged, not the control over the initiation of the response such as paralysis, but the control over response amplitude. Thus, when an S with this particular lesion attempted to freeze, the responses which were, before, small movements of the head, etc. (therefore unrecorded as breakings of the beams), overshot their intended amplitude, resulting in locomotor activity. When the buzzer was off and bar-pressing activity was initiated, only a measure of pressure exerted on the bar would have shown any post-operative change.

In a pilot study for this paper, a significant decrement in bar-pressing was noted in the group receiving an extensive lesion within motor area I. At that time E made several notations that S did not seem to have the proper proprioceptive feedback. That is, S responded in a way which resembled bar-pressing, but these responses were of an ineffective amplitude, such as "pawing at the bar," "holding down the bar for an exaggerated length of time." Possibly with larger lateral lesions a similar phenomenon would have been noted in the major study.

The introduction of this paper begins by citing studies which have attempted to determine the effect of frontal ablation on anxiety. Most of these studies use
activity in some way as a response measure and report hyperactivity post-operatively which has reduced freezing. Perhaps due to the close spatial relationship of motor area I and the prefrontal area, most of this literature actually reports a phenomenon which is brought about by a motor lesion, and has nothing to do with a more generalized effect, justifying the term anxiety. It may simply be over-responding.

Of course, no study, including the present one, makes a direct test of this hypothesis. It is a post hoc explanation. However, several experimental designs come to mind: (1) a study employing bar-pressing as the acquisitioned response followed by ablation of premotor area I and a test using a pressure exerted on the bar as a measure of over-responding; (2) a study similar to number one in which only a certain pressure on the bar is effective, no more or no less (this alternative would predict a great decrement in response rate); (3) studies similar to numbers one and two, manipulating placement of the lesion within motor area I.

Of course, many previous studies need repeating, using only the motor area I lesion to see if the various effects demonstrated could be replicated with this particular lesion.

There seems to be little doubt that whatever the function of the frontal area, it is closely connected with the motor activities of the cortex. Its very location in relation to known motor areas makes it suspect. Whether
any study has been able to separate its activities from other motor activities remains to be seen, but these points make the frontal area as participant in intricate motor activity an unavoidable consideration.
SUMMARY

An animal study has been conducted which was designed to determine further the nature of the extinction of the freezing component of the conditioned emotional response following frontal ablation.

Two alternative outcomes were considered possible in this experiment. One was that a frontal lesion would only eliminate the conditioned freezing component and nothing else. The other alternative was that the extinction of the freezing component was the result of incompatible generalized hyperactivity brought about by the frontal lesion.

The former of these alternatives implies that once the operation is performed, the animal would discontinue freezing and resume the behavior most probable, whereas the second alternative would assume that hyperactivity would compete with any other discrimination behavior as well as freezing. Therefore, the study was designed using a Skinner-box in which a bar-pressing habit was trained and a conditioning paradigm (buzzer and shock) was introduced during the bar-pressing session in later acquisition stages. Thus, S exhibited conditioned suppression of bar-pressing in addition to freezing.

Two different lesions were performed; one was a bi-
lateral lesion in the most anterior region of the frontal area, and the other was a lateral lesion slightly posterior to this location. A group of sham-operated animals was also included.

Among the most significant findings was that only the freezing component of the syndrome of behaviors conditioned to the buzzer was extinguished by the operations (conditioned suppression of bar-pressing was unaffected).

This first finding was viewed as only partial support for the alternative that it is incompatible hyperactivity rather than a specific effect which accounts for the decrement in freezing. Some reservations were, however, expressed: (1) hyperactivity was not demonstrated in the overall activity measure, and (2) bar-pressing in other periods (i.e., when the CS was not present) showed no decrement.

With the additional finding that the loss of the freezing component was observed only in the lateral lesioned group, the following explanation was offered.

The discussion pointed out that the locus of the effective lesion is a part of motor area I, and further, that this area was included in many lesions reported in papers demonstrating hyperactivity. It is therefore suggested that the control that this area has over motor behavior was concerned with amplitude of "out-going" responses. When this area is damaged, the animal over-responds and the resulting behavior has come to be called hyperactivity. If this is
the case, then an animal may "unfreeze" after frontal ablation simply because the small amplitude responses which the animal made while freezing before ablation are now large amplitude responses. Conditioned suppression of bar-pressing was not affected because this over-responding would not be directed toward bar-pressing any more than the pre-operative small amplitude responses mentioned above. Furthermore, conditioned suppression of bar-pressing was the more overlearned response, and therefore possibly more resistant. Thus, bar-pressing outside of the conditioning session would be unaffected because bar-pressing is not as dependent upon response amplitude as is general activity. Since bar-pressing did continue, incompatible generalized activity measures showed no increase.

Concluding remarks of the discussion suggest experimental designs which might bring forth further evidence concerning the role of motor area I and the prefrontal area in response amplitude control.
BIBLIOGRAPHY


VITA

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Candidate: Roger Warren McIntire

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Approved:

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Major Professor and Chairman

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EXAMINING COMMITTEE:

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