THE EFFECTS OF FRONTAL LESIONS ON LEARNING AND RETENTION OF AN AVOIDANCE RESPONSE IN THE DOUBLE-GRILL SHUTTLE BOX

by

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INTRODUCTION

In animals, the usual response to strong fear-producing stimuli is either extreme immobility or escape behavior. It has recently been suggested that two mutually opposing anatomical systems operate during the performance of these responses (Vanderwolf, 1962). One of these systems leads to the inhibition of motor activity and evokes a "freezing" or "crouching" response. The other system leads to the initiation of motor activity, usually manifested by running or jumping. Presumably, the inhibitory system is dominant over the excitatory system during freezing behavior as seen in a conditioned emotional response (CER) to a buzzer which signals oncoming shock. The converse would hold true during the performance of a conditioned avoidance response (CAR) such as running from one end of a box to the other in order to avoid shock.

Both behavioral and electrophysiological work have suggested that the inhibitory system involves an efferent pathway which originates in the frontal cortex (Kaada, 1951; McCleary, 1961). Traditional studies on the effects of frontal lesions upon positively reinforced learning appear to support the notion that the frontal cortex is involved in inhibitory processes. These studies are summarized below. In learning situations evoking fear, however, the role of the frontal cortex is more uncertain.

Although one study, using the CER, suggests an inhibitory role (Maher and McIntire, 1960), another study using the CAR suggests that the frontal cortex may also have an excitatory function.

The purpose of the present paper is to offer evidence showing that these two experiments are not incompatible when the frontal cortex is considered as having both inhibitory as well as excitatory influences depending upon the nature of the learned response involved. This hypothesis will be made more explicit following a review of the relevant literature.

Some Effects of Frontal Lesions in Positively Reinforced Learning Situations

It appears that traditional studies on the effects of frontal lesions in positively reinforced learning situations support the notion that the frontal cortex is involved in inhibitory processes. The effects of frontal lesions have been manifested in the form of perseverative behavior, failure to extinguish, impairment of habit reversal and the inability to habituate. Each of these is discussed below.

Perseveration is a phenomenon which indicates an inability to change a previously performed response in light of new sensory and motor information. Such a process of change would be disturbed, according to the inhibition hypothesis (above), as a result of lesions in

the frontal cortex. There are several studies that are consistent with this point of view. Maher (1955) designed an apparatus in which a rat could make errors not only of the perseverative type, but also other types as well, including "anticipatory" errors. The animals had to choose one of four differently marked doors. The correct doors for the first three compartments had a striped pattern while the last correct door was marked with a disc. If the animal chose the disc before he got to the last choice point, the error was "anticipatory"; conversely, if he chose a striped door at the last choice point, the error was "perseverative." Results showed that frontal animals exhibited a significantly greater number of perseverative errors than did controls. They were not different from controls in other errors.

Perseverative behavior has also been demonstrated in the form of resistance to extinction of bar-pressing in a Skinner box. Mello (1961) trained 63 rats either to a schedule of continuous reinforcement or to a fixed reinforcement ratio of 6:1. The animals were trained to a criterion of 600 reinforcements over a period of six days after which animals in the experimental group were given frontal lesions. Following a 48-hour recovery period, all animals were given two 30-minute reinforcement sessions followed by two 30-minute extinction sessions and then a series of alternate reinforcement and extinction sessions over a period of ten days. Results showed:

(1) no difference between operated and normal rats during the postoperative "retention" testing for either reinforcement schedule; and (2) during the first two extinction trials a marked decrease in number of responses for all groups; however, during sessions three, four, and five, the fixed-ratio frontal rats increased their number of responses over time--persisting in a stereotyped manner. All other groups dropped down to a significantly lower level of responding. The author concluded that the fixed-ratio frontal rats persisted in a perseverative response without reinforcement either because they failed to distinguish the extinction situation from the reinforcement situation or because of a lowered ability to adapt to a different environmental situation (Mello, 1961). Such a capacity would, of course, require the perception and integration of sensory and motor cues prerequisite to the inhibition of an old and initiation of a new series of responses.

The process of perseveration would presumably interfere with the suppression or modification of a preestablished set. Bourke (1954) found that frontal lesions ranging from one to six per cent of the cortex had no significant effect on learning a simple Y-maze problem. However, when a habit reversal task was given by preventing the animals from entering the previously reinforced alley, frontal animals needed significantly more trials to learn the new habit. This difference

was increased further when the task was made more complex by adding on more maze units.

However, in the same study Bourke found that frontal rats, when tested on their ability to learn brightness discrimination reversals, did not perform significantly differently from normal rats. Both frontals and normals showed evidence of "learning-to-learn" discrimination reversal tasks. One explanation offered by Bourke as to why frontals were inferior in maze reversal problems but not in brightness discrimination reversals was the possibility that damage of motor areas from frontal lesions may have destroyed the ability to utilize motor cues important for solving the maze problems. The discrimination problem evidently did not involve as much dependence upon motor cues.

Incidently, there is also evidence to suggest that habit reversal ability is dependent not only upon the kind of motor cues that must be perceived in order to perform the task but also upon the nature of the sensory cues involved. Thompson (1963) found that when rats were tested for retention of a habit reversal problem using negative reinforcement as punishment for entering the wrong arm of a T-maze, there was no significant difference between the performance of frontal and normal rats. An explanation of the discrepancy between these results and those obtained by Bourke (positive reinforcement in a Y-maze), according to Thompson, is that one of

the functions of the frontal cortex may be to amplify information lacking "vividness" (a function which presumably would not be necessary using extremely intense stimuli such as shock as reinforcement). If this were the case, then the inhibitory functions of the cortex would be required only in those instances where discrimination and integration of motor and sensory cues were more complex for the rat.

