The effect of frontal lesions on frustration-fixation behavior in the rat.

Dennison Alvord Smith
University of Massachusetts Amherst
THE EFFECT OF FRONTAL LESIONS ON
FRUSTRATION-FIXATION BEHAVIOR IN THE RAT

Dennison A. Smith
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INTRODUCTION
Although the function of the frontal cortex is largely unknown, there appear to be two distinct symptoms that result from frontal lobe damage. One major symptom is an impairment of the ability to inhibit motor activity or preferred modes of response. The other symptom is a change in mood and attitude. These symptoms can best be illustrated by describing the behavioral changes that occur in man after frontal damage.

The first kind of impairment, an inability to suppress certain kinds of motor activity, expresses itself in man in the form of restlessness, hyperactivity, perseveration of motor movement, task perseveration, and an inability to suppress preferred modes of response (Milner, 1964; Teuber, 1964; Luria, 1965). Luria (1965) has described two kinds of motor perseverations that result from damage to frontal areas in man. The first type of motor perseveration results from damage to more medial frontal areas and motor ganglia of the striatum. This behavioral alteration appears as compulsive repetitions of movements. In this case, the inertia of a motor act once begun is not easily stopped. The other sort of perseveration is a perseverance of a program of action. In this case, once the frontal patient begins the task, he is unable to switch his behavior to accomplish other tasks. This kind of impairment is more closely associated with damage to lateral areas of the frontal lobe. It is not surprising then that humans
with damaged dorso-lateral frontal cortex show impairment on the Wisconsin Card Sorting Task where they must switch from sorting cards into color groups, to sorting cards according to the number of figures on the card or according to the shape of the figures (Milner, 1964). Likewise, subjects with more posterior frontal damage, when asked to draw only one circle draw more than one; once begun the act could not be inhibited (Luria, 1965). In summary, it appears that humans with frontal lobe damage show impairment on all tasks in which they must alter responses or switch from one response set to another.

The other category of behavioral change that results from frontal lobe damage in humans is the alteration of the patient's moods and attitudes (Fulton, 1951). Frontal lobotomies have long been used in cases of intractable pain and anxiety in order to alleviate the anxiety. In the case of pain, the person reports that he feels the pain but he is undisturbed by it. The patient lacks foresight as to his role in the course of events and thus does not anticipate the consequences of the pain (Teuber, 1964).

Animal Behavior

Hyperactivity:

Behavioral deficits in prefrontal humans have their analogues in animal behavior. Motor unrest is common in animals with frontal lesions (Beach, 1941; Kennard et al.,
1941; Ruch and Shenkin, 1943). This motor unrest is usually accompanied by distractability and excitability. Such hyperactivity results largely from visual stimulation since blind frontal monkeys or frontal monkeys placed in the dark are not hypermobile (Kennard et al., 1941; Issac and Devito, 1958). Hyperactivity is specific to locomotion (Ruch and Shenkin, 1943). Presumably, the hyperactivity results from a release of inhibitory control exerted by the posterior orbital frontal cortex on somatomotor activity (Kaada, 1960).

**Habit perseveration (monkeys, dogs and cats)**

Animals with frontal lesions also show motor and habit perseverations. Mishkin (1964) refers to this as "an inability to suppress whatever response normally prevails in the given situation." Just as in human perseveration, the motor habit or act "once initiated gets stuck and persists indefinitely, being continuously executed or perseverated in spite of the absence of the stimulus originally responsible [for it's initiation.]" (Brutkowski, 1965) This deficit accounts for the frontal animal's inability to perform tasks of delayed response, alternation, reversal or any other task that requires the animal to alter his response set or motor act. Examples of this kind of deficit are numerous.

Settlage and his associates (1956) found that if monkeys trained on a black-white discrimination problem were required to perform a position discrimination after
after lobotomy, the frontal animals were worse than the control group in their ability to learn the new task. This was also true of monkeys that learned a left-right discrimination and then after lesioning were required to learn the black-white discrimination. Settlage attributed this deficit to an inability to suppress, modify, or forget previously learned behavior patterns.

Brush et al. (1961) found that monkeys with lesions of the frontal cortex have difficulties in overcoming experimentally established object or stimuli preferences or aversions. Using the W GTA, their monkeys were first given two test objects. In the first trial, the informing trial, objects could be rewarded, the baited condition, or unrewarded, the unbaited condition. In the baited condition, the task on the following ten test trials was to continue to choose the object which on the first trial was associated with reward. In the unbaited condition, the task in the following test trials was to switch responses to the object that had not been associated with nonreward on the informing trial. Monkeys were found to be impaired only in the unbaited condition in which they had to choose the object which they did not choose during the informing trial.

Mishkin (1964) has extended the original findings of Brush. He found similar results in a situation where he presented one object alone for five trials and either baited it or left it unbaited. This was followed by test
trials in which the animal had to choose the object if it had been reinforced or pick the other object if the original was unbaited. Again the frontal animals were impaired in the situation where the original object was unrewarded and the animals had to choose the other object. Impairment resulted even in this case where the animals had to suppress their response associated with the nonrewarded object. This showed an inability to alter the response set once it was established in the original five informing trials.

