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THE ROLE OF PERISTRIATE CORTEX IN VISUALLY GUIDED BEHAVIOR IN THE RAT

Stephen Charles Milliser

A Dissertation Submitted to the Faculty of the Graduate School of Loyola University of Chicago in Partial Fulfillment of the Requirements for the Degree of Doctor of Philosophy in Psychology

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Contents

Introduction

Until the 1950's most of the experimental evidence available concerning the neuropsychological processes mediating visually guided behavior dealt with the effects of cortical lesions on the rat's ability to acquire or retain brightness and/or pattern discriminations. Lashley's series, "Mechanism of Vision" (1930-1942), has proven to be the most influential of this research. The problem of elucidating the neural mechanisms underlying vision and visual discrimination has received renewed attention in the last several years. However, some of Lashley's conclusions concerning the neural mediation of vision and visual discrimination continue to be accepted without serious re-examination in relation to more recent evidence; evidence that was not available to Lashley.

Lashley (Lashley, 1939; 1942; Lashley and Frank, 1934) concluded that the striate area (area 17) of the rat's brain was both functionally autonomous and equipotential in mediating the animal's reaction to visual patterns. The purpose of the research reported below was to re-examine Lashley's conclusion concerning the neural mediation of visual habits in the rat.

A limited review of some of the evidence that precipitated this re-examination is presented below.

Lashley (1934) pointed out that the striate area (area 17) of the rat brain had been generally agreed to be that area of the cortex located on the dorsal convexity of the occipital pole and receiving input from the pars dorsalis of the lateral geniculate nucleus of the thalamus, with the anterior-lateral margin of this area receiving binocular input. Accepting Lashley's definition of striate cortex in the rat, the anatomical and neuropsychological evidence presented below indicates that cortex other than striate cortex may be involved in the mediation of visual habits.

Polyak (1957) reported that in the rat fibers from the optic tract enter the lateral geniculate and invade both its ventral and dorsal divisions. Beyond the lateral geniculate, optic tract fibers enter the pulvinar of the thalamus, where they form the zonal stratum. Posterior to the thalamus, optic tract fibers enter the superior colliculus via its brachium or arm. Zeman and Innes (1963) stated that the pars posterior of the lateral nucleus of the thalamus is intimately connected with the primary visual system through both afferent optic tract fibers and a reciprocal projection system with areas of the occipital cortex. These authors (Zeman and Innes, 1963) suggested that the existence of these fiber connections would seem to indicate that the pars posterior is the homologue of the primate pulvinar. Apparently Polyak (1957) used the term pulvinar when referring to the pars posterior. Kappers, Huber,

and Crosby (1960) reported that the pars dorsalis of the lateral geniculate receives fibers from the optic tract, pars posterior, superior colliculus, and other adjoining thalamic nuclear masses. It sends fibers to the striate cortex (area 17), superior colliculus, and surrounding nuclei of the thalamus. The pars ventralis, according to Kappers, Huber, and Crosby (1960), of the lateral geniculate receives fibers from both the optic tract and superior colliculus.

Krieg (1957; 1963) reported that the striate area (area 17) of the rat's cortex receives input from the pars dorsalis of the lateral geniculate and sends projections to area 18 of the peristriate cortex and back to the lateral geniculate. Peristriate cortex (areas 18 and 18a as defined by Krieg) surrounds the striate cortex (area 17). Area 18, the medial portion of the peristriate cortex, exchanges connections with the pars posterior of the lateral nucleus of the thalamus (or pulvinar) and projects to area 18a. Area 18a, the lateral portion of the peristriate cortex, receives input from area 18 and sends fibers to the superior colliculus.

In an early study Lashley (Lashley and Frank, 1934) investigated the effects of various cortical lesions on the retention of a simultaneous discrimination habit in the rat. Extent and location of the lesion were determined both by visual inspection of the surface of the brain and by the pattern of thalamic degeneration. The results of the investigation led

Lashley to conclude that only one sixth of one striate area, provided it included the projection of the binocular field, is not only capable of mediating detailed vision but also of mediating the retention of visual memories. However, Lashley did report that while some subjects with lesions involving only part of both striate areas exhibited near perfect retention of the visual discrimination habit, other subjects with similar lesions required many more trials for relearning than for original learning. With regard to visual function of cortical areas surrounding the striate area (peristriate cortex), Lashley concluded that either this area does not function in the mediation of the habits studied (horizontal vs vertical striations and erect vs inverted equilateral triangles) or, if visual function does depend upon peristriate cortex, peristriate cortex is equipotential for the habits studied. It is the prior conclusion that has been most widely quoted and accepted by others and by Lashley himself.

In a later study Lashley (1939) investigated the function of small, unilateral remnants of the rat's striate cortex. Rats were first trained on three simultaneous discrimination problems (white vs black; horizontal vs vertical striations; and erect vs inverted triangles). On completion of training, half of the subjects were subjected to surgery in which the entire striate area of the right hemisphere and all but the lateral portion of the striate area of the left hemisphere was destroyed. After

recovery from surgery subjects were retrained on the three original discriminations and presented with a series of equivalence tests to determine the capacity of the animals to recognize single properties of these figures apart from the original stimulus configuration. The results showed that several of the operated animals required more retraining on the original discriminations than could reasonably be explained as due to the passage of time alone. Also, all operated subjects that were able to relearn the original discriminations failed a larger proportion of the equivalence tests than did the normals.

More recent research on the rat, and on other rodents, appears to indicate more conclusively than Lashley's findings that peristriate cortex is involved in mediating certain types of visually guided behavior. Fields (1969) reported the results of gross electrical recording from rat occipital cortex during the presentation of various visual patterns. His results indicate that information regarding the size of patterns is received in medial areas of the occipital cortex and that information regarding the shape of the patterns is received in more lateral areas of the occipital cortex. His diagrams indicate that recording was not limited to striate cortex, but that his data includes potentials recorded from peristriate cortex as well as striate cortex. Fields' data indicate a spacial segregation of function within the rat's visual system. Hall and Diamond (1968) and Kaas, Hall, and Diamond (1970) re-

ported finding, in the hedgehog, an area of posterior neocortex outside of the striate area that influences pattern discrimination. They found that lesions of this area, lesions that left striate cortex essentially intact, resulted in severe pattern discrimination deficits. This area receives input from the lateral nucleus of the thalamus and appears to be analogous to areas of peristriate cortex in the rat.

Research involving cats and monkeys clearly indicates that peristriate cortex mediates certain visually guided behaviors in these animals. Meyer (1963) and Wetzel (1969), using cats as subjects, reported that animals with large occipital lobe lesions that had spared part of the striate area still exhibited deficits on pattern discrimination tasks. Mishkin (1966; 1969) reported that lesions of peristriate cortex in the monkey (areas 18 and 19) produced pattern discrimination deficits. Several investigators have revealed visual discrimination deficits in monkeys resulting from lesions of inferior temporal cortex (Inferior temporal cortex lesions usually include most of areas 20 and 21 and parts of areas 18 and 19). Pribram (1969) reported deficits in visual pattern discrimination in monkeys with inferior temporal cortex lesions. Olson, Leary, and Thompson (1967) reported that in several species of primates inferior temporal cortex lesions resulted in size discrimination deficits. Wegner (1968) reported that extensive lesions of areas 18, 19, and 21 left the monkey's capacity to make simple pattern dis-

criminations intact; but these lesions resulted in deficits on more complex discrimination tasks, especially those involving discrimination reversals. Butter (Butter, 1965; 1968; Butter and Doehrman, 1968; Butter and Gekoski, 1966) reported several types of visual discrimination deficits resulting from inferior temporal cortex lesions in the monkey.

Data reported by Hubel and Wiesel (1965; 1968) indicates that both striate and peristriate (areas 18 and 19) cortex receive visual input in both cats and monkeys. In recording from single units (cells) in the occipital cortex of the cat, Hubel and Wiesel (1965) reported finding three projection areas in each hemisphere (visual areas I, II, and III). Histological examination led them to conclude that the three visual areas were almost identical to histological areas 17, 18, and 19. In a later investigation Hubel and Wiesel (1968) reported finding that in the monkey there was less segregation of the three projection areas in terms of histologically defined areas; however, they again reported that cells in both striate and peristriate cortex respond to retinal stimulation.

Thus, both anatomical and neuropsychological data indicate that peristriate cortex is involved in visually guided behavior. If this is true and if it is legitimate to compare rat striate cortex with striate cortex in the monkey and cat, then Lashley's conclusion that striate cortex in the rat is functionally autonomous is incorrect.