These results illustrate the obvious point that not all inhibitory processes require that the frontal cortex be intact. It seems that the inhibitory functions of the frontal area are needed only for particular tasks depending, perhaps, upon the kind and/or peculiar combination of motor and sensory cues that must be perceived and acted upon during performance of the task.

So far, the learned responses presumably inhibited by the frontal cortex have been of a more or less voluntary nature. However, evidence has also been found for the inhibitory influence of the frontal area upon an autonomic response, namely on habituation of the heart rate in the rat. Habituation is a learning process similar to extinction (cf., Mello, 1961) in that they both require the suppression of a response. Extinction results in suppression of a learned response while habituation usually requires suppression of a natural or unlearned response. Glaser and Griffin (1962) studied the effects of frontal lesions on the habituation of the

heart rate response to a cold stimulus applied to rats' tails. Results showed that in normal, unilateral frontal, and occipito-parietal rats, increased heart rate due to the stimulus became smaller and smaller over a period of ten days, while in bilateral frontal rats no habituation whatsoever occurred. However, bilateral frontal lesions failed to disrupt an established habituation indicating, perhaps, that the frontal areas are involved in the formulation but not the performance of habituation.

Some Effects of Lesions in the Frontal Cortex and Related Subcortical Structures in Negatively Reinforced Learning Situations

Inhibitory processes in general seem most apparent in negatively reinforced learning situations. These situations often require behavior exemplified by either the conditioned emotional response (CER) or the conditioned avoidance response (CAR). The CER consists of crouching or "freezing" and is usually accompanied by vocalization and defecation. This response may either be made in response to or in anticipation of a noxious stimulus. The CAR induces the opposite of crouching, i.e. the initiation of avoidance activity, and may be made both in response to ("escape response") or in anticipation of ("avoidance response") a noxious stimulus. Thus, an important characteristic of fear is that it can initiate contradictory

responses (e.g., freezing vs. running) one of which usually dominates over the other depending upon the situation. A possible subcortical basis for this dual mechanism (and one which implicates the frontal area) can be derived from many different studies. Vanderwolf (1962) found extensive medial thalamic destruction to destroy completely the ability of both naive and pre-operatively trained rats to make effective avoidance responses. This deficit could not be attributed to a loss of fear, as measured by the amount of defecation and urination, nor could it be attributed to a motor disability, since the thalamic animals could escape from the shock just as well as the normals. Furthermore, it turned out that the medial thalamic lesions interfered only with the animal's ability to initiate appropriate movement in response to negative (fear producing) stimuli--i.e. shock; when positive reinforcers were used such as food, water, or opportunity for exploration, the thalamic rats showed little impairment in the initiation of voluntary movement and were even more active than normal rats (Vanderwolf, 1962). Thus, the medial thalamus seems to play a role in initiation activity (e.g., the CAR) in a fear situation.

Another structure, the cingulate cortex, seems to exert an excitatory influence on behavior. Rats with anterior (Peretz, 1960) and posterior (Thomas and Slotnick,

1962) cingulate lesions were both inferior to normal rats in ability to learn a CAR. The effects could not be attributed to a general intellectual impairment since the lesioned rats were not inferior to normals in the acquisition of tasks employing positive reinforcement such as maze learning. It was hypothesized by Thomas and Slotnick (1962) that these lesions impaired acquisition of the CAR by causing an increased tendency to freeze in response to fear, precisely the same symptom produced by the medial thalamic lesions. The cingulate region is known to receive projection fibers from the anteroventral and anterodorsal nuclei which are closely linked (as is the medial thalamus) to part of the thalamic non-specific system (Lashley, 1941; Nauta and Whitlock, 1954). Thus, the cingulate cortex as well as the anterior and medial thalamic nuclei seem to constitute an excitatory system operating in response to fear.

Anatomical evidence for an opposing, inhibitory system is found in studies on the effects of lesions in areas of the septum, medial hypothalamus, and frontal cortex.

Brady and Nauta (1953), Thomas, Moore, Harvey, and Hunt (1959), and King (1958) have described a behavioral syndrome following septal lesions in rats which is characterized by increased startle response and more rapid acquisition of avoidance response in comparison with

normals. Brady and Nauta (1953) also found that septal rats were inferior to normals in the performance of a CER which required the inhibition of a lever response.

McCleary (1961) attributed these effects to a general loss of response inhibition normally governed by the septum and related structures. He fould the following results: (1) cats with bilateral lesions in the subcallosal (septal) area were deficient in passive avoidance behavior (inhibiting a feeding response in threat of shock) but were at least as good as normals in the acquisition of a CAR (double-grill shuttle box; and (2) cats with bilateral cingulate lesions were inferior to normals in the acquisition of the CAR but not significantly different from normals in the acquisition of the passive avoidance response. From these results and the electrophysiological work of Kabat (1936) and others, McCleary concluded that there exists an inhibitory pathway which originates in the frontal cortex, converges caudally through the septal region, and then descends to the brain stem via the ventral hypothalamus.

The possibility that the ventral hypothalamus could be involved in such a pathway was suggested by the work of Levine and Soliday (1960). Using a shuttlebox, these authors demonstrated facilitated acquisition of a CAR due to ventral hypothalamic lesions.

A possible role for the frontal cortex in the proposed inhibitory pathway is one of the major concerns

of this paper. As previously mentioned, there are many studies which indicate an inhibitory function for the frontal area in learning situations--except, possibly, those situations where the sensory cues are especially vivid and/or the required integration of motor and positional cues is not too complex. This may also be the case in learning situations involving the CER or CAR.