Mishkin concluded that it is neither stimuli nor object preferences or aversions that perseverate, nor is it responses that perseverate; but rather it is "central sets", or what might be called innate response tendencies, that once initiated dominate the behavior of frontal monkeys in choice situations. He bases this conclusion on the studies previously mentioned and on a study of one trial learning. In this latter experiment, he presented an object for one trial and baited the object or did not bait it. In the following trials the object was paired with various other objects. As before, in the baited condition, the monkey was required to choose the originally rewarded object. In the unbaited condition the animal was required to choose the other object in order to be rewarded. In this case the monkeys always chose the novel object. Mishkin interpreted this as the perseveration of a central set of choosing novel objects (Mishkin, 1964).
Recently, however, Mishkin's conclusion has been put in doubt by a study done by French et al. (1965). They conducted a two choice, simultaneous discrimination problem in which objects associated with reward or nonreward were either held constant or varied. It was expected that if animals had an abnormal tendency to choose novel objects, then they would have more trouble with the constant-varied (C+V-) condition in which the rewarded object was always the same (constant) and the nonrewarded objects were varied, than with the varied-constant (V+C-) condition in which the rewarded objects were novel and the constant object was unrewarded. In French's study no tendency to choose novel objects was seen. In reversal situations, where constant rewarded objects were unrewarded or where constant unrewarded objects were rewarded, or in situations in which the rewarded object was changed from its original constant or varied form, the frontal animals showed more impairment than the controls. The one exception to this was that the normal animals were more impaired in reversal situations where aversion to the constant stimulus was first established and then the constant object was rewarded, than in a task where a preference was first established for the constant object and in reversal the choice was not rewarded. In other words, the V+C- to C+V-condition was harder to learn than the C+V-to V+C-condition. The experiment not only demonstrated that aversions are hard to overcome when the rewarded object is
varied and hard to learn, but it also shows the pre-reversal training did not set up an abnormal tendency to choose the varied object since in the $V+C-$ to $C+V-$ situation the frontals did not make significantly more errors than they did in the $C+V-$ to $V+C-$ condition. French and his associates concluded that frontal subjects were able to achieve or maintain successful performance as long as one object of a stimulus pair remained constant in its stimulus characteristics and in its association with reward or nonreward.

Much of Pribram's work supports the notion that frontal animals are impaired in situations in which discriminanda or the outcomes of choice (reward or punishment) are not held constant (Pribram, 1964, 1967).

In his earlier work, Pribram and his associates (1964) explored the behavior of monkeys in multiple choice situations. They found that frontal monkeys persisted longer than non-lesioned animals in choosing objects that had previously been reinforced but were now no longer rewarded. This was not true if a novel object was introduced as the rewarded object at the time reinforcement conditions were changed. Nor was it true if all but the previously rewarded object were reinforced at the time problem conditions were altered. In both cases frontal monkeys made fewer errors than did the control animals.

A paradox seems to exist. In certain situations frontal subjects persist longer in their previous response pattern, while in others, frontal animals more readily
switch to new responses. Pribram believes that the difference lies in the fact that in the first situation, a shift in response to one of the other objects did not always result in reinforcement, while in the other two experiments shifting to a novel cue or shifting to any of the other objects was always rewarded. According to Pribram, this behavior is representative of an increased sensitivity to reinforcement in the frontal animal.

While it may be that one result of frontal damage is an increased sensitivity to reinforcement, Pribram has more recently stated that the most important function of the frontal cortex is its role in recent memory (Pribram, 1967). He feels that the deficit seen in frontal subjects is the result of an inability to properly code or resolve successive input information. This conclusion would also predict that any ambiguity in terms of reinforcement conditions or cues associated with reinforcement would result in less ability to solve the problem. Because the animal cannot solve the problem, it may revert to random behavior or even more likely, it may continue with its previous response.

Another situation in which perseverative behavior has been found is in go-no go situations. In these situations the subject does not choose where to go according to the stimuli presented, but it chooses whether to go or not to go on the basis of the cues presented. A series of exper-
Iraents, (Pribram, 1956; Mishkin and Pribram, 1955) have shown that monkeys are impaired in delayed alternation tasks where identical cues are presented in left-right positions or in up-down positions. In these cases, the monkeys respond repetitively to one of the stimuli. They reasoned that the deficit of the frontal animal might be limited to situations in which cues have different spatial dimension, however, the spatial dimension is removed in go-no go tasks. Here one cue is presented that signals approach and another cue is presented that signals not to approach. Mishkin and Pribram have not found frontal monkeys to be impaired on these tasks. However, this cannot be attributed to the removal of the spatial aspect of the task since frontal animals are impaired on nonspatial object alternation tasks. Furthermore, other experiments have shown that frontal monkeys and animals with caudate lesions often do show deficits in go-no go situations (Battig et al., 1962). In Battig's experiment the task was to displace a cardboard plaque for food when a tone was on, and not to displace it when the tone was off. This task was also done using color and pattern cues. The difference between the Pribram and Mishkin studies and the experiment by Battig and his co-workers, is that all of Battig's animals had received long training on displacing a plaque before the lesion.
The monkeys in the Mishkin and Pribram study were lesioned first and then were trained to approach or avoid. Thus Battig's animals had a strong response tendency to displace the plaque for food and could not overcome this tendency through training.

These go-no go situations have many similarities with much of the conditioning work with frontal animals (Brutkowski, 1964, 1965). Brutkowski has reported disinhibition of response on negative trials both in classical and instrumental conditioning. The situation in the instrumental conditioning task in analogous to a go-no go situation. Using frontal dogs, Brutkowski trained them to place a paw on a food tray when the positive C. Ss. (auditory, visual, tactile) were presented, but not to do it when the negative C. Ss. were given. Animals were unable to withhold leg placing responses on negative trials after they were given frontal lesions.

**Habit perseveration (rats)**

Experiments with rats have demonstrated perseverative interference resulting from ablation of the frontal poles. The deficits produced by frontal ablation appear to be largely limited to maze performance. Bourke (1964) has shown that rats show no deficits in performing Y maze discrimination tasks or the reversal of the task. Frontal rats could also initially learn a four choice maze but were impaired on the reversal of the task. He explains
the deficit in the maze task in terms of task complexity. The more complex the task, the more perseverative interference.

Dabrowska (1964a, 1964b) has found that rats with ablated frontal regions show deficits in maze habits which appear to be due to the rat's inability to perform chained motor acts. He found that in a situation where the rats were required to learn a maze in which there were four choice points with four entrance points at each choice point, that animals took approximately fifty trials to master the maze. The task the rat had to perform was to choose all the entrances on the right, all those at the left, or all the second entrances from the right etc. With successive changes of the correct choice points, all rats showed faster mastery of the new correct pathway. After the third change, animals required approximately twenty trials to learn the reversal. Frontal lesions were given to half the rats and then all animals were tested again on further reversals. The shamoperated control animals were better on the changed choice points after the operation than were the frontal animals and they improved with successive reversals. However, after the operation the frontal rats took about fifty trials to learn the changed choice point and did not improve with successive reversals. It was noted that if the task was made more difficult by making choices dissimilar at the various choice points,
no animal mastered the task. Dabrowska does not see this as a deficit in the rat's capacity to learn how to learn, but rather as a loss of the ability to chain motor acts. The situation with different choices at the different choice points shows the importance of this ability in these maze problems.