In summary, a re-examination of the role of peristriate cortex in visually guided behavior in the rat seemed justified in the light of recent anatomical and neuropsychological data. Three experiments are reported below. They were designed primarily to examine the possibility that peristriate cortex plays a role in visually guided behavior in the rat, not to examine a particular theory concerning that role. Experiment 1 involved the acquisition of simultaneous brightness and pattern discriminations followed by a series of critical trials. Experiment 2 involved the retention of both simultaneous brightness and pattern discriminations. Experiment 3 involved the acquisition of successive brightness and pattern discriminations.

Experiment 1

This experiment involved simultaneous brightness and pattern discriminations followed by a series of critical trials. A simultaneous discrimination is a fairly simple discrimination task; both stimuli are presented at the same time and the subject must choose between them. Learning theorists often refer to this type of task as a recognition task. Acquisition was studied prior to retention for the simple reason that acquisition seemed more primary than retention. The critical trials were added because Lashley (1939) had previously found that rats with bilateral posterior lesions that left unilateral remnants of striate cortex intact were inferior to normals on such tasks.

Subjects

Forty male Long-Evans hooded rats weighing approximately 250 grams at the start of the experiment served as subjects; subjects were maintained in either individual or double cages and allowed free access to food and water.

Apparatus

The apparatus used was an original piece of equipment designed to incorporate the jumping response of the Lashley jumping stand (Lashley, 1930) together with the shock motivation of the Thompson box (Thompson and Bryant, 1955) (refer to

figure 1 page 41). The jumping platform was a rectangular grid (50cm by 15cm) elevated 13cm above the grid floor of the apparatus. The doors that held the stimulus cards were approximately 20cm square, hinged at the bottom, elevated 5cm from the grid floor, and had frosted plexiglass fronts. The roof of the front half of the apparatus was angled to serve to deflect a subject through the door below in case it jumped too high. The stimulus doors were approximately 8cm apart with a triangular wedge between them that served to guide a subject that jumped toward the area between the two doors through the nearest stimulus door. A platform behind the stimulus doors and approximately 2.5cm below the level of the bottom doors served as a landing platform for the subjects. Both the grid floor and the jumping platform of the apparatus were constructed from $\frac{1}{4}$ inch (0.65cm) diameter stainless steel rods and were electrified by a Foringer model 1154 shock source; the polarity of the grid bars was scrambled by a Foringer model 1155 shock scrambler. During training the door holding the negative stimulus card could be closed and locked so that it would not open if a subject jumped against it. The apparatus was located in a semi-darkened room and lighted from above and behind by two 15 watt florescent bulbs. Stimuli consisted of white patterns pasted on black construction paper (refer to figure 2 page 42).

Procedure

The subjects, upon arrival from the supplier, were habituated to our animal room for a minimum of ten days prior to being assigned to experimental conditions. Subsequently, subjects were assigned to one of three groups in a random fashion. Two experimental (operate) groups of 15 subjects each were subjected to bilateral posterior aspirative lesions and the remaining group of ten subjects served as normal controls.

surgical procedure

Approximately 21 days prior to the start of behavioral testing subjects in the two operate groups were subjected to bilateral posterior aspirative lesions. Anesthesia (sodium pentobarbital) was administered intraperitoneally at a dosage level of 50mg/kg. Each subject was placed in a Kopf "U-frame" head holder with rat adaptor. An incision approximately 4cm long was made through the skin on the dorsal surface of the skull. A small trepine hole was made in the parietal bone covering each hemisphere. Using a rongeur, each trepine hole was enlarged so that as much bone as possible between bregma anteriorly, lambda posteriorly, the auditory meatus laterally, and the central sinus medially was removed. The exposed dura was then exised. Using a small glass pipette, neocortex was aspirated. For subjects in one operate group (the total posterior group), an attempt was made to aspirate all exposed neocortex. For subjects in the other operate group (the partial

posterior group), a horseshoe shaped area of neocortex including as much of areas 18 and 18a and excluding as much of area 17 as possible was aspirated. Following aspiration the wound was closed using silk sutures. When an animal recovered from the effects of the anesthesia it was returned to its home cage. All lesioned subjects were housed individually for at least two weeks subsequent to surgery. Subjects in the third group served as normal controls.

behavioral procedure

Each subject was introduced to the apparatus by a method similar to that described by Lashley (1930). The grid floor of the apparatus was electrified throughout each trial; current level was approximately 1ma throughout the study. At the start of each trial the subject was placed near the center of the jumping platform (grid) facing the doors holding the stimulus cards. Approximately 19 seconds later the platform grid was electrified (approximately 0.5ma) so that the subject would jump to escape foot shock. When the subject jumped against the door holding the positive stimulus card, the door opened and the subject escaped from the apparatus. When the subject jumped against the locked door holding the negative stimulus card, the animal fell to the floor of the apparatus and received foot shock until it knocked down the unlocked stimulus door holding the positive stimulus card and escaped from the apparatus. This procedure (making the subject perform the correct response before

ending the trial) is commonly referred to as a correction procedure. A trial ended when the subject, after knocking down the door holding the positive stimulus card, escaped from the apparatus.

On the first day of behavioral testing each subject was presented with a black-white discrimination problem with the white card being the positive stimulus for all subjects. The position of the positive stimulus was determined by a Gellerman (1933) series, modified so that the positive stimulus appeared an equal number of times on the right and left sides during each day's session of twenty trials. Each subject was given twenty trials per day with a 30 second inter-trial-interval until either (a) a criterion of 18 correct out of 20 responses was reached or (b) the total number of trials exceeded 300.

All subjects that reached criterion on the black-white discrimination problem were subsequently presented with a pattern discrimination problem (erect vs inverted equilateral triangles; see figure 2). Procedure and criterion were the same as for the black-white discrimination problem, with the erect triangle being the positive stimulus for all subjects. Subjects that reached criterion on the pattern discrimination problem were then given a series of equivalence tests (figure 2); these tests for equivalence were similar to those described by Lashley (1938).

histological procedure

At the conclusion of the experiment all operate subjects

were sacrificed and perfused with normal saline followed by 10% formalin. The brains were removed and drawings of the lesions were made using diagrams similar to those introduced by Lashley (1930). Subsequently, the brains were frozen and sectioned at 35-40 micra. Approximately every fifth section was mounted and stained with thionin; the stained sections were examined for extent of cortical and subcortical damage as well as for thalamic degeneration.

Results

Of the forty subjects, thirty-five (10 normals, 11 total posteriors, and 14 partial posteriors) finished the experiment. One partial posterior became infected after surgery and was eliminated prior to testing. Three total posteriors died either during surgery or shortly after surgery and one total posterior became infected prior to testing. Histological analysis (see appendix 1 for histological details) indicated that the total posterior lesions were smaller than intended (see procedure section). Lesion diagrams indicate that damage to peristriate areas was incomplete and that, in some animals, the very posterior portion of area 17 appeared intact. Histological analysis also indicated that the partial posterior lesions were not exactly as intended (see procedure section). The partial posterior lesion included portions of area 17, but in all animals in the partial posterior group over 50% of both striate areas remained intact. The partial posterior lesions in areas 18 and 18a

were subtotal. For all operate subjects the amount of subcortical damage was minimal.

Behavioral differences between the three groups were analyzed in terms of both trials-to-criterion and total errors by an extension of the median test to more than two groups (Siegel, 1956). A nonparametric statistical analysis was chosen because the data (especially for the total posterior group) was clearly in violation of the homogeneity of variance assumption underlying traditional parametric statistical tests such as analysis of variance (see appendix 1 for presentation of raw data and detailed presentation of statistical analysis).

On the acquisition of the black-white discrimination no significant differences were found between the three groups in either trials-to-criterion (Chi Square (X^2) = 0.88, df = 2, $p > 0.05$) or total errors (X^2 = 0.88, df = 2, $p > 0.05$). For acquisition of the pattern discrimination significant differences were found between the three groups in both trials-to-criterion (X^2 = 15.74, df = 2, $p < 0.001$) and total errors (X^2 = 17.68, df = 2, $p < 0.001$). Two group comparisons showed that the partial posterior group required more trials to reach criterion (X^2 = 6.17, df = 1, $p < 0.02$) and made more errors (X^2 = 10.97, df = 1, $p < 0.001$) than the normal group and that the total posterior group required more trials to reach criterion (X^2 = 15.57, df = 1, $p < 0.001$) and made more errors (X^2 = 11.93, df = 1, $p < 0.001$) than the partial posterior group. Thus, on

acquisition of the pattern discrimination task the normals required fewer trials and made fewer errors than the partial posterior group and the partial posterior group, in turn, required fewer trials and made fewer errors than the total posterior group.