The effects of frontal lesions on the CER seem to be fairly consistent with the notion of an inhibitory role for the frontal cortex. Maher and McIntire (1960) preoperatively conditioned rats to freeze or crouch in response to a buzzer in anticipation of a shock (CER). In addition, a measure of "emotionality" was included by recording the amount of defecation. Rats given frontal lesions following training showed complete loss of the immobility response when presented with the buzzer (without shock)--although they defecated just as much as they had done previously when the shock was on. According to these authors, the lack of reduction in amount of defecation indicated that the animals were still just as emotional when presented with the buzzer and thus did not experience a loss of memory. It was concluded that the loss of the CER was therefore due to an inability to inhibit motor activity in a fear situation. This conclusion is consistent with the notion that the intact frontal cortex exerts an inhibitory influence in the presence of fear and that the frontal area may be functionally connected with

the septal-hypothalamic pathway discussed above.

Effects of frontal lesions on the CAR have only been investigated to date by one worker, and the results, at first glance, seem to be in the direction opposite to that predicted by the inhibition hypothesis. According to this hypothesis, since septal lesions result in normal (if not better than normal) acquisition of a CAR, the same effect should be observed in the case of lesions in the frontal area. Thompson (1963) trained rats to jump and hold onto the edge of the walls of a box with their forefeet to avoid shock signaled by an auditory or visual stimulus. Four rats were given frontal lesions while 4 control rats were given lesions in the somesthetic area. One problem with the experiment was the fact that three Ss in the frontal group were trained to an auditory conditioned stimulus (CS) while only one frontal and all four control Ss were trained to a visual CS. Even though the task was not the same for all animals, however, the results were quite clear in that all the frontals exhibited a complete loss of retention and inability to relearn the CAR while all the control animals showed 100% retention. Thompson also made lesions in the dorsomedial nucleus in another group of rats. These animals showed no retention of the CAR when either a visual or an auditory CS was used and showed considerable difficulty in relearning it. The latter results are consistent with Vanderwolf's findings when damage was done to the entire medial thalamus--except that none of the medial

thalamic rats exhibited any learning whatsoever. Further comparisons between dorsomedial thalamic and frontal rats in Thompson's study showed that all of these animals made anticipatory responses such as squeaking to the onset of the CS even when no CAR was made. However, the frontal rats differed from the dorsomedial thalamic rats in that they were still capable of making occasional "spontaneous responses" (at the wrong time) during retraining while the dorsomedial rats were not (although the unconditioned response of these animals was preformed with the same "agility and vigor" as that shown by the frontal and control rats). Thus, Thompson concluded that the dorsomedial nucleus "is a major integrating center for instigating 'anticipatory' conditioning behavior." Furthermore, since this nucleus has anatomical connections with the diffuse thalamic system (Johnson, 1961) the limbic system (Guillery, 1959) and the frontal cortex (Clark, 1948), Thompson argued that lesions in these structures should also impair the ability to initiate anticipatory responses in a fear situation.

The impairment due to frontal lesions has already been noted and seems, at first glance, contradictory to the inhibition hypothesis. In a further study by Thompson (1965), it was shown that lesions in the septofornix area abolished retention of the CAR (in the same apparatus used above) and seriously impaired relearning. These results are contrary to the effects of septal lesions found by King (1958) and

McCleary (1961). However, Thompson offered an explanation for this discrepancy by pointing out that King's lesions destroyed the more rostral region of the septum while Thompson's lesions destroyed the posterior septo-fornix area. This would be important for Thompson's notion that the dorsomedial nucleus and related structures are crucial for the CAR especially in view of recent anatomical evidence by Powell (1963) that the posteroventral septal area projects more strongly to the thalamic nuclei than does the anterodorsal septal area destroyed by King's lesions. Although Thompson did not mention the possibility, it may be the case that there are two efferent pathways which transverse the septal region, one of which projects to the thalamic system, and thus would constitute part of the cingulate-thalamic excitatory system, the other of which projects to the ventromedial nucleus of the hypothalamus, constituting part of the frontal-septal-hypothalamic inhibitory pathway proposed by McCleary (1961) and others. It is significant that ventromedial hypothalamic lesions, mentioned previously as facilitating acquisition of a CAR, result in no impairment of acquisition or retention of the CAR used in Thompson's experiments (Thompson, 1965).

Statement of the Problem

Since both the inhibitory and excitatory efferent pathways proposed above would originate in the frontal cortex, it is not surprising that the effects of frontal lesions

differ depending upon the type of avoidance response required. Thompson (1965) has stated that in the apparatus used in his experiments crouching or freezing behavior is almost never observed in either normal or lesioned animals. However, the exact opposite is found when an apparatus such as the double-grill shuttle box is used and animals are continually required to move back into the compartment that was to be avoided on the immediately preceding trial. In the latter case, the poor performers are, indeed, those rats which tend to exhibit strong freeze responses (Thomas and Slotnick, 1962). Thus, suppose for a moment that frontal lesions cause both a decreased ability to initiate an anticipatory response as well as a decreased tendency to freeze. Then, in Thompson's apparatus, where freezing is irrelevant and is never observed, the decreased ability in initiation of an anticipatory response should indeed result in a decrement in performance. However, when the double-grill shuttle box is employed, decreased anticipatory ability might conceivably be balanced out by the decreased tendency to freeze in which case no change in performance would be observed.

The purpose of the three experiments described here is to test the hypothesis that frontal lesions do not result in the impairment of learning and retention of an avoidance response in the double-grill shuttle box. Confirmation of this hypothesis would be consistent with postulated functions of the frontal cortex and related subcortical pathways discussed earlier in the paper.