Lukaszewska (1963, 1964) feels that even normal rats show strong perseverative tendencies. She attributes this to the small amount of prefrontal cortex in the rat. She found that if normal rats, after reaching the goal box in a Y-maze, were trained to return to a start box in an arm different from the one in which they had originally started, that the rats had a strong tendency to retrace the path they made on their original run. Normal rats could be trained to overcome this tendency but rats with frontal lesions could not. In contrast, the Y-maze discrimination task in which the animal must just run to the correct stimuli is a task which the frontal rat appears to be able to solve (Thompson, 1964). Lukaszewska (1964) has further shown that maze problems depend primarily on proprioceptive motor cues for their mastery. She has found that it is harder for rats to overcome perseverative tendencies in Y-mazes where the angles are slight, as opposed to mazes in which there are sharp angles. Furthermore, animals that were blinded and thus deprived of visual-motor cues showed no more than normal impairment
in learning the correct return run to the start box.

It should be noted at this point that rats trained on the Lashley Jumping Stand show strong response preferences. When given a choice they often only jump to one side or to one of the stimulus windows. Unlike the simple Y-maze discrimination task, punishment is an important part of the jumping stand situation. Since the rat that must solve a problem on the Lashley Jumping Stand shows much perseveration normally, it would be assumed that performance of this task might be differently affected by frontal lesions than the Y-maze discrimination task used by Thompson.

**Alterations of emotionality (reduced fear)**

The other common result of lobotomy in animals, as in man, is an alteration in emotionality and motivation. Many studies have concluded that frontal lesions reduce fear and raise the animal's threshold to frustration. Streb and Smith (1955) noted the elimination of the conditioned emotional response of crouching in rats after frontal lesions. Maher and McIntire (1960) also noted the loss of the CER of crouching after frontal pole lesions but the autonomic response of defecation was not eliminated. Lichtenstein (1950) found that lesions of the frontal lobes eliminated feeding inhibition in dogs that were shocked while they ate. The related anxiety symptoms of barking, tachycardia, tremors and disordered respiration
were also eliminated. Waterhouse (1957) abolished a conditioned fear discrimination ability in monkeys after frontal ablations but a conditioned food discrimination was not abolished. Using a DRL testing situation, Stamm (1964) measured the number of timed responses and the number of multiple responses, those responses occurring within two seconds after the initial response was made. In this type of problem the animal must slow its rate of response as pressing the bar again too soon leads to no reinforcement. In this problem frontally ablated monkeys showed fewer multiple responses than the controls. This could not be attributed to a motivation reduction since frontal monkeys did not show a lower than normal number of timed responses. Stamm feels that frontal lobotomies raise thresholds to frustration and consequently frontal animals do not show the frustration response of multiple bar pressing.

Miles (1960) also finds that frontal lesions raise frustration thresholds. Animals were trained to make an approach response of displacing a block for a food reward. After a strong approach response was established the animal was punished three times during every one hundred trials by having a bar fall on his hand as he reached to displace the block. Latencies were recorded on post-punishment and post-reward trials. For the frontal monkeys no drop in latency was observed on post-punishment trials. This was not true for the control group. Finally, a typical finding
in the work done with frontal animals is the impairment on active avoidance tasks (Cornwell, 1966). Presumably this deficit can be attributed to elimination of the fear response in the avoidance task.

**Alterations in emotionality (increased fear)**

In contrast to the evidence just presented, other studies have shown that frontal lesions in various animals may have the opposite effect of sensitizing the animal to reinforcement or nonreinforcement. This may result from a release of inhibitory control on the autonomic and reticular activating systems. Brutkowski (1964) has shown that prefrontal dogs, besides showing disinhibition of conditioned responses to negative C. Ss., also show larger conditioned and unconditioned responses to positive C. Ss. and U. C. Ss. This is representative of an increase in emotionality according to Brutkowski. He interprets this change as an increase in drive and not a loss of general inhibition. He views the hypothalamus as a center inhibited by frontal cortical areas and concerned with the modulation of drives and motivated behavior (Brutkowski, 1964, 1965). Pribram (1964) has also adopted a similar interpretation.

Other evidence supporting this view of increased emotionality is that of Weiskrantz and Wilson (1958), who have shown that monkeys' threshold to shock is lowered in a Sidman-avoidance situation after removal of the lateral
frontal cortex. Aulenyter and Brutkowski (1960) have shown a decrease in latency of the classical defense reaction in dogs after frontal ablations.

In regard to the previous evidence showing that frontal animals are impaired in active avoidance situations, both Brutkowski (1964) and Maher et al. (1961) feel that the deficit is the result of hyperemotionality and hyperactivity that produce competing responses.

Although these studies on the behavior of frontal animals lead to a diversity of conclusions, it appears that most of the results can be explained in terms of just two effects. First, frontal lesions appear to make the animal hyperreactive to all stimuli, especially reinforcement. Secondly, frontal lesions, as Pribram suggests, appear to produce a deficit in recent memory such that ambiguous discriminanda or ambiguous reinforcement conditions are not easily learned. Successive input information abnormally interferes with previously learned associations in the frontal animal.

Most of the studies can be explained by these two conclusions. Certainly, the maze performance of frontal rats can be reduced to a simple inability to perform a chained motor sequence which may merely indicate a loss in recent memory. This deficit might also explain the difficulty that frontal monkeys show when they must perform an alternation task (Pribram, 1967). Somehow
the animal seems to forget which response in the sequence it must make next. Likewise, many of the experiments related to habit perseveration reduce to either a problem of increased sensitivity to reinforcement or a recent memory problem. For instance, Settlage and his associates (1956) found frontal monkeys to be deficient in their ability to switch from responding to a problem according to dark or bright cues, to responding to the problem according to position. Changing the task altered the meaning to the dark and bright cues, and they no longer signaled the correct or the incorrect response. Their meaning as to reinforcement outcome was therefore somewhat ambiguous. When the meaning of the cues was altered between the two tasks, the frontal animal had difficulty in assigning an outcome to them.