Because none of the total posterior subjects acquired the pattern discrimination within 300 trials, only the 10 normals and the 10 partial posterior subjects that did acquire the pattern discrimination within 300 trials were given the equivalence tests. No significant differences were found between the two groups on the equivalence tests.

Discussion

The three groups were similar in performance on the black-white discrimination task. These results were consistent with the findings of others (Horel, 1968; Horel, Bettinger, Royce, and Meyer, 1966; Lashley, 1922; 1929; 1935; Meyer, Yutzey, and Meyer, 1966; Thompson, 1969).

The finding that the total posterior group required more trials and made more errors than the partial posterior group on the pattern discrimination task would indicate that Lashley's emphasis on the importance of striate cortex in visual pattern discrimination was justified. However, the finding that the partial posterior group differed significantly from the normals is not consistent with functional autonomy of the striate cortex for visual habits and indicates that peristriate cortex may be

functional in pattern discrimination in the rat. Alternative possibilities include: (1) that the deficit exhibited by the partial posterior group was due to subtotal damage to area 17 present in these animals; (2) that the deficit exhibited by the partial posterior group was the result of a general reduction in potential resulting from the amount of nervous tissue damaged or destroyed. The first possibility would be inconsistent with results reported by Lashley (1939; Lashley and Frank, 1934) and others. The second possibility is inconsistent with other results reported by Lashley (1942).

The similarity between the partial posterior and the normal groups in performance on the equivalence tests was unexpected. Lashley's own data (Lashley, 1939) had indicated the possibility of a difference here and the results of some investigations with higher mammals seemed to indicate that peristriate cortex might function in the transfer from one visual discrimination to another (Wegner, 1968).

The results lend support to the hypothesis that peristriate cortex is involved in the mediation of visually guided behavior in the rat. The results indicate that peristriate cortex, like striate cortex, is more important in the acquisition of a simultaneous pattern discrimination than a simultaneous brightness discrimination. Also, the results indicate that while rats with peristriate lesions are retarded in the acquisition of a simultaneous pattern discrimination habit, they can acquire

such a discrimination given a sufficient number of trials. The fact that Lashley (1939; Lashley and Frank, 1934), using a similar apparatus, found that extensive subtotal damage to area 17 produced little or no deficit on the acquisition of a simultaneous pattern discrimination habit would argue against attributing the deficits exhibited by the partial posterior subjects to the subtotal damage to area 17 present in all of these animals.

Experiment 2

This experiment involved the relearning of simultaneous brightness and pattern discrimination habits. Lashley (1939; 1942; Lashley and Frank, 1934) had investigated both the acquisition and relearning of visual discrimination habits in reaching his conclusion concerning functional visual cortex in the rat. The data from Experiment 1 suggests that some revision of Lashley's conclusion was needed. The research of Mishkin (1966; 1969) and others has implicated cortical areas outside of the striate area in the retention or relearning of visual discrimination habits in higher mammals. Investigation of the role of peristriate cortex in the retention or relearning of simultaneous visual discrimination habits was a logical extension of Experiment 1.

Subjects

Forty male Long-Evans hooded rats weighing approximately 225 grams at the start of the experiment served as subjects; subjects were maintained in either individual or double cages and allowed free access to food and water.

Apparatus

The apparatus used in Experiment 2 has been described in the apparatus section for Experiment 1. The stimuli used in Experiment 2 are illustrated in figure 2.

Procedure

The subjects, upon arrival from the supplier, were habituated to our animal room for a minimum of 10 days prior to being assigned to experimental conditions. Subsequently, subjects were assigned to one of three groups in a random fashion. Two experimental (operate) groups of 15 subjects each were to be subjected to bilateral posterior aspirative lesions and the remaining group of 10 served as normal controls.

surgical procedure

The surgical procedure used in Experiment 2 has been described in the surgical procedure section of Experiment 1.

behavioral procedure

All subjects were introduced to the apparatus, trained to perform a black-white discrimination, and trained to perform a pattern discrimination as described in the behavioral procedure section of Experiment 1. Following the pattern discrimination training, subjects in one of the operate groups were subjected to the partial posterior lesion described in the surgical procedure section; subjects in the second operate group were subjected to the total posterior lesion described in the surgical procedure section. The remaining 10 subjects served as normal controls. Approximately 21 days after completion of the pattern discrimination training all subjects were again trained to perform a black-white discrimination. All subjects reaching criterion on the black-white discrimination (criterion,

as in Experiment 1, was 18 correct out of 20 responses prior to reaching a total of 300 trials) were subsequently trained on the pattern discrimination.

histological procedure

At the conclusion of the behavioral testing the histological procedures described in the histological procedure section of Experiment 1 were followed.

Results

Of the forty subjects, thirty-three (10 normals, 11 total posteriors, and 12 partial posteriors) finished the experiment. All operate subjects that did not finish the experiment died either during surgery or from complications arising from surgery. Histological analysis (see appendix 2 for histological details) indicated that the total posterior lesions were smaller than intended. Lesion diagrams indicated that the damage to peristriate cortex was incomplete and that, in some animals, the very posterior portion of area 17 appeared intact. The histology also indicated that the partial posterior lesions were not as intended. In all animals in the partial posterior group the lesion included portions of area 17 and damage to both areas 18 and 18a was subtotal. For all operate subjects subcortical damage was minimal.

Behavioral differences between the three groups were analyzed in terms of both trials-to-criterion and total errors by an extension of the median test to include more than two

groups (Siegel, 1956) (refer to appendix 2 for presentation of raw data and detailed presentation of the statistical analysis).

On the acquisition of the black-white discrimination habit, prior to surgery, no significant differences were found between the three groups in terms of either trials-to-criterion ($X^2 = 0.82$, df = 2, $p > 0.05$) or total errors ($X^2 = 0.49$, df = 2, $p > 0.05$). On acquisition of the pattern discrimination (erect vs inverted triangles) no significant differences were found in terms of either trials-to-criterion ($X^2 = 0.49$, df = 2, $p > 0.05$) or total errors ($X^2 = 0.82$, df = 2, $p > 0.05$).

On the relearning of the black-white discrimination habit, significant differences were found between the groups using trials-to-criterion ($X^2 = 10.01$, df = 2, $p < 0.01$) but not in terms of total errors ($X^2 = 4.21$, df = 2, $p > 0.05$). Two group comparisons showed that the partial posterior group required more trials than the normal group to reach criterion ($X^2 = 6.60$, df = 1, $p < 0.02$). On the relearning of the pattern discrimination significant differences were found between the three groups in terms of both trials-to-criterion ($X^2 = 21.33$, df = 2, $p < 0.001$) and total errors ($X^2 = 21.00$, df = 2, $p < 0.001$). Two group comparisons showed that the partial posterior group required more trials to reach criterion than the normal group ($X^2 = 6.60$, df = 1, $p < 0.02$) and made more errors than the normal group ($X^2 = 6.60$, df = 1, $p < 0.02$); also, the total posterior group required more trials to reach

criterion than the partial posterior group (X^2 = 19.33, df = 1, $p < 0.001$) and made more errors than the partial posterior group (X^2 = 19.33, df = 1, $p < 0.001$).

Discussion

In general, the results indicate two things: that all three groups were comparable prior to surgery and that both striate and peristriate cortex appear to be involved in mediating the retention of visual discrimination habits.

The fact that the three groups did not differ significantly on the acquisition of either the black-white or pattern discrimination habit is indirect evidence that the groups were comparable prior to surgery.

The fact that the three groups differed significantly on the relearning of both the black-white discrimination habit (using trials-to-criterion)and the pattern discrimination habit (in terms of both trials-to-criterion and total errors), with the lesion groups requiring more trials and making more errors than the normal group, indicates that both the total posterior and partial posterior lesions impaired retention of both discrimination habits. The impairment in the retention of a brightness discrimination habit and a pattern discrimination habit by lesions including both striate areas is well documented (Horel, Bettinger, Royce, and Meyer, 1966; Lashley, 1935; 1942; Lashley and Frank, 1934; Meyer, Yutzey, and Meyer, 1966; Thompson, 1969). It is the impairment of the retention

of a visual discrimination habit by a lesion including much of the peristriate area but sparing more than 50% of the striate cortex that, in the rat, has not been reported in the literature.