EXPERIMENT I: EFFECTS OF FRONTAL LESIONS ON AVOIDANCE LEARNING

This experiment employed a double-grill shuttle box in which two compartments were separated from each other by a runway. The purpose of the runway, not traditionally included in a double-grill shuttle box, was to increase the time required for an avoidance response in order to obtain a more sensitive measure of response latencies. If frontal lesions produce a decreased tendency to freeze which balances out a decreased ability to initiate an avoidance response, the performance of frontal and normal rats should not be significantly different.

Method

Subjects.

Eighteen experimentally naive female albino rats of Sprague-Dawley[1] strain were used: At the time of surgery, they weighed 192-234 gm. Normal food intake and weight gains were observed for all Ss two weeks after surgery.

Apparatus.

The conditioning box consisted of two boxes (each 4 in. wide X 6 in. long X 6 in. high), one at each end of a runway 24 in. long X 4 in. wide X 6 in. high. The floor of the boxes and runway was a grid having 1/8 in. bars separated

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1. Ss were purchased from Dan Rolfsmeyer, Madison, Wisconsin.

7/8 in. from each other. The top of the boxes and runway was covered by wire mesh. Illumination was by means of a fluorescent ceiling light overhead. Suspended above the apparatus was a mirror enabling the experimenter to observe behavior within the box from a sitting position alongside. Each box was separated from the runway by a guillotine door which was opened manually, but simultaneously with the onset of the buzzer-shock timing device. A one ma. shock was delivered to the grid from an Applegate (model 230) constant-current stimulator. The buzzer produced a noise of 74 db. against an ambient level of 54 db. The timing sequence of buzzer and shock was controlled by three Hunter Decade Interval Timers.

Surgery.

All operations were carried out under Nembutal anesthesia following an injection of atropine sulfate. The skull was exposed and two holes, one on each side of the midsagittal suture 2-4 mm. anterior to the coronal suture, were drilled. More bone was chipped away, until approximately 2-3 mm. of tissue (the frontal poles or that area designated as Area 10 by Krieg, 1947) was exposed. Once the dura was carefully removed, the exposed cortex was aspirated through a thin metal tube attached to a vacuum pump. This latter portion of the operation was viewed under a microscope. Gelfoam was applied to the wound, and the skin was drawn together with skin clamps. An injection

of 20,000 units of penicillin was given immediately after the operation.

Postoperative Training.

Training began two weeks after the operation. Each S, selected in random order, was placed in one of the two boxes separated by the runway. This box was then darkened with a cover placed over the top. The runway and the box at the other end, however, were both well illuminated. Thus, visual as well as auditory cues were relevant to performance of the CAR.

On the first day of training, 5 escape trials were given first, followed by 10 avoidance trials. On an escape trial, the buzzer was sounded for 10 seconds, after which the door was opened manually and the start box and runway immediately electrified until S ran to the box at the other end. A covering was then placed over the goal box and another escape trial was given in like manner; i.e., after 10 seconds of buzzer, the door opened and Ss had to run to the box at the other end. After 5 such escape trials, 10 avoidance trials were given where the onset of the buzzer coincided with the opening of the door. During the 10 second duration of the buzzer, if S ran to the box at the other end of the runway, no shock was given. At the offset of the buzzer, however, if S had not reached the other box, the start box and runway were again electrified until S successfully reached the other end. The buzzer

always terminated with the onset of the shock. The inter-trial interval was 30 seconds. On each subsequent day of training, only 2 escape trials were given, followed by 13 avoidance trials. Escape and avoidance latencies were recorded on each trial. Training continued until each S reached a criterion of successful avoidance on 85 percent of the trials on any one day of training.

Results

Table 1 presents the mean learning scores for the frontal and normal groups. All animals reached criterion and Mann-Whitney U Tests[1] showed differences between groups to be nonsignificant ($p > .1$) for all measurements. (See Appendix A for all U values and corresponding p values). Using a t-test,[1] a significant decline was found in mean escape latencies within each group between first and last days of training (frontals: $t=4.8$, $df=8$, $p < .01$; normals: $t=3.1$, $df=8$, $p < .02$). Thus, escape learning was concomitant with avoidance learning. However, as stated above, no statistical differences between groups were observed in measurements of either latency or number of errors.

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1. The Mann-Whitney U Test was used instead of the t-test because the assumption of homogeneity of variance required for the applicability of a parametric test could not be satisfied. The only parametric test employed in Experiments I, II, and III is the t-test for correlated measures. Since these t's were correlated, they dealt with difference scores and did not show the heterogeneity of variance found when dealing with between-group uncorrelated comparisons. Parenthetically, it should be noted that these t's are not crucial to the main point of this paper, namely, that frontal lesions do not impair acquisition or retention of the CAR, and they are included only because they may be of interest to the reader.

Table 1

Experiment I: Mean Scores For Normal and Frontal Rats

Measure	Normals	Frontals
Errors to criterion	16.00	18.44
Sessions to criterion	2.89	3.22
Latency of escape, first session	13.14	12.68
Latency of escape, last session	11.32	11.22
Latency of avoidance, last session	3.79	3.24

EXPERIMENT II: EFFECTS OF FRONTAL LESIONS ON ACTIVE AND PASSIVE AVOIDANCE LEARNING

Since the traditional double-grill shuttle box does not employ a runway separating the two compartments, the criticism could be made that the results of Experiment I are limited only to the particular type of apparatus used. Therefore, it was decided to replicate these results using the more conventional type of shuttle-box. In addition, a slightly modified training procedure was employed and the frontal lesions were slightly larger. After training on the active CAR, an attempt was made to use the same apparatus for passive avoidance training. It was hypothesized that in frontal rats, the proposed decreased ability to initiate anticipatory responses as well as the decreased tendency to freeze should produce a net result of no impairment on the particular passive avoidance task used in this experiment.