The experiments in which object or stimuli preferences were established by reinforcement are also explicable using these ideas. Frontal animals persist longer in choosing previously rewarded objects after they are no longer because of their increased sensitivity to reinforcement. Since the reinforcement value of the stimulus might be raised for the frontal animal, it takes longer to extinguish its response. Likewise, the frontal animal would be confused because the stimulus cues are no longer associated with a specific reinforcement outcome.
Evidence concerning changes in emotionality in frontal animals can be easily explained from this point of view. On the one hand, evidence of hyperemotionality is indicative of the frontal animal's increased sensitivity to reinforcement. On the other hand, decreased emotionality is evidence of the frontal animal's inability to link events in temporal sequence. The inability to anticipate the outcome of a stimulus cue or event when it's meaning was ambiguous for the animal would appear as hypoemotionality.

A study by Zielinska (1966) clearly indicates that the frontal subject is both hyperreactive to reinforcement and at the same time unable to properly code or distinguish successive inputs in regard to the meaning of discriminanda. He has found that frontal lesions have a differential effect on escape and avoidance in the cat. His cats had to bar press to avoid shock. After frontal lesions, the avoidance latencies increased but at the same time the cats showed faster than normal escape latencies when the shock was on. This can only be explained by postulating that frontal lesions increase sensitivity and at the same time interfere with the association of discriminanda with their outcomes.

Conflict induced fixated behavior

Another situation in which perseverative behavior is seen is in the frustration-fixation problem of Maier. In this problem rats are placed on a jumping stand from which they must jump within thirty seconds or a shock is applied. The animal's problem is to respond to the window
that will open to give access to the food reward. If the wrong choice is made, the animal bumps against the window and falls to a net below. In simple dark-bright discrimination problems almost all of the subjects are able to solve the problem, but if the animal is first given an insoluble problem in which all the alternatives (right, left; dark, bright) are reinforced randomly fifty percent of the time, most of the animals are unable to solve a subsequent soluble problem. During the insoluble problem the animal develops a stereotype response. This involves either jumping consistently to one side or to the bright or dark window. This response pattern becomes fixated so that in the soluble problem only about 15-20% of the animals ever alter their response pattern.

This behavior is all the more unusual because the deficit is not due to an inability to discriminate (Feldman, 1953). Analysis of jumping latencies has shown that animals jump faster to correct windows than to incorrect windows. Feldman and Green (1967) have specified the conditions under which fixations occur but have not been able to explain why those conditions produce fixated behavior. All that can be said is that conflict leads to behavior stereotypy that persists even when conditions change permitting more adaptive behavior.
STATEMENT OF THE PROBLEM
PURPOSE

Behavior rigidity is characteristic of both conflict and lesion induced perseverative behavior. It is possible that these are similar kinds of behavior. This study investigates the behavior of normal and frontally ablated animals in the Maier paradigm in order to determine the differences and similarities between these two kinds of perseverative behavior.

HYPOTHESES

Predictions concerning the behavior of frontal animals are generated from the hypothesis that frontal lesions produce both hypersensitivity to reinforcement and a loss in the ability to properly assign a reinforcement outcome to a cue when the meaning of the cue has been made ambiguous by changing the conditions of the problem. Predictions of the effects of the insoluble problem are based on previous research which has shown that random punishment results in stereotyped behavior which is not easily altered in subsequent soluble problem conditions. Predictions concerning the behavior of rats in the reversal problem are based on the previous research of Maier and Klee (1948) who showed that rats rarely learn reversal problems on the Lashley Jumping Stand.

Group S: This group only receives the soluble problem. Since none of the response dimensions have previously been punished, the animals should freely learn the problem's solution. In
the reversal problem, this group should be unable to learn the problem's solution because the correct window had previously been associated with 100% punishment.

Group IS: This group receives the soluble problem before the insoluble problem. All the dimensions of response are associated with punishment 50% of the time. The animals should not be able to cope with the insoluble problem and this should result in stereotyped behavior. In the soluble problem about half of the rats should eventually solve the problem. Solutions should occur because the animals have only been given an eight day rather than a 16 day insoluble problem. These animals should be able to adopt the correct response more easily than animals given a longer insoluble problem.

Group LS: This group receives a frontal lesion before the soluble problem. Since each of the stimulus windows is held constant in its relationship to reward and punishment, this group should have little trouble in mastering the correct response. If these lesioned animals are more sensitive to reinforcement, those that solve the problem should switch their responses to the correct window more quickly than the nonlesioned animals. Frontal lesions have been noted to create deficits in avoidance. It is possible that this group as well as the other lesioned groups will show a deficit in their avoidance of the grid shock by exhibiting higher response latencies. However, avoidance of grid
shock in this experiment is based on the ability of the animal to perform a timed response. It is not based on the animal's ability to associate a C. S. with a particular reinforcement outcome. Stamm (1964) has shown that frontal lesions do not interfere with timing responses. If this is so the frontal rats should not show an avoidance deficit. If one result of frontal lesions is increased sensitivity to reinforcement, this group should show the greatest differential latencies between the correct and incorrect windows during the soluble problem.

In reversal this group should be more impaired than the nonlesioned group. Both the previous association of the correct window with punishment and the animal's inability to associate a new reward outcome with a stimulus that had previously been associated with another reinforcement outcome should prevent these animals from solving. In the first instance, the fear of the side opposite to the side on which the rat has stereotyped should prevent the animal from responding on that side. In the second instance, if the rat does respond to the other side, he may not be able to properly interpret or code the relationship between the stimulus cue and it's related reinforcement.

Group ILS: None of the rats of this group should solve the problem. Like normal rats given an insoluble problem, the animals of this group should fall back on a stereotyped response during the insoluble problem. This behavior should
persist throughout the soluble problem. One reason for this would be the fear of the previously abandoned response alternative. The frontal lesion would also make it difficult for the rat to alter its response. Like Pribram's monkeys, these frontal rats should not change their response pattern unless it results in the constant outcome of reinforcement.