The exact nature of the impairment is not clear from the data alone. The partial posterior lesion group was inferior to the normal group in relearning both the black-white discrimination and pattern discrimination habits, which indicates that at least part of the tissue included in the partial posterior lesion is normally involved in the retention of both habits. However, the total posterior lesion group was inferior to the partial posterior lesion group in relearning the pattern discrimination habit, which indicates that the retention deficit was more severe for the total posterior group.

In Experiment 1 the data indicates that peristriate cortex and striate cortex are not necessary for the acquisition of a black-white discrimination habit; whereas both areas seem to be involved in the acquisition of a pattern discrimination habit, with striate cortex being essential for such a discrimination. This would seem to indicate that the deficits exhibited by the total and partial posterior groups in the relearning of the black-white discrimination habit were retention or memory deficits. That is, both striate and peristriate cortex are involved in mediating the learning of the black-white discrimination habit in normal animals, but the learning of this habit can be mediated by structures other than occipital

neocortex (possibly the lateral geniculate nucleus of the thalamus). However, the deficits exhibited in the relearning of the pattern discrimination habit would seem to include more than just a retention deficit. Since subjects with either a total or partial posterior lesion demonstrated, in Experiment 1, a deficit in the acquisition of the pattern discrimination habit, their relearning deficit should include, at least in part, their deficit in the ability to acquire a pattern discrimination habit.

In summary, the data from Experiment 2 increases our confidence in the hypothesis that peristriate cortex is normally involved in mediating visually guided behavior in the rat. Again, this conclusion is valid only if it is reasonable to assume that the deficits exhibited by the partial posterior animals are not due to subtotal damage to area 17; such damage occured in all partial posterior subjects.

Experiment 3

This experiment involved the acquisition of successive brightness and pattern discrimination habits. Previous research (Thompson and Malin, 1961) has indicated that rats with large posterior neocortex lesions are unable to acquire a successive brightness discrimination habit. The fact that animals with similar lesions can acquire a simultaneous brightness discrimination habit (Lashley, 1935; 1942; Lashley and Frank, 1934; Meyer, Yutzey, and Meyer, 1966; Thompson, 1969) would seem to indicate that the successive discrimination habit and the simultaneous discrimination habit require different capacities. Having previously investigated the effects of lesions of peristriate cortex on the acquisition and retention of simultaneous discrimination habits, the research reported below was designed to assess the effects of lesions of peristriate cortex on the acquisition of two successive discrimination habits.

Subjects

Thirty-eight male Long-Evans hooded rats weighing approximately 250 grams at the start of the experiment served as subjects; subjects were maintained in either individual or double cages and allowed free access to food and water.

Apparatus

The apparatus used in Experiment 3 has been described in the apparatus section for Experiment 1; the stimuli used in Experiment 3 are the same as those used in Experiments 1 and 2 (refer to figure 2).

Procedure

The subjects, upon arrival from the supplier, were habituated to our animal room for a period of at least 10 days prior to being assigned to experimetal conditions. Subsequently, subjects were assigned to one of three groups in a random fashion. Two experimental (operate) groups, one of 13 and one of 15 subjects, were to be subjected to bilateral posterior aspirative lesions and the remaining group of 10 subjects served as normal controls.

surgical procedure

The surgical procedure employed in Experiment 3 has been described in the surgical procedure section for Experiment 1.

behavioral procedure

All subjects were introduced to the jumping apparatus as described in the behavioral procedure section for Experiment 1. Subsequently, all subjects were trained on a successive black-white discrimination task. When both white cards were presented, the left stimulus door remained unlocked; when both black cards were presented, the right stimulus door remained unlocked. Criterion was the same as that used in Experiment 1.

All subjects that reached criterion on the black-white discrimination within 300 trials were subsequently trained on a successive pattern discrimination task. When both erect triangle cards were presented, the left stimulus door remained unlocked; when both inverted triangle cards were presented, the right stimulus door remained unlocked. Criterion on the pattern discrimination task was the same as that used in Experiment 1.

histological procedure

At the conclusion of the behavioral testing the histological procedures described in the histological procedure section of Experiment 1 were followed.

Results

Of the thirty-eight subjects, thirty-two (10 normals, 10 total posteriors, and 12 partial posteriors) finished the experiment. All operate subjects that did not finish the experiment died either during surgery or from complications arising from the surgery. Histological analysis (refer to appendix 3 for histological details) indicated that the total posterior lesions were smaller than intended. Lesion diagrams indicated that the damage to the peristriate cortex was incomplete and that, in some animals, the posterior portion of area 17 appeared intact. The histology also indicated that the partial posterior lesions were not as intended. In all animals the partial posterior lesion included portions of area 17 and the damage to peristriate cortex was subtotal. For all operate

subjects, both total posterior subjects and partial posterior subjects, subcortical damage was minimal.

Behavioral differences were analyzed in terms of both trials-to-criterion and total errors by an extension of the median test to include more than two groups (Siegel, 1956) (refer to appendix 3 for presentation of raw data and detailed presentation of the statistical analysis).

On the acquisition of the successive black-white discrimination habit significant differences were found between the three groups in terms of both trials-to-criterion ($X^2 = 20.00$, $df = 2$, $p < 0.001$) and total errors ($X^2 = 20.00$, $df = 2$, $p < 0.001$). Two group comparisons showed that the normal group required fewer trials to reach criterion ($X^2 = 11.73$, $df = 1$, $p < 0.001$) and made fewer errors ($X^2 = 11.73$, $df = 1$, $p < 0.001$) than the partial posterior group; also, the partial posterior group required fewer trials to reach criterion ($X^2 = 11.73$, $df = 1$, $p < 0.001$) and made fewer errors ($X^2 = 6.60$, $df = 1$, $p < 0.02$) than the total posterior group. Only five of the total posterior group and eleven of the partial posterior group reached criterion on the black-white discrimination task within 300 trials and were subsequently presented with the pattern discrimination problem. For the median test an expected frequency of five per cell is strongly recommended (Siegel, 1956), consequently the subjects in the total posterior group (having an expected frequency of only 2.5 per cell) were not included

in the analysis. All five total posterior subjects tested on the pattern discrimination task failed to reach criterion within 300 trials. On acquisition of the successive pattern discrimination the partial posterior subjects required more trials (X^2 = 21.00, df = 1, $p < 0.001$) and made more errors (X^2 = 21.00, df = 1, $p < 0.001$) than the normal control subjects.

Discussion

As in Experiments 1 and 2, the data indicates that at least part of the tissue included in the partial posterior lesion is normally involved in visually guided behavior in the rat. Assuming that the reported deficits were not due to subtotal damage to area 17 (such damage was present in all partial posterior subjects), it appears as if peristriate cortex is normally involved in visually guided behavior in the rat. Previous research (Thompson and Malin, 1961) indicated that large posterior neocortical lesions prevented rats from acquiring a successive brightness discrimination habit. The data reported in Experiment 3 is, by and large, consistent with these findings. Only 5 of the 10 total posterior subjects were able to acquire the black-white discrimination habit within the 300 trials allowed and only one total posterior subject was able to reach criterion on the black-white discrimination task in less than 200 trials. The fact that the partial posterior subjects differed significantly from the normal subjects in terms of both trials-to-criterion and total errors

on the acquisition of the successive black-white discrimination habit seems to indicate that peristriate cortex is involved in the normal acquisition of this task. It is also possible that the deficit found for the partial posterior animals resulted from subtotal damage to area 17.

Because so few of the total posterior subjects (only 5 out of 10) were able to acquire the black-white discrimination habit within 300 trials (a prerequisite for being tested on the pattern discrimination task), the analysis of the pattern discrimination data was limited to a comparison between the normal and partial posterior groups. The fact that the partial posterior group required more trials and made more errors than the normal group on the acquisition of the successive pattern discrimination habit indicates that at least part of the tissue included in the partial posterior lesion is normally involved in the acquisition of a successive pattern discrimination habit. If it is correct to assume that the deficits observed in the partial posterior animals was not the result of subtotal damage to area 17 (such damage was present in all partial posterior animals), then it would appear that peristriate cortex is normally involved in the rat's acquisition of a successive pattern discrimination habit.