Method

Subjects.

The subjects were ten experimentally naive female rats of the Sprague-Dawley strain. At the time of surgery, they weighed 174-255 gm.

Apparatus.

The conditioning apparatus was the same as that used in Experiment I except that the runway was omitted and the

boxes were placed together and separated by one guillotine door.

Surgery.

Operations were the same as before except that the bilateral frontal lesions were larger and resulted in removal of all dorsal and dorsolateral tissue anterior to the coronal suture.

Postoperative Training.

Task I. One week after the operations five frontal and five normal rats were trained on an active avoidance task and were required to move back and forth between the compartments at the signal of the buzzer and simultaneous opening of the guillotine in order to avoid a 1 ma. shock.

As before, each compartment could be illuminated through the top by an overhead light. Each S, selected in random order, was placed in one of the two compartments and this compartment was then darkened with a cover placed over the top. The other compartment remained illuminated by the overhead light. Following a trial, this compartment was then darkened and the other compartment became illuminated. Training was given in repeated "sessions" where each session consisted of 10 escape trials followed by 10 test trials. On an escape trial the guillotine door opened simultaneously with the onset of the buzzer and shock was given .2 seconds later. No animal was capable of making an escape or avoidance response with a latency of less than .7 seconds.

Buzzer and shock terminated together as soon as S had its entire body within the opposite compartment--at which time the door was also closed. On an avoidance trial, the door opened simultaneously with the onset of the buzzer but the shock was delayed five seconds. If S made an avoidance response by moving into the next compartment before the end of this five second period the buzzer was terminated and the door closed. The intertrial interval was 15 seconds except at the end of a set of ten test or escape trials, at which time there was an interval of one minute.

As many sessions as were needed for each S to reach criterion were given, and training of all Ss took place on one day. Criterion was defined as 9 out of 10 correct avoidance responses during any set of 10 test trials. As soon as S reached criterion he was returned to his home cage. On the next day each S was given 20 test trials intended as a test of "short-term retention." If an S did not reach criterion at this time (defined as 18 out of 20 possible avoidance responses) training was resumed as described above until S reached a criterion of 9 out of 10 possible avoidance responses on any set of 10 test trials. All Ss either reached criterion on the 20 retention trials or on test trials given during retraining.

Task II. Immediately after criterion was reached (either after the 20 retention test trials or after retraining), all Ss were trained on a passive avoidance task using the same apparatus. This task required them to wait

three seconds from the time the door went up until the buzzer sounded, whereupon they again had to move into the opposite compartment during the five second period the buzzer sounded in order to avoid shock. As before, if S did not move into the compartment during this five second period, the shock and buzzer continued until it did so. If S moved into the adjacent compartment during the three second waiting period before the onset of the buzzer, shock was delivered until S returned to the starting box, whereupon the door was closed and the trial started again 15 seconds later. Thus, the task required, first of all, passive avoidance followed by an active CAR. Two sessions of training were given each day until S reached a criterion of only two errors during any set of ten test trials. An error was defined as either going into the opposite compartment during the three second waiting period (a "passive error") or failing to avoid during the five second duration of the buzzer preceeding the shock (an "active error"). At the end of five days, if criterion was not reached, training was terminated.

Task III. The animals that reached criterion on Task II were then trained on Task III which was exactly the same as Task II except that the waiting period was now extended to six seconds instead of three seconds.

Results

Table 2 presents the mean learning, retention, and relearning scores for both groups on Task I. Both frontal and normal Ss experienced a significant decrease in latency of escape responding between the first and last ten escape trials (frontals: $t=7.6$, $df=4$, $p < .001$; normals: $t=9.9$, $df=4$, $p < .001$) showing that increased ability to escape shock accompanied learning of the avoidance response. However, Mann-Whitney U Tests failed to show a significant difference ($p > .1$) between groups in either number of sessions, errors to criterion, escape latencies on first or last ten escape trials, or latencies of avoidance on the last ten test trials of training (see Appendix B for U and p values). As far as retention on the next day was concerned, Mann-Whitney U Tests also failed to yield significant differences between groups on either number or errors ($U=11$, $p > .1$) or latencies of avoidance ($U=9$, $p > .1$). As shown in Table 2, an equal number of Ss in each group failed to reach criterion on the retention test. However, the mean latency of avoidance for normal Ss tended to increase (see Table 2) between the last ten test trials of training and the retention test trials, although this increase was not significant ($t=1.5$, $df=4$, and $p > .05$), while the frontal Ss experienced a significant decrease in mean latency of avoidance ($t=4.1$, $df=4$, $p < .02$). Thus, when mean difference scores (between last 10 test trials

Table 2

Experiment II: Mean Learning, Retention and Relearning Scores for Normal and Frontal Rats on Task I

Measure	Normals	Frontals
LEARNING		
Errors to criterion	14.00	18.40
Sessions to criterion	2.80	3.80
Latency on first ten escape trials	1.81	1.54
Latency on last ten escape trials	.90	.87
Latency of avoidance on last ten test trials	1.28	1.77
RETENTION		
Errors	4.4	3.8
Latency of avoidance[1]	1.88	1.41
Number of rats not avoiding on 18 out of 20 test trials	3	3
RELEARNING		
Errors to criterion	2.5	1.67

[1]Including scores for those rats that did not reach the criterion of 18 out of 20 correct retention trials.

and the twenty retention test trials) for between groups were compared, a Mann-Whitney Test was significant at the .056 level ($U=3$). As far as relearning is concerned, no significant difference between groups was found in number of errors to criterion ($U=2$, $p > .05$).