Group LIS: This group should behave much like the ILS group. However, if frontal lesions raise frustration thresholds and produce a less fearful animal, this group should be less influenced by the punishment received during the insoluble problem. If this occurs this group should be better able to solve the soluble problem than the ILS group. Finally, both this group and the ILS group should show little differentiation between the correct and the incorrect window. The absence of differentiation would be a result of the frontal animal's inability to associate a specific reinforcement outcome with a specific stimulus.
METHOD
Subjects

Forty-eight Sprague Dawley male albino rats were used in this study. All animals were approximately 60 days old at the start of the experiment. The rats were housed in individual cages and given free access to food and water, except during the first five days of training when they were fed 40 grams of moistened rat chow per day.

Apparatus

A semi-automatically controlled Lashley Jumping stand similar to the one described by Feldman (1948) was used. The essential features of the apparatus are a pair of 6 in. sq. translucent Plexiglas windows which can be independently locked or unlocked and differentially illuminated; a jumping platform with an electric grid placed eight and one-half inches in front of and between the stimulus windows; a net four feet below the platform into which the animals fall if an incorrect response is made; and behind the windows a platform on which the animals land if a correct response is made. An Applegate (model #228) shock source and a Foringer scrambler were used to produce the grid shock. The shock intensity employed was .35 ma. The windows were illuminated with 25 watt bulbs.

Procedure

Training: The training procedure used in this study was similar to that used by Feldman (1953). For three days the
rats were allowed to become accustomed to eating on the stand. Following the familiarization stage, jumping training was begun. In the beginning both windows were left open with the grid one inch away from the platform. The animals were taught to walk from the grid to the platform. On successive days the grid was moved one inch further back from the platform so that at the end of a week the animals were jumping $3\frac{1}{2}$ inches. With jumping established, the windows were gradually closed until the animals were jumping at fully closed windows. All animals were given ten training trials a day during this period. The lighting of the windows was switched randomly between the two sides. Guidance was used to minimize the formation of response preferences. The guidance technique consisted of forcing the animals on even numbered trials to respond to the side opposite to the one they had responded to on the preceding trial.

Preference trials: On the completion of the training stage, the rats were given 40 trials, ten a day, in order to determine their most dominant response tendency before the discrimination problem was given. Both windows were again unlocked during this period of training, but for the first time animals were given a shock if they did not jump within 30 seconds. The food reward was removed at this time. The light was switched randomly between the two windows. Animals were given free choices until they responded three
times in a row to the same side, or to the same light stimulus. If the animal made three similar responses in a row, it was guided on the next trial to the opposite side or to the opposite light stimulus depending on its mode of response.

**Discrimination and discrimination reversal problems:** Five groups, four with ten rats and one with eight rats were established. An attempt was made to place an equal number of dark, bright and position preference animals in each group. Three of the groups were given frontal lesions; three were given an eight day, ten trials a day, insoluble problem; and two of the groups were tested for their ability to learn the reversal of a learned discrimination. The groups were as follows:

1) **LIS group:** This group received the lesion, then the eight day insoluble problem, and then the soluble problem.

2) **ILS group:** This group received the eight day insoluble problem, then the lesion, and was finally tested on the soluble problem.

3) **LS group:** This group received the lesion and was tested on the twenty day soluble problem. The rats in this group that solved the problem were then tested for their ability to learn the reversal of the original soluble problem.

4) **S group:** This group received only the twenty day soluble problem. Those that mastered this problem were tested for the ability to learn the reversal problem.
Two-hundred trials, ten a day, were given in the reversal problem.

5) IS group: This group received the eight day insoluble problem. This was followed by the soluble problem.

Those groups that received the eight day insoluble problem jumped to windows that were randomly associated with reward, window unlocked, or punishment, window locked. All response dimensions; left, right, bright, dark were randomly reinforced 50% of the time. During this problem all rats showed response stereotypes.

After lesioning, after the insoluble problem, or after both, depending on the group, all animals were given a soluble problem in which either the dark or the bright window was correct. The correct window was determined individually for each rat such that, if the rat showed a response preference or a response stereotype to a side or to the bright window, it would be given a dark correct problem. If, however, the animal's predominant response was to go to the dark window, it was given a bright correct problem. Trials continued until a subject made no more than one error in three consecutive days of testing or until the rat had been tested for a total of 200 trials (20 days).

Finally, two groups were given a reversal problem. These groups were required, as were all the groups, to make 29 correct responses out of 30 before they were considered to have reached solution criterion. For those rats
that did solve in the IS and S groups, an additional twenty trials were given after solution and before reversal testing was begun.

**Surgery:** All animals were given nembutal anesthesia (40 mg./kg.) and mounted in a Kopf stereotaxic instrument. Because of a lack of time, the S group did not have an incision made as did the other shamoperated group, group IS. All of the rats except those in the S group had the skin over their skulls retracted and burr holes were drilled through the skull using DeGroot coordinates: A-P ± 10.6, L ± 2, D ± 3.5. All of these rats had the electrode inserted into their brains. However, only the lesioned animals had current applied through the electrode. A C. H. Stoebling Lesion Producing Device (model # 58040) was used to apply the 5.0 ma. D. C. lesioning current. Current was applied for 30 seconds. The burr holes were then filled with gelfoam and the skin was sutured with nylon thread.

**Histology:** When the experiment was over, the subjects were sacrificed with an overdose of nembutal, perfused with saline and 10% formalin and the brains were removed. The brains were imbedded in paraplast and cut into 20 micron thick sections. Five coronal sections through the area of greatest damage were stained for each of the lesioned animals. All sections were stained using the Kluver-Barrera method.
RESULTS
Preference trials

The curves on the left in Figure 1 show that during the four days of preference trials, the jumping latencies for the unguided trials of the IS, LS, LIS, and ILS groups did not differ significantly, $F = 1.56$. The average jumping latency for the four days of trials was 17 sec. for the LS group, 13 seconds for the IS group, 14 seconds for the LIS group, and 15 seconds for the ILS group. The jumping latencies of the $S$ group were not included in this analysis nor are they represented in Figure 1. The rats in the $S$ group were not trained with the 40 animals in the other groups. Hence, the experimenter felt it unfair to compare their jumping latencies. The $S$ group had an average jumping latency, during the unguided preference trials, of 8 seconds.