Summary Discussion

The results of the three experiments designed to investigate the role of peristriate cortex (areas 18 and 18a as defined by Krieg, 1957; 1963) in visually guided behavior in the rat are reported above. The finding that subjects with total posterior lesions (see surgical procedure section for Experiment 1) showed deficits in the acquisition and retention of simultaneous discrimination habits and in the acquisition of successive discrimination habits appear to lend support to Lashley's (Lashley, 1939; 1942; Lashley and Frank, 1934) conclusion that striate cortex is functionally autonomous in mediating the rat's reaction to visual patterns. Lashley's research had led him to conclude that "the visual system is independent of other parts of the neocortex in the integration of even complex visual associations" (Lashley, 1942, p. 218). According to Lashley (1942) the striate area, or area 17, is the only neocortical component of the visual system. However, the addition of subjects receiving partial posterior lesions (see the surgical procedure section for Experiment 1) to the research reported above indicates that the striate area, or area 17, is not functionally autonomous for visually guided behavior in the rat. The results of the three experiments reported above appear to indicate that either (1) subtotal

damage to area 17 produces rather severe visual deficits in the rat or (2) that peristriate cortex (areas 18 and 18a) is normally involved in the mediation of visually guided behavior in the rat. The first conclusion mentioned above results from the fact that all animals with partial posterior lesions suffered subtotal damage to area 17. Research reported by others appears to indicate that subtotal damage to area 17 does not produce severe visual deficits. Lashley and Frank (1934) reported that one sixth of one striate area was capable of mediating detailed vision and the retention of visual memories. Lubar, Schostal, and Perachio (1967) found that substantial subtotal lesions of area 17 (some lesions included more than 25% of area 17) in the rat resulted in no visual pattern discrimination deficit.

The autonomy of striate cortex, or area 17, in mediating pattern discrimination habits in the rat has been challanged by the research reported by Mize, Wetzel, and Thompson (1970) as well as by the research reported above. Mize, Wetzel, and Thompson (1970) found that rats with extensive bilateral posterior neocortical lesions, lesions including both striate and peristriate cortex, could acquire a simultaneous visual discrimination in which the positive and negative stimuli did not differ in terms of brightness. Their stimuli consisted of white patterns on a black background with each stimulus containing an equal amount of black and white area. The posi-

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tive and negative stimuli differed only in terms of the amount of black-white edge or border.

While Mize, Wetzel, and Thompson (1970) have produced data that indicates that a visual pattern discrimination can be acquired by rats without striate cortex (indicating that striate cortex is not essential for the acquisition of such a discrimination habit), the research reported in the present study indicates that rats with most of the striate area intact are impaired in the acquisition and retention of several visual discrimination habits. It should be pointed out that Mize, Wetzel, and Thompson (1970) did find that rats with bilateral posterior neocortical lesions required more trials than normal rats to acquire a simultaneous pattern discrimination habit.

The function served by peristriate cortex in the rat has not been conclusively demonstrated by the research reported above; the scope of the research is too limited for such a broad conclusion and all lesions of peristriate cortex included subtotal damage to area 17. However, in the light of research reported by others concerning the effects of subtotal lesions of the striate area, the results reported seem to implicate peristriate cortex (areas 18 and 18a) in the mediation of visual memories.

In Experiment 1 the comparisons between the partial posterior and normal groups indicate a deficit in the acquisition of a simultaneous pattern discrimination habit.

Since it is reasonable to assume that learning implies memory, the results are consistent with the proposal that peristriate cortex mediates visual memories. In Experiment 2 the comparisons between the partial posterior and normal groups indicate deficits in the relearning of both simultaneous black-white and pattern discrimination habits. A relearning deficit has been traditionally considered an indication of memory impairment. The results of Experiment 2 are also consistent with the proposal that peristriate cortex mediates visual memories. In Experiment 3 the comparisons between the partial posterior and normal groups indicate a deficit in the acquisition of both successive black-white and pattern discrimination habits. In a successive discrimination task only one of two or more stimuli is presented on any given trial. The organism has two or more responses it can make (in the above study go left or go right) and each response is appropriate (correct) in the presence of a different stimulus. The organism must recall which stimulus is the cue for which response. It is reasonable to assume that visual memory (recalling the stimulus not present) would facilitate the acquisition of such a task. Therefore, within the limits imposed above, the results of Experiment 3 are also consistent with the proposal that peristriate cortex mediates visual memories.

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Figure 1

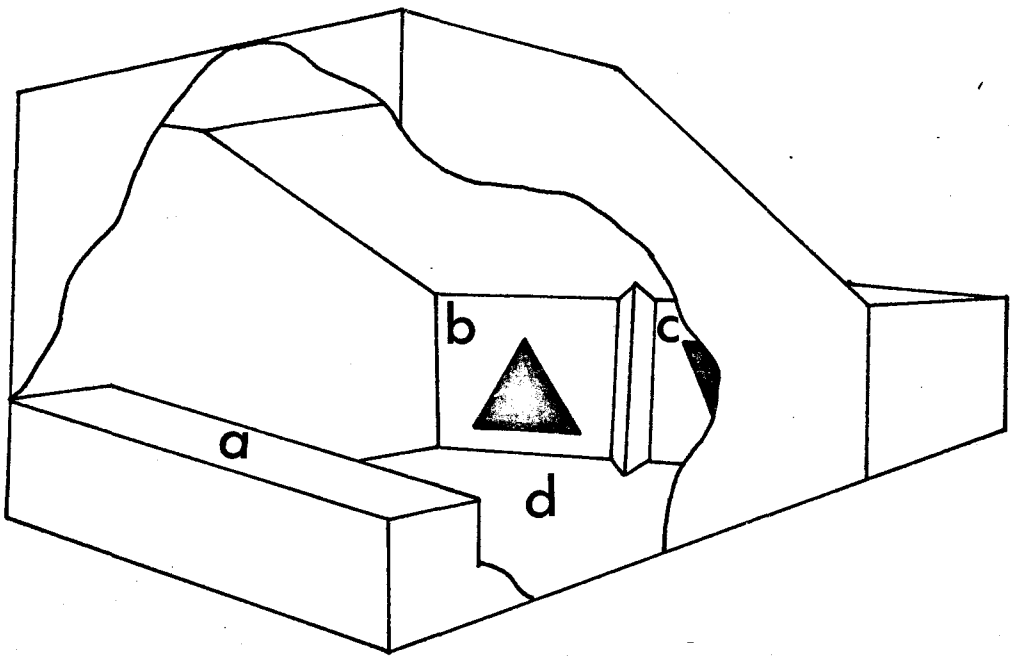


Figure 1: Apparatus used in Experiments 1, 2, and 3. Subject is placed on upper grid (a) and jumps toward stimulus doors (b and c), if door is locked subject falls on lower grid (d).

Figure 2

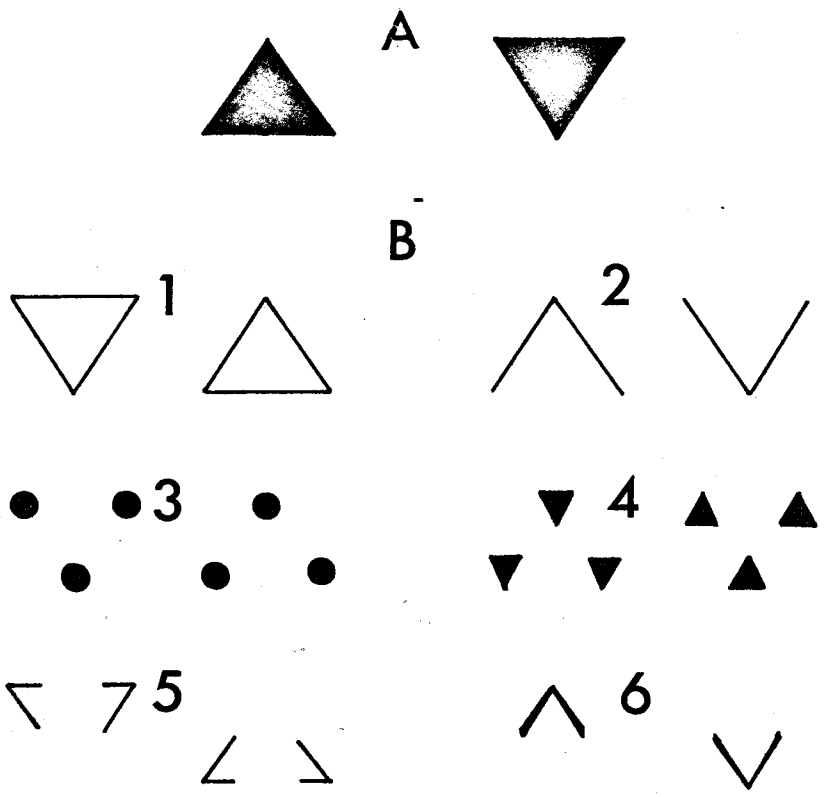


Figure 2: Stimuli used in Experiments 1, 2, and 3. Actual stimuli were white figures on a black background. Triangles (A) were used as pattern stimuli in Experiments 1, 2, and 3. Other patterns (B) were used in equivalence testing in Experiment 1. Scale approximately 1/10.