Table 3 presents mean learning scores for normal and frontal rats on Task II.

Although the mean scores appear quite divergent in many cases, there were no significant differences between groups on any of the measures, Mann-Whitney U Tests all yielding $p > .05$ (see Appendix C for U and p values). Since one rat in each group did not reach criterion, their scores are not included in the analyses.

Table 4 presents mean learning scores for normal and frontal rats on Task III (six-seconds passive avoidance). Large variance and the small number of Ss within each group precluded adequate analyses of these data. However, it may be significant to note that an equal number of Ss in each group were able to reach criterion, and the means between groups do not appear widely divergent from one another.

Table 3

Experiment II: Mean Learning Scores for Normal and Frontal Rats on Task II

Measure	Normals	Frontals
Errors to criterion		
Passive errors on escape trials	6.50	3.40
Passive errors on test trials	3.0	1.5
Active errors on test trials	9.5	2.0
Errors on first session		
Passive errors on escape trials	3.0	3.40
Passive errors on test trials	1.00	1.60
Active errors on test trials	2.80	1.60
Latency		
Escape trials: first session	.87	.89
Escape trials: last session	.95	.89
Test trials: first session[1]	1.23	1.65
Test trials: last session	1.51	1.80
Sessions to criterion	3.25	1.25
Number of rats not reaching criterion	1	1

[1]Only latencies of the correct (avoidance) response were included..

Table 4

Experiment II: Mean Learning Scores for Normal and Frontal Rats on Task III

Measure	Normals	Frontals
Errors to criterion[1]		
Passive errors on escape trials	6	5.7
Passive errors on test trials	5.0	6.3
Active errors on test trials	8.3	12.0
Errors on first session		
Passive errors on escape trials	1.75	2.50
Passive errors on test trials	1.75	2.25
Active errors on test trials	2.75	2.50
Latency		
Escape trials: first session	.77	.86
Escape trials: last session	1.00	.88
Test trials: first session[1,2]	1.74	1.63
Test trials: last session	1.86	1.50
Sessions to criterion	3.33	3.33
Number of rats not reaching criterion	1	1

[1]Scores were not included for the single rat in each group who did not reach criterion.

[2]Only latencies of the correct (avoidance) responses were included.

EXPERIMENT III: EFFECTS OF PARIETAL LESIONS ON AVOIDANCE LEARNING AND THE EFFECTS OF PARIETAL AND FRONTAL LESIONS ON RETENTION OF A LEARNED AVOIDANCE RESPONSE

Experiments I and II seemed to offer strong evidence that frontal lesions (large or small) do not disrupt active avoidance learning in the double-grill shuttle box and even facilitate performance on a short-term retention test. However, it was deemed necessary to make lesions in an area immediately adjacent to the frontal cortex in order to provide a better control for the effects of cortical lesions per se. For this reason, the effects of parietal lesions on both learning and retention of the CAR were investigated employing the same apparatus (with a slightly modified training procedure) used in Experiment II. Furthermore, the effects of frontal lesions upon retention of a preoperatively trained CAR (not investigated in either Experiment I or II) were compared to the effects of parietal lesions, under the same experimental conditions.

Method

Subjects.

Twenty-two experimentally naive female rats of the Sprague-Dawley strain were used during the course of this experiment. Their weights at the time of the first surgical operation ranged from 184-233 gm.

Surgery and Training Procedure.

Prior to any training, six of the rats were given bilateral parietal lesions resulting in removal of all dorsal and dorsolateral tissue 4-5 mm. posterior to the coronal suture. In all other respects the surgical procedure was the same as that described in Experiment II except that ether instead of nembutal was used throughout the operation.

One week later, the six parietal rats and sixteen normal control rats were trained on task I in the manner described in Experiment II except that only two training sessions were given each day until S reached criterion. Training was terminated eight days later for those Ss that failed to reach criterion during this period.

Each S, upon reaching the criterion of 9 out of 10 avoidances on any set of 10 test trials was placed in his home cage and allowed to rest without interruption for one week, whereupon he was then given 10 test trials as a retention test. Following retention, eight of the 16 control Ss were given frontal lesions as described in Experiment II and the other eight control Ss were given parietal lesions. Two Ss in each group died during surgery. One week after the operation, each S was again given 10 test trials and the difference in number of errors between this set of test trials and the 10 test trials given before the operation was measured. Those Ss that

did not reach criterion were retrained until they did so.

Results

Table 5 presents some of the mean learning scores for the 16 normal Ss and the 6 postoperatively trained parietal Ss. Mann-Whitney U Tests showed a significantly greater number of days required for training in the parietal than in the normal group ($U=5$, $p < .02$) but no significant difference in number of errors to criterion ($U=27$, $p > .1$). Furthermore, on the first day of training, the parietals made significantly fewer total number of errors than did the controls ($U=6.5$, $p < .002$) even though the escape latencies of the parietals were significantly higher ($U=16$, p .02). These differences, however, disappeared later in training. Thus, no significant differences ($p > .05$) in total number of errors between groups were found for the second ($U=32$, $p > .05$) or third day ($U=24$, $p > .05$) of training and differences in escape latencies on the last 10 escape trials of training were not significant ($U=48$, $p > .1$).