Insoluble problem trials

Three groups, LIS, ILS and I3, were given the eight day insoluble problem. Their response latencies are shown on the right of Figure 1. An analysis of variance done on the jumping latency data revealed no significant difference between the groups in regard to their over all response latency, $F = 1.28$. The average jumping latency for the eight days of trials for the IS group was 18 seconds; for the LIS group, 20 seconds; and for the ILS group, 20.5 seconds. These groups were also examined to see if the frontal group (LIS) was more or less flexible in its response pattern during the insoluble problem. All groups
Figure 1: Average jumping latencies during the 4 days of preference trials and 8 days of the insoluble problem.

L-S: Lesion, Soluble problem group
I-S: Insoluble problem, soluble problem group
L-I-S: Lesion, insoluble problem, soluble problem group
I-L-S: Insoluble problem, lesion, soluble problem group
were quite similar in terms of the last trial on which a deviant response was made and on the total number of deviant responses made from their final fixation. The ILS group averaged 8.1 deviations from the final fixation, and the last deviation was made on approximately the 44th trial. The IS group averaged 9.1 deviations. The last deviant response occurred, on the average, on the 37th trial. In comparison the lesioned group, LIS, averaged 13.1 deviations from the final response stereotype. The last deviation, on the average, was made on the 39.7th trial.

Histology

The brains of the LS, ILS, and the LIS groups were examined for damage resulting from the lesioning current. Figure 2 shows representative coronal sections of three different rat brains from three different groups; # 34 (ILS), # 25 (LIS), # 3 (LS). Rats 34 and 25 did not solve the soluble problem. Rat 3 solved both the soluble problem and its reversal. In Figure 2 a dorsal view of a representative lesioned brain is also presented, Rat 39 (ILS).

As these figures demonstrate, damage was limited to cortex anterior to the lateral ventricles and dorsal to the olfactory bulbs. In almost all of the animals damage was primarily anterior to DeGroot anterior-posterior coordinate +10.6. Likewise, damage to the frontal tips was limited to the gray matter that was not the most lateral nor the most medial in that area, but rather in
Figure 2: A dorsal view of a typical lesioned rat brain (rat # 39) showing the extent of damage plus representations of three coronal sections taken from the area of greatest destruction: Rat # 3, section # 8, (group LS); Rat # 25, section # 5, (group LIS); Rat # 34, section # 4, (group ILS).
between. All lesions were quite uniform in size. No relationship between behavioral deficit and lesion size could be established.

**Soluble problem (number of solvers)**

Table 1 presents the data on the number of solvers for the five groups. For the IS group, 6 out of 10 rats solved. For group LS, 7 out of 10 solved. For group S, 7 out of 8 rats solved. Only 2 out of ten rats solved in the ILS group while none of the 10 rats in the LIS group solved.

An overall Chi Square analysis of the number of solvers in the five groups revealed a significant difference between the groups in terms of the number of rats that eventually solved the problem. Chi Square = 18.53, P < .01. Further Chi Square analysis comparing the number of solvers in specific groups also showed significance. The number of solvers in the IS and the LS groups was significantly greater than the number in the combined LIS and ILS group, Chi Square = 12.80, P < .01. Likewise, the number of solvers in the IS group was significantly greater than the number of solvers in the combined ILS and LIS group, Chi Square = 11.30, P < .01.

**Soluble problem (percent of correct responses)**

Figure 3 presents in graphic form the percent of correct responses over trials for the five groups. Analysis of this data (Table 2) showed that although there was no group effect on this measure, there were trial effects,
TABLE 1

Comparisons among the numbers of solutions of the soluble problem for all groups.

<table>
<thead>
<tr>
<th>Group:</th>
<th>Solvers</th>
<th>Nonsolvers</th>
</tr>
</thead>
<tbody>
<tr>
<td>S</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>LS</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>IS</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>ILS</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>LIS</td>
<td>0</td>
<td>10</td>
</tr>
</tbody>
</table>

S soluble problem
L frontal lesions
I insoluble problem
Figure 3: Average percent of correct responses during the soluble problem for the LS (lesion, soluble problem), SR (soluble problem), IS (insoluble problem, soluble problem), LIS (lesion, insoluble, soluble problem), and ILS (insoluble problem, lesion, soluble problem) groups.
TABLE 2

Summary of the analysis of variance for the percent of correct responses over trials in the soluble problem.

<table>
<thead>
<tr>
<th>Source of variance</th>
<th>Degrees of freedom</th>
<th>Mean squares</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>939</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Between Ss</td>
<td>46</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A (Groups)</td>
<td>4</td>
<td>16223.00</td>
<td>1.44</td>
</tr>
<tr>
<td>Ss/A</td>
<td>42</td>
<td>11227.00</td>
<td></td>
</tr>
<tr>
<td>Within Ss</td>
<td>893</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T (Trials)</td>
<td>19</td>
<td>981.70</td>
<td>9.03**</td>
</tr>
<tr>
<td>AT</td>
<td>76</td>
<td>185.70</td>
<td>1.70**</td>
</tr>
<tr>
<td>SsT/A</td>
<td>798</td>
<td>108.70</td>
<td></td>
</tr>
</tbody>
</table>

** P < .001 level of significance
F = 9.30, P < .001, and trial by groups effects, F = 1.70, P < .001. Both of these effects are evident in Figure 3. Groups LIS and ILS show almost no increase in their responses to the correct window. All except two of the animals in these two groups continued with their fixation to the incorrect bright window (2 rats) or remained position fixated (16 rats). The group that only received the soluble problem showed very rapid solution of the problem. By the 12th day, seven of the eight rats had solved the problem. One subject in this group remained position fixated. A comparison of the LS and Is groups is interesting. Figure 3 shows that the LS and S groups have similarly shaped acquisition curves; the IS group on the other hand shows a different rate of solution. If the acquisition curves from the solvers in these three groups are examined, Figure 4, shows that the solvers in the S and LS groups do not differ significantly from one another, while the solvers of the IS groups do show a differently shaped learning curve, F = 9.76, P < .001. The curves show that the S and LS solvers solve early in testing while the IS group in contrast shows solution of the problem on later trials.