Appendix 1

Appendix 1 includes the raw data, summary statistics, data analysis, lesion descriptions, and lesion diagrams for the subjects in Experiment 1

Raw Scores

subject	black-white total trials	black-white total erros	pattern total trials	pattern total errors
normal subjects:				
22A	23	9	43	15
7A	8	3	28	9
19A	1	1	117	34
6A	29	16	14	8
16A	12	5	20	8
4A	40	12	0	0
9A	24	8	52	22
1A	35	17	59	33
15A	54	19	84	35
12A	13	5	161	53
total posterior subjects:				
17A	204	91	300	150
14A	74	23	300	135
24A	120	62	300	155
27A	13	6	300	127
39A	75	20	300	146
42A	10	1	300	148
52A	24	9	300	138
56A	4	2	300	159
58A	8	5	300	145
72A	0	0	300	116
99B	29	10	300	162
partial posterior subjects:				
25A	100	41	268	118
26A	242	84	279	142
35A	42	15	94	40
40A	3	1	38	16
48A	0	0	56	24
49A	0	0	214	88

51A	0	0	206	69
53A	22	6	42	12
55A	2	1	300	164
2B	0	0	128	42
57A	15	7	95	38
3B	28	12	158	40
69A	19	3	300	137
70A	32	15	88	36

Descriptive Statistics

group	black-white total trials	black-white total errors	pattern total trials	pattern total errors
normal control	M = 23.9 SD = 15.3	M = 9.5 SD = 5.9	M = 57.8 SD = 47.6	M = 21.7 SD = 15.8
total posterior	M = 51.0 SD = 60.5	M = 20.8 SD = 27.9	M = 300 SD = 0.0	M = 143.7 SD = 13.5
partial posterior	M = 36.1 SD = 62.7	M = 13.2 SD = 22.3	M = 161.8 SD = 94.4	M = 69.0 SD = 49.5

Statistical Analysis

Because the data violates the homogeneity of variance assumption underlying classical parametric statistical tests such as analysis of variance, the median test (Siegel, 1956) was used to determine statistically significant (alpha of 0.05 or less) differences.

black-white total trials

	normal	total posterior	partial posterior
number of subjects above median	6	6	6
number of subjects below median	4	5	8

$X^2 = 0.88$, df = 2, $p > 0.05$

black-white total errors

	normal	total posterior	partial posterior
number of subjects above median	6	6	6
number of subjects below median	4	5	8

$X^2 = 0.88$, df = 2, $p > 0.05$

pattern total trials

	normal	total posterior	partial posterior
number of subjects above median	2	11	5
number of subjects below median	8	0	9

$X^2 = 15.74$, df = 2, $p < 0.001$

pattern total trials: two group comparisons

normal - partial posterior

	normal	partial posterior
number of subjects above median	2	10
number of subjects below median	8	4

$X^2 = 6.17$, df = 1, $p < 0.02$

partial posterior - total posterior

	partial posterior	total posterior
number of subjects above median	2	11
number of subjects below median	12	0

$X^2 = 15.57$, df = 1, $p < 0.001$

pattern total errors

	normal	total posterior	partial posterior
number of subjects above median	1	11	6
number of subjects below median	9	0	8

$X^2 = 17.68$, df = 2, $p < 0.001$

pattern total errors: two group comparisons

normal - partial posterior

	normal	partial posterior
number of subjects above median	1	11
number of subjects below median	9	3

$X^2 = 10.97$, df = 1, $p < 0.001$

partial posterior - total posterior

	partial posterior	total posterior
number of subjects above median	3	10
number of subjects below median	11	1

$X^2 = 11.93$, df = 1, $p < 0.001$

Histology

This section included both a written and pictorial description of the histological analysis of each operate subject.

17A: In both hemispheres the lesion included all but the most posterior extent of area 17. Area 18 was damaged only in its lateral most area. The lateral area of area 18a was extensively damaged in both hemispheres. The posterior portion of area 18a received minimal damage in both hemispheres. Subcortical damage

was limited to the dorsal portion of the right hippocampus. Degeneration was evident throughout the pars dorsalis of the lateral geniculate. There was also degeneration in the dorsal and lateral areas of the pars posterior in both hemispheres.

14A: In both hemispheres a small portion of the posterior area of area 17 remained intact. Damage to the lateral portion of both area 18 and 18a was extensive in both hemispheres. The posterior portion of area 18a received only minimal damage in both hemispheres. In both hemispheres lesion depth was never beyond the corpus callosum. In both hemispheres moderate to heavy degeneration was evident in both the pars dorsalis and pars posterior.

24A: In the right hemisphere the lesion included almost all of area 17. In the left hemisphere the lesion did not extend far enough posterior to include all of area 17. In both hemispheres damage to the lateral portions of areas 18 and 18a was extensive. The posterior portion of area 18a received only minimal damage. Lesion depth stopped at the corpus callosum in both hemispheres. Degeneration was extensive in the left pars dorsalis and pars posterior. Much lighter (milder) degeneration was present in the right pars dorsalis and pars posterior.

27A: In both hemispheres the lesion included all but some very posterior remnants of area 17. Damage to the lateral portion of both area 18 and 18a was extensive. The posterior extent of area 18a remained essentially intact. Subcortical damage was limited to the dorsal tip of the right hippocampus. Degeneration was light. The heaviest degeneration was found in the central portion of the pars dorsalis in each hemisphere. Little evidence of degeneration was found in either pars posterior.

39A: In each hemisphere a small portion of the posterior area of area 17 remained intact. This intact portion of area 17 was slightly larger in the right hemisphere. Damage to the lateral area of both area 18 and 18a was fairly extensive. Damage to the lateral portion of area 18a was greater in the right hemisphere. Subcortical damage was limited to the very dorsal portion of the right hippocampus. Degeneration was heavy in the pars dorsalis and light in the pars posterior in each hemisphere.

42A: The lesion in each hemisphere was small. In both hemispheres a fairly significant portion of the posterior part of area 17 remained undamaged. Damage to the lateral portion of area 18 was extensive in both hemispheres. Damage to the lateral portion of area 18a was quite extensive in the left hemisphere but less extensive in the right hemisphere. The posterior portion of area 18a was minimally damaged in each hemisphere.

Subcortical damage was nonexistent. Degeneration was present in most of the pars dorsalis of each hemisphere and in the dorsal-lateral area of the pars posterior of each hemisphere.

52A: In both hemispheres a small portion of the posterior part of area 17 remained undamaged; the undamaged part of area 17 was larger in the left hemisphere. Damage to the lateral extent of both area 18 and 18a was extensive in both hemispheres. The posterior portion of area 18a was minimally damaged. Subcortical damage due to the surgery was nonexistent; however, a small tumor was found in the posterior-lateral area of the right hemisphere. The tumor was less than 1mm in diameter and could be seen in only two sections. Degeneration was evident throughout the pars dorsalis in each hemisphere and in the dorsal-lateral portion of the pars posterior in each hemisphere.

56A: In both hemispheres a small part of the posterior portion of area 17 remained intact. Damage to the lateral extent of area 18 and 18a was extensive in both hemispheres. The posterior portion of area 18a was only minimally damaged in each hemisphere. Subcortical damage was nonexistent. Degeneration was evident throughout both the right and left pars dorsalis. Little evidence of degeneration was found in the pars posterior of either hemisphere.

58A: In each hemisphere a small portion of the posterior part of area 17 remained intact. Damage to the lateral portion of area 18 and 18a was extensive in each hemisphere. Damage to the posterior portion of area 18a was minimal in both hemispheres. Subcortical damage was nonexistent. Degeneration was present throughout the pars dorsalis of each hemisphere. Degeneration was evident in the dorsal-lateral area of the pars posterior of each hemisphere.