Table 6 presents mean scores before and after learning within each group. Both normals and parietals experienced a significant decrease in mean latency of escape responses between the first and last 10 escape trials (parietals: $t=6.1$, $df=5$, $p < .01$; controls: $t=6.8$, $df=15$, $p < .001$); also, the normal Ss experienced a significant decrease in

Table 5

Experiment III: Comparison of Mean Learning Scores of Normal and Parietal-Lesioned Rats

Measure[1]	Normals	Parietals
Errors to criterion	34.31	51.67
Days to criterion[2]	4.69	7.50
Total errors, first day of training	17.18	10.67
Total errors, second day of training	7.44	10.00
Total errors, third day of training	3.69	7.17
Latencies on first ten escape trials	1.64	2.42
Latencies on last ten escape trials	.95	.96

[1]Mean scores of the three parietal and two normal animals that did not reach criterion by the eighth day of training are included.

[2]Animals which failed to reach criterion were arbitrarily assigned a score of 8.0.

mean latency of avoidance responses (t=3.1, df=14, $p < .01$) between the last 10 test trials of training and the 10 retention test trials given one week later. No retention measures are included for the parietal group since three out of six Ss failed to reach criterion even by the end of the eight days allotted for training. Thus it is apparent that since parietals were inferior to normals in number of days required for training but not in the total number of errors made during this period, that the parietal Ss had a relatively high level of avoidance responding, but one which was not quite high enough to reach criterion.

Table 7 presents the retention scores for the normal Ss that received either frontal or parietal lesions after reaching criterion. Results show that on the retention test all of the frontals still reached criterion whereas none of the parietals did so. When the mean differences between errors on the ten retention test trials given before and after the lesions were compared for the two groups, a Mann Whitney U Test showed that the mean increase in errors of 4.6 for the parietals was significantly greater (U=0, $p < .008$) than the mean decrease of .20 for the frontals. Five of the six parietals, however, were capable of relearning the task to criterion--with significantly fewer errors than during original training (t=3.8, df=4, $p < .02$), and relearning usually took place within one or two days. Thus, the deficit in retention

Table 6

Experiment III: Mean Scores Before and After Learning--Within Normal and Parietal Groups

Measure	Normal	Parietal
Latency on first ten escape trials	1.64	2.42
Latency on last ten escape trials	.95	.96
Latency of avoidance on last ten test trials of training	1.50	--[1]
Latency of avoidance on ten retention test trials (one week after training)	1.18	--
Errors on ten retention test trials (one week after training)	.60	--

[1]Since three of the six parietal animals failed to reach criterion, the mean scores for the entire parietal group have been omitted for the last three measurements.

Table 7

Experiment III: Mean Retention Scores for Frontal and Parietal Rats

Measure	Frontal	Parietal
Per cent reaching criterion	100	0
Preoperative retention errors	.60	.40
Postoperative retention errors	.40	5.0
Postoperative minus preoperative retention errors	-.20	4.6
Preoperative retention avoidance latency[1]	1.16	1.34
Postoperative retention avoidance latency[1]	1.38	2.07
Postoperative minus preoperative retention avoidance latency[1]	-.22	.73

[1]Since none of the parietals reached criterion on the first ten retention trials after the lesion, their postoperative retention avoidance latencies were taken from the last ten test trials of retraining to criterion.

seemed only temporary in the case of the parietal lesions.

The mean latency of avoidance responses tended to increase for frontal Ss between the 10 retention test trials given before and after the lesions. However, this increase proved to be nonsignificant ($t=2.2$, $df=4$, $p > .05$). Mean latency of avoidance responses for the parietal Ss, however, increased significantly between the 10 test trials before the lesion and the last 10 test trials during retraining to criterion ($t=2.9$, $df=4$, $p < .05$). These mean increases for each group did not prove to be significantly different from each other ($U=6$, $p > .05$).

GENERAL DISCUSSION

The results of Experiments I and II are straightforward: frontal lesions do not significantly impair the acquisition of a CAR in the double-grill shuttle box. In fact, on Task I in Experiment II, the decreased latencies of avoidance found the next day after criterion was reached suggest that frontal lesions facilitate performance on a short-term retention test. In contrast, the normal Ss in Experiment I did not show a significant decrease on the short-term retention test but the normal Ss in Experiment III did show such a decrease when retention was measured one week later. Perhaps this decrease in avoidance latency indicates a decreased tendency to freeze over the passage of time. If so, a facilitation of this decrease after frontal lesions would be consistent with the notion that the frontal cortex exerts an inhibitory influence in a fear situation. As far as long-term retention is concerned, the results of Experiment III show that frontal lesions, when given after training, do not result in any loss of retention, either in terms of response latency or number of errors.

Experiment II does not provide clear-cut evidence concerning the effects of frontal lesions on passive avoidance learning. The fact that just as many frontal as normal Ss proved capable of learning the task suggests that frontal lesions do not cause any change in normal ability to learn

a passive avoidance task. However, since the variation was large within each group for both tasks and the number of Ss in each group was small, due to a failure of many Ss to learn the task, adequate statistical analyses of the results were not possible. Thus, any conclusion on the basis of these results must be tentative.

The results of Experiment III, showing deficits in both learning and retention of the CAR after lesions in the parietal area, require some explanation. As far as ability to learn was concerned, parietal Ss showed the following characteristics: (1) a higher initial escape latency during escape trials, (2) fewer errors during the first day of training, in comparison with normal Ss; and (3) the ability to make avoidance responses at a relatively high level without quite reaching criterion (at least half of the parietals did this).

There is additional evidence which may aid in explaining these results: first of all, it must be pointed out that on the first day of training, when the parietals made significantly fewer errors than the controls, the experimenter noticed (after training) that four parietal Ss did not have food in their cages. Thus, it is possible that these Ss were hungrier than normals (whose cages contained food) and consequently maintained a higher level of activity. Secondly, it is extremely difficult to make large lesions in the parietal area without causing some damage to the underlying cingulate region.