Soluble problem (differential jumping latencies)

In Figure 5 latencies to the correct and the incorrect windows are presented for the position fixated nonsolvers of the LS, IS, LIS, and ILS groups. Latencies are presented over 10 groups of 20 trials. Analysis of the data (Table 3)
Figure 4: Curves showing the average rate of solution of the soluble problem for the solvers in the S (soluble problem), LS (lesion, soluble problem), and IS (insoluble problem, soluble problem) groups.
revealed significant window, $F = 54.2$, $P < .001$; trials, $F = 2.05$, $P < .01$; and window by trial effects, $F = 7.23$, $P < .001$. This demonstrated that the rats were able to discriminate the correct from the incorrect window even though they did not solve the problem. Furthermore, although latencies in general increased over trials, the difference between jumping latencies to the correct and the incorrect windows became more pronounced as testing continued.

The analysis revealed that there was an overall latency difference between the groups that just missed the acceptable confidence level, $F = 2.64$, $.05 < P < .10$. The fact that group latency differences just missed the appropriate significance level and the fact that a groups by trials interaction did exist, $F = 1.20$, $P < .001$, suggests that groups did differ in regard to their latencies. In general, combining latencies for correct and incorrect responses, the ILS and LIS groups showed a greater overall increase in jumping latencies than did the IS or LS groups.

Of special interest is the fact that there was a significant third order, groups by trials by windows, interaction, $F = 4.71$, $P < .001$. This fact can be clearly seen in Figure 5. Here the graph shows that although all groups show differential latencies to the correct and the incorrect windows, the groups that have been given both
Figure 5: Jumping latencies to the correct and incorrect windows during the soluble problem for the LS, IS, LIS and ILS groups.
### TABLE 3

Summary of the Analysis of Variance of Latencies for Position Fixated Nonsolvers in the LS, IS, LIS, and ILS Groups.

<table>
<thead>
<tr>
<th>Source of Variance</th>
<th>Degrees of Freedom</th>
<th>Mean Squares</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>959</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Between Ss</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A (Groups)</td>
<td>3</td>
<td>3258.00</td>
<td>2.64**</td>
</tr>
<tr>
<td>Ss/A</td>
<td>20</td>
<td>1230.00</td>
<td></td>
</tr>
<tr>
<td>Within Ss</td>
<td>936</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B (windows)</td>
<td>1</td>
<td>11985.00</td>
<td>54.20***</td>
</tr>
<tr>
<td>AB</td>
<td>3</td>
<td>62.33</td>
<td>.280</td>
</tr>
<tr>
<td>S3/A</td>
<td>20</td>
<td>221.50</td>
<td></td>
</tr>
<tr>
<td>T (Trials)</td>
<td>19</td>
<td>52.60</td>
<td>2.05**</td>
</tr>
<tr>
<td>AT</td>
<td>57</td>
<td>30.00</td>
<td>1.20***</td>
</tr>
<tr>
<td>ST/A</td>
<td>380</td>
<td>25.60</td>
<td></td>
</tr>
<tr>
<td>BT</td>
<td>19</td>
<td>99.80</td>
<td>7.23***</td>
</tr>
<tr>
<td>ABT</td>
<td>57</td>
<td>65.10</td>
<td>4.71***</td>
</tr>
<tr>
<td>SBT/A</td>
<td>380</td>
<td>13.80</td>
<td></td>
</tr>
</tbody>
</table>

* .05 < P < .10  ** P < .01  *** P < .001  levels of Significance
the insoluble problem and the lesion show a much less pronounced latency difference between the windows than do the groups that have received only the insoluble problem or the lesion. This effect is most noticeable in the later trials. An analysis of the latencies of the nonsolvers to the correct window on the last day of testing showed an overall significant difference between the groups, \( F = 5.53, P < .05 \). No significant difference could be found between the LS and IS groups on the last day, nor could significant differences be found between the LIS and ILS groups. However, a comparison of the average IS and LS latency to the correct window on the last day revealed a highly significant difference, \( F = 15.60, P < .001 \). This suggests that latencies to the correct window increased when the subject had received both the lesion and the insoluble problem.

**Reversal learning**

Both the solvers in the LS group and the solvers in the S group were tested for their ability to solve a reversal. Thus, after the subjects had reached criterion of solution on the initial problem, the problem was altered and the previously incorrect bright window became the correct window. As expected both groups showed great difficulty in learning this problem. Figure 6 shows the total number of responses made by each group on each of 20 days of testing. Although two out of 7 normal rats were able to solve the reversal, and only one of the frontal subjects solved the reversal,
Figure 6: Total number of correct responses for the frontal (LS) and normal (S) groups during the 20 days of reversal testing.
the lesioned animals made significantly more responses to the correct window, \( F = 6.49, P < .05 \). Therefore, although the frontal rats showed significantly less rigidity in their responses, they failed to show more solutions. The two animals that solved in the S group first switched to a position response before solving. One of these rats was the first to solve the initial soluble problem, while the other one was the last to solve. Of the remaining five rats in this group, one other switched to a position habit while the remaining four continued to respond consistently to the dark window. In the LS group two of the nonsolvers continued to respond consistently to dark, two changed to position habits, one solved, and two rats showed occasional responses to the bright window but responded essentially to dark even though a correct response may have been made. This regression back to the previous response pattern after correct responses had occurred even though it resulted in a greater percentage of punished responses, was characteristic of the frontal group throughout testing. Position or window stereotyped responses often were altered but this did not always result in a consistent change in the rat's response pattern. This was not true of the normal group. Once deviation occurred in this group, new consistent response patterns emerged.
DISCUSSION
The principle findings of this study may be summarized as follows:

A) Both the lesion and the insoluble problem impaired the rat's ability to master a soluble discrimination problem. However, the impairment of the lesioned animals appeared to be different from that of the animals given the insoluble problem. Those animals that solved the soluble problem in the lesioned (LS) group solved at the same rate as the nonlesioned controls (group S). On the other hand, the rats that were given the insoluble problem required more trials to solve the soluble problem. Jumping latencies were similar for the LS and IS groups.

B) No significant differences appeared between the ILS and LIS groups. The placing of the lesion before or after the insoluble problem did not significantly improve or lessen the ability of the rats to solve the soluble problem. Nor were there differences in response latencies in the insoluble and soluble problem between these two groups.