72A: In both hemispheres a small part of the posterior portion of area 17 remained intact. Damage to the lateral portion of area 18 and 18a was extensive in both hemispheres. The posterior portion of area 18a was minimally damaged in each hemisphere. Subcortical damage was limited to the dorsal tip of the right hippocampus. Degeneration was present throughout the pars dorsalis of each hemisphere. Degeneration was also present in the dorsal-lateral area of the pars posterior of each hemisphere.

99B: In both hemispheres the lesion appeared to include all of area 17. Damage to the lateral portion of area 18 and 18a was extensive in both hemispheres. The posterior portion of area 18a remained essentially intact in each hemisphere. Subcortical

damage was nonexistent. Problems with the staining of the tissue made it impossible to accurately determine the extent and location of thalamic degeneration.

25A: In both hemispheres all but the posterior portion of area 17 remained essentially intact. In both hemispheres only the medial portion of area 18 remained intact. In both hemispheres only the posterior portion of area 18a remained intact. Subcortical damage was nonexistent. Degeneration was evident in the medial area of the pars dorsalis. Degeneration was present in the dorsal-lateral area of the pars posterior in each hemisphere.

26A: In both hemispheres all but the posterior portion of area 17 remained intact. The damage to the posterior area of area 17 was more extensive in the left hemisphere. In both hemispheres the medial portion of area 18 escaped with only minimal damage. In both hemispheres all but the most posterior portion of area 18a was included in the lesion. Subcortical damage was nonexistent. Degeneration of the pars dorsalis was evident only in the medial portion in both hemispheres. Degeneration was present in the dorsal-lateral area of the pars posterior in both hemispheres.

35A: All but the posterior portion of area 17 remained intact in both hemispheres. In both hemispheres damage to area 18 was limited to its lateral portion. In both hemispheres the lesion included all but the posterior portion of area 18a. Subcortical damage was nonexistent. Degeneration of the pars dorsalis was present in the medial portion in both hemispheres. Degeneration was present in the dorsal-lateral area of the pars posterior in both hemispheres.

40A: All but the posterior portion of area 17 remained intact in both hemispheres. Damage was limited to the lateral portion of area 18 in both hemispheres. Damage to area 18 was more extensive in the left hemisphere. In both hemispheres the lesion included all but the posterior portion of area 18a. Subcortical damage was nonexistent. Degeneration was present only in the medial portion of the pars dorsalis in both hemispheres. Degeneration was present in the dorsal-lateral portion of the pars posterior in both hemispheres.

48A: All but the posterior portion of area 17 remained intact in both hemispheres. Damage was limited to the lateral portion of area 18 in both hemispheres. The lesion included all but the posterior portion of area 18a in both hemispheres. There was no damage to structures lieing below the corpus callosum.

Degeneration was light and limited to the dorsal-lateral portion of the pars posterior and the dorsal portion of the pars dorsalis in each hemisphere.

49A: In both hemispheres only the posterior portion of area 17 was included in the lesion. Damage was shallow and limited to the lateral portion of area 18 in both hemispheres. The lesion included all but the posterior portion of area 18a in the left hemisphere. In the right hemisphere the lesion included all but the posterior and extreme lateral portions of area 18a. Subcortical damage was nonexistent. Degeneration was present in the dorsal-medial portion of the pars dorsalis in each hemisphere. Degeneration was present in the dorsal-lateral portion of the pars posterior in both hemispheres.

51A: In both hemispheres only the posterior portion of area 17 was included in the lesion. The lesion was limited to the lateral portion of area 18 in both hemispheres. The lesion included all but the posterior portion of area 18a in both hemispheres. There was no damage to structures lieing below the corpus callosum. Degeneration was limited to the dorsal-lateral area of the pars posterior and a small area of the dorsal-medial portion of the pars dorsalis in both hemispheres.

53A: In both hemispheres only the posterior portion of area 17 was included in the lesion. The lesion was limited to the lateral portion of area 18 in both hemispheres. The lesion included all but the posterior portion of area 18a in both hemispheres. There was no subcortical damage in either hemisphere. In the left hemisphere degeneration was found in the medial-lateral area of the pars posterior and a small area in the medial portion of the pars dorsalis. In the right hemisphere degeneration was present in the dorsal-lateral area of the pars posterior and a small area in the dorsal-medial portion of the pars dorsalis.

55A: In both hemispheres all but the posterior portion of area 17 remained intact. The lesion was limited to the lateral portion of area 18 in both hemispheres. Damage to area 18 was more extensive in the right hemisphere. The lesion included all but the posterior portion of area 18a in both hemispheres. There was no damage to structures lieing below the corpus callosum. In both hemispheres degeneration was limited to the medial-lateral area of the pars posterior and a small area in the medial portion of the pars dorsalis.

2B: In both hemispheres all but the posterior portion of area 17 remained intact. The lesion damage was limited to the lateral portion of area 18 in both hemispheres. The lesion included

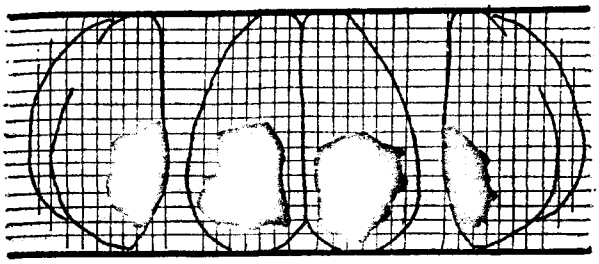
all but the posterior portion of area 18a in both hemispheres. There was no subcortical damage in either hemisphere. Degeneration in both hemispheres was limited to the dorsal-lateral area of the pars posterior and a small area in the medial portion of the pars dorsalis.

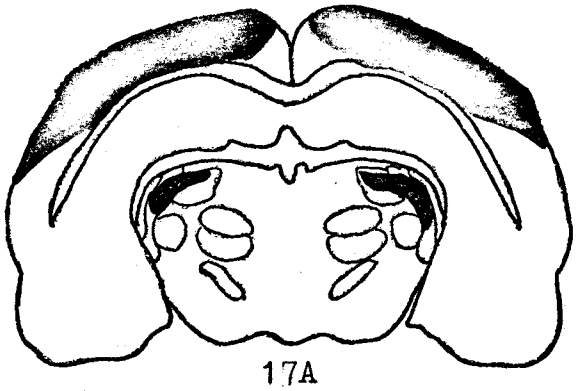
57A: In both hemispheres only the posterior portion of area 17 was included in the lesion. The lesion was limited to the lateral portion of area 18 in both hemispheres. In the left hemisphere the lesion included all but the posterior and extreme lateral portion of area 18a. In the right hemisphere the lesion included all but the posterior portion of area 18a. There was no subcortical damage in either hemisphere. In both hemispheres degeneration was limited to the lateral portion of the pars posterior and a small area in the medial portion of the pars dorsalis.

3B: In both hemispheres nearly all of area 17 remained intact. The lesion was limited to the lateral portion of area 18 in both hemispheres. The lesion included all but the posterior portion of area 18a in both hemispheres. Subcortical damage was nonexistent. In both hemispheres degeneration was limited to the dorsal-lateral area of the pars posterior and a small area in the medial portion of the pars dorsalis.

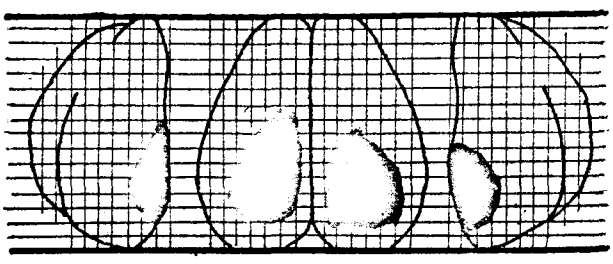
69A: In both hemispheres only the posterior portion of area 17 was included in the lesion. The lesion was limited to the lateral portion of area 18 in both hemispheres. The lesion included all but the posterior portion of area 18a in both hemispheres. Subcortical damage was nonexistent. In both hemispheres degeneration was found in the dorsal-lateral area of the pars posterior and a small area in the medial portion of the pars dorsalis.