Thirdly, as mentioned earlier, Thomas and Slotnick (1962) found that damage to the cingulate area impaired acquisition of a CAR in a double-grill shuttle box. Last of all, a more recent study by Thomas and Slotnick (1963) showed that cingulate Ss were inferior to normal Ss when both were under a low hunger drive, but actually superior to food-satiated normals when under a high hunger drive. These authors concluded that the effects of an increased tendency to freeze in the double-grill shuttle box following cingulate lesions was overcome by an increased tendency to move, due to a high hunger drive. Thus, with respect to the parietals in this experiment, it is possible that their initial superiority on the first day of training was due to the combined effect of cingulate damage and a high hunger drive. This would also explain their inferior performance in comparison with normals during subsequent training sessions when both groups of Ss were food satiated.

It is more difficult to explain why the parietal Ss had a higher initial escape latency than normals. It is possible that parietal lesions, by destroying the somesthetic area, may change the cutaneous perception of pain. Further research would be needed, however, to test this notion.

The major accomplishment of this paper is the demonstration that frontal lesions do not impair the acquisition or retention of a CAR in the double-grill shuttle box. This is consistent with the hypothesis that decreased ability to initiate an anticipatory response is balanced out by a

decreased tendency to freeze. This hypothesis, together with the postulated inhibitory and excitatory pathways discussed earlier may explain how frontal lesions would cause a deficit in the CER (Maher and McIntire, 1960) as well as a deficit in the CAR when freezing behavior is irrelevant to performance (Thompson, 1963) but cause no deficits in the CAR in the double-grill shuttle box (the present study).

SUMMARY AND CONCLUSION

On the basis of studies of frontal and subcortical lesions it was hypothesized that both an inhibitory and an excitatory pathway originate in the frontal cortex. Thus, it was argued that a frontal lesion should result in both a decreased tendency to freeze as well as a decreased tendency to initiate an anticipatory response. These opposing tendencies should produce a net result of no decrement in the double-grill shuttle box where both freezing and anticipatory tendencies are relevant to performance of the CAR. The following results, using the double-grill shuttle box were consistent with this hypothesis:

1. Frontal lesions failed to disrupt both learning and retention of the CAR.
2. Effects of lesions in the frontal cortex were not found in the case of lesions in the adjacent parietal-cingulate region where impairment of both learning and retention was found.

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APPENDIXES

APPENDIX A

Mann-Whitney U Values for
Experiment I

Measure	$U^{1,2}$
Errors to criterion	36
Sessions to criterion	48
Latency of escape, first session	60
Latency of escape, last session	33.5
Latency of avoidance, first session	57

[1] $n_1 = n_2 = 9$.

[2] p .1 for all values.

APPENDIX B

Mann-Whitney U Values for
Experiment II: Task I

Measure	$U^{1,2}$
LEARNING	
Errors to criterion	16.5
Sessions to criterion	5
Latency on first ten escape trials	9
Latency on last ten escape trials	7
Latency of avoidance, last ten test trials	8
RETENTION	
Errors	11
Latency of avoidance	9
RELEARNING	
Errors to criterion	2

[1] $n_1 = n_2 = 5$, except for the relearning condition where $n_1 = 2$ and $n_2 = 3$.

[2] p .1 for all U values.

APPENDIX C

Mann-Whitney U Values for
Experiment II: Task II

Measure	n_1	n_2	U^2
Errors to criterion			
Passive errors on escape trials	4	4	6.5
Passive errors on test trials	4	4	7.5
Active errors on test trials	4	4	4
Errors on first session			
Passive errors on escape trials	5	5	9.5
Passive errors on test trials	5	5	10.5
Active errors on test trials	5	5	10
Latency			
Escape trials, first session	5	5	12
Escape trials, last session	5	5	8.5
Test trials, first session	5	5	6
Test trials, last session	4	4	4
Sessions to criterion	4	4	3

[1] p .2 for all U values

THE EFFECTS OF FRONTAL LESIONS ON LEARNING AND RETENTION OF AN AVOIDANCE RESPONSE IN THE DOUBLE-GRILL SHUTTLE BOX

by

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The primary purpose of this study was to investigate the effects of frontal lesions on learning and retention of a conditioned avoidance response (CAR) in the double-grill shuttle box. In Experiment I, nine frontal Ss and nine normal Ss were found not to be significantly different in the acquisition of a CAR in a shuttle box having the two compartments separated by a runway. In Experiment II, five frontal Ss were found not to be significantly different from five normal Ss in acquisition of a CAR in a shuttle box having the two compartments adjacent to one another. However, frontals were shown to be superior to normals on a short-term retention test. Following the completion of this test, two passive-active avoidance tasks were given but the results were equivocal. In Experiment III, the effects of parietal lesions on acquisition of the CAR were investigated in order to obtain a better control for the effects of frontal lesions. Six parietal Ss were found to require a significantly longer period of training on the CAR than sixteen normal Ss although they did not have a significantly greater number of errors. On the first day of training, however, these parietals did have significantly fewer errors than the normals even though their escape latencies were higher. These results were interpreted as indicative of damage to the cingulate region. Of the fourteen out of sixteen normals that had reached criterion on the CAR, six Ss were given frontal lesions, six other Ss

were given parietal lesions and retention was then measured one week later. Results showed that all the frontal Ss still reached criterion on the retention test whereas none of the parietals did so. Five out of six parietals, however, relearned to criterion significantly faster than they had done when first trained to criterion. The decrement in performance for parietal Ss was again interpreted as indicative of damage to the cingulate region.

The results of these experiments are consistent with the hypothesis that frontal lesions produce both a decreased ability to initiate anticipatory responses as well as a decreased tendency to freeze thereby producing no impairment of performance in a double-grill shuttle box.