C) A comparison of the performance of the LS and IS groups with the performance of the LIS and ILS groups in the soluble problem revealed that when the insoluble problem was given to the lesioned subjects its effect in reducing solutions was significantly greater than when given to the non-lesioned animals. Furthermore, for the LIS and ILS non-solvers, the latencies to the correct window in the later trials were significantly higher than the latencies of the
IS and LS groups.

D) The S group differed from the LS group in the reversal problem. The lesioned group showed significantly more responses to the correct window. However, this did not create a greater number of solutions in the LS group.

In the LS group three animals out of ten did not solve the soluble problem. This would indicate that frontal lesions created a slight, but not significant, deficit in the ability of the rat to solve the soluble problem. Thompson (1964) previously found that frontal lesions did not impair rats learning a Y-maze discrimination task. This would support the conclusion that the difference between the LS and the S groups, in terms of their ability to solve the problem, is only a chance difference. It is reasonable then, to draw the conclusion that frontal lesions do not lessen the rats' ability to solve a discrimination problem on the Lashley Jumping Stand.

The absence of a difference between the LIS and the ILS groups would indicate that frontal lesions have little effect in altering the emotional effects of punishment. The groups that received the lesion before the insoluble problem showed similar response latencies to the ILS and IS groups in the insoluble problem. During the soluble problem the rats in the LIS group, if anything, were less able to solve the problem than the ILS rats. The conclusion that can be drawn from this is that frontal lesions do not raise
frustration thresholds as Stamm (1964) suggests. Nor do lesions reduce the fear that results from punishment. A reduction of fear would probably have resulted in faster jumping latencies to the incorrect window for all the lesioned groups.

The fact that the insoluble problem was significantly more potent in producing fixated behavior when given to the lesioned animals can be explained in several ways. However, our results most nearly fit the interpretation which holds that frontal animals are impaired in situations in which the outcomes of discriminable cues are not held constant. Frontal rats given only the soluble problem had a constant outcome associated with each of the stimulus windows. Hence, they showed no deficit in learning the task. On the other hand, the frontal groups (LIS and ILS) that were given the insoluble problem did not have this advantage. Each window was associated with random punishment and reward. Neither stimulus window had a constant reward value. Like the frontal monkey, the frontal rat, if it is hypersensitive to reinforcement, would under these conditions, have more difficulty than normals. Furthermore, the variable character of the stimulus windows would be difficult to learn for the frontal animals. This occurs because frontal animals have difficulties in distinguishing cues that successively change their outcome value.

The latencies of the LIS and ILS groups to the correct window were significantly higher than those for the IS and
LS groups. This difference can also be attributed to an inability in the frontal animal to properly associate discriminanda with their outcomes in the soluble problem. Confusion as to the outcome of the two windows would certainly reduce any differential response between the two windows.

The reversal data showed that both the frontal group (LS) and the normal group (S) had difficulty learning the reversal problem. In our experiment, one of the frontal rats and two of the normal animals learned the reversal. In comparison, Maier and Klee (1948) using a similar procedure found that none of their animals solved the reversal although some of the rats switched to a position response and thus reduced punishment from 100% to 50%. What is interesting in our experiment is that the frontal animals showed fewer complete reversals even though they made significantly more responses to the correct window. Normal animals that alter their response pattern and thereby increase the number of reinforced responses, always shift to another response pattern. This was not true of the frontal animals even when they were rewarded for breaking their stereotype response. This result also could be due to an inability in the frontal subjects to associate the window with its reinforcement outcome after the insoluble problem.

None of the groups showed a deficit in the avoidance
of the grid shock. Although some of the rats in the LIS and ILS groups did not avoid the grid shock, this was also true for the IS group. Hence, no conclusion could be drawn as to whether frontal lesions create avoidance deficits in this situation. The absence of avoidance in this case could just as easily developed from the fear of jumping towards a locked window.

Finally, this study directed itself toward determining if there were any similarity between lesion induced and conflict induced perseverative behavior. Two results indicate that these two kinds of behavior are not alike and result from different causes. First, in the soluble problem the LS solvers solved early in the testing period while the solvers of the IS group solved late in the testing period. In the absence of a difference in the rates of learning the correct from the incorrect window among the nonsolvers, it must be concluded that the IS group learns the problem's solution as quickly as the LS group but is less able to make use of what they have learned.

A difference between the lesioned and nonlesioned animals is also evident in the reversal data. The fact that some frontal animals occasionally responded to the correct window without permanently altering their response pattern indicates that frontal rats have a deficit in learning the meaning of the cues. The normal animals, once their ongoing response pattern can be "broken" can learn a new response.
In summary, it would appear that lesioned animals have a different deficit from conflicted animals. However, at the present time it cannot be said that the difficulty of the frontal animal is due to a lesion specific to the frontal area. Lesions in other areas of the brain might also have produced a similar deficit. Further research should be carried out to resolve this question.
Two groups of male rats were tested on the Lashley Jumping Stand to assess the effects of frontal lesions on their ability to solve a simple dark-bright discrimination problem and its reversal. Three other groups of male rats were tested to examine the effects of the lesion on behavior in the Maier paradigm.

Results indicated that perseverative behavior increased when an eight day insoluble problem was given to a frontally lesioned group rather than when given to a non-lesioned group. Likewise, the differential latency between the correct and the incorrect windows decreased when both the lesion and the insoluble problem were given.

The lesioned and the nonlesioned groups differed in the reversal problem in that the lesioned animals showed fewer reversals although they made a significantly greater number of responses to the correct window.

These results were explained in terms of two hypotheses. One hypothesis states that the frontal animal is hypersensitive to reinforcement. Hence, when the frontal animal is rewarded for altering its response it does so but if not immediately rewarded it continues longer with its previous response even when it is unrewarded. The second hypothesis states that frontal animals have a deficit in recent memory. This deficit is evident when the meaning of stimulus cues is altered successively and thereby made ambiguous. Finally it was concluded that the perseveration of frontal rats differs from the perseverative behavior seen
in the normal rat.
REFERENCES


Ruch, T. C., & Shenkin, H. A. The relation of area 13 on the orbital surface of the frontal lobes to hyperactivity and hyperphagia in monkeys. J. of Neurophysiol., 1943, 6, 349-360.