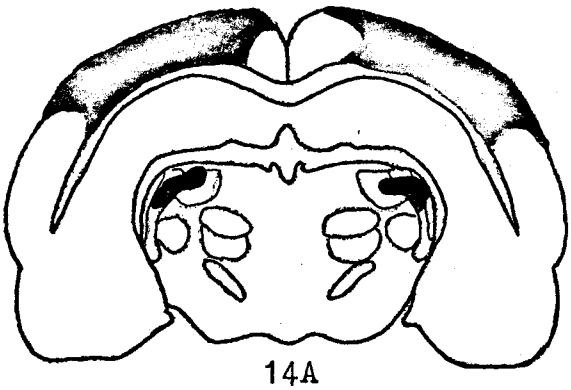
70A: In both hemispheres only the posterior portion of area 17 was included in the lesion. The lesion was limited to the lateral portion of area 18 in both hemispheres. The lesion included all but the posterior portion of area 18a in both hemispheres. There was no damage to structures lieing below the corpus callosum in either hemisphere. In both hemispheres degeneration was limited to the medial area of the pars posterior and a small area in the medial-lateral portion of the pars dorsalis.





17A





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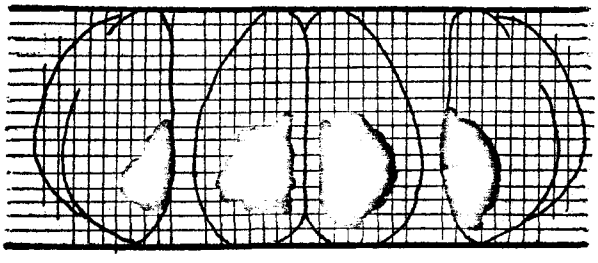

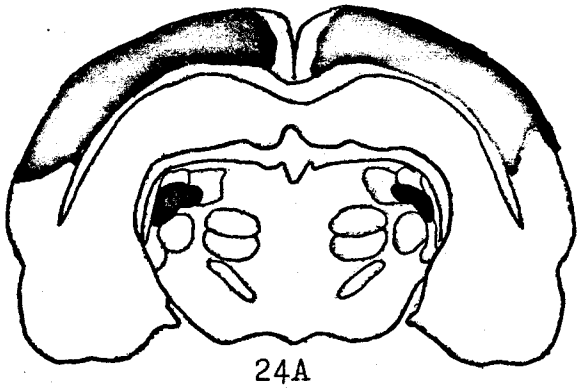

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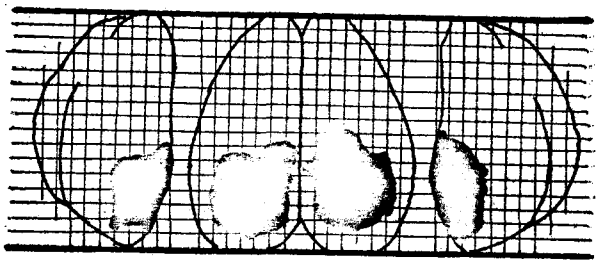

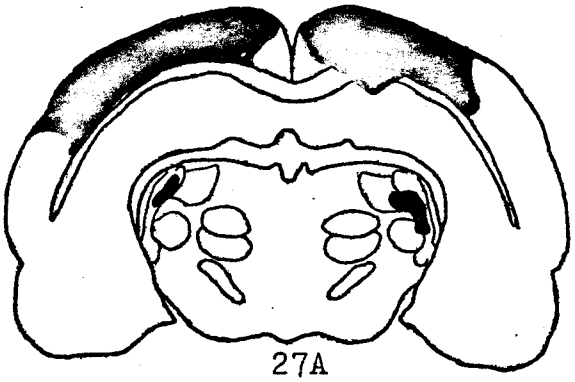

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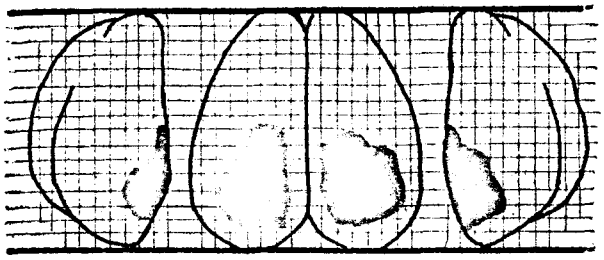

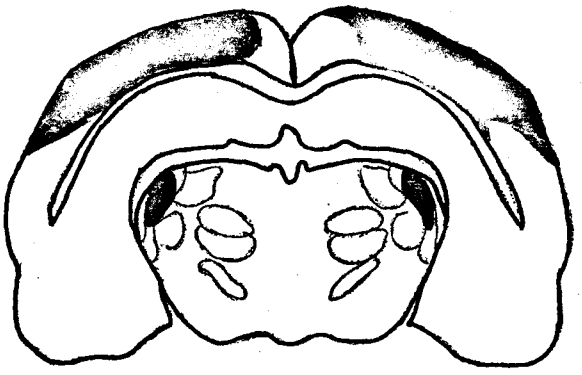

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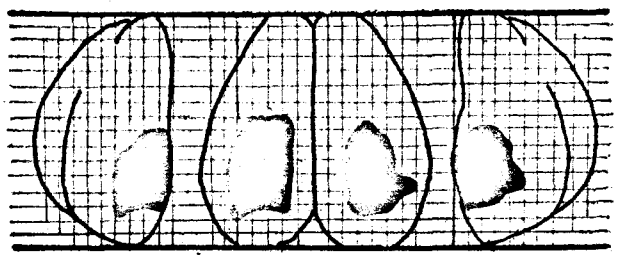

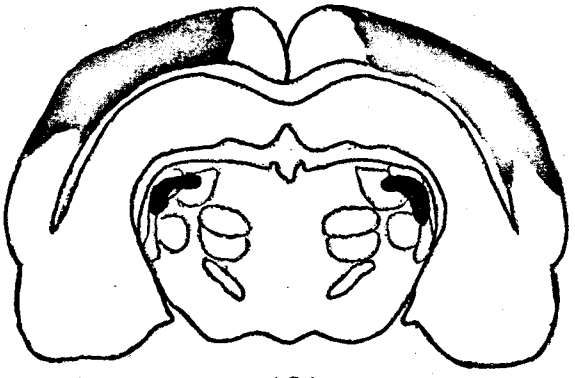

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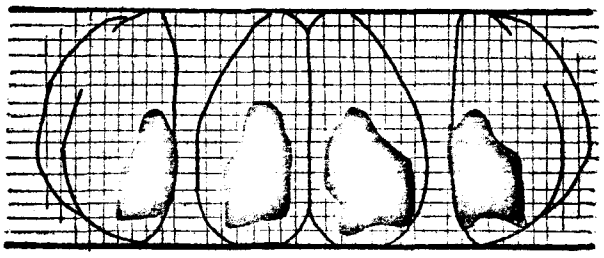

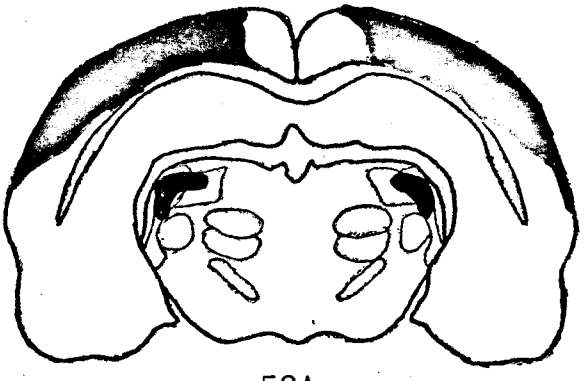

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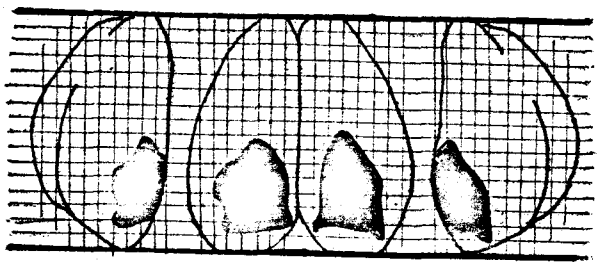

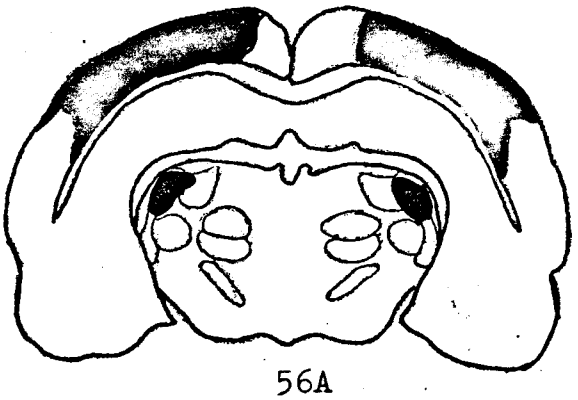

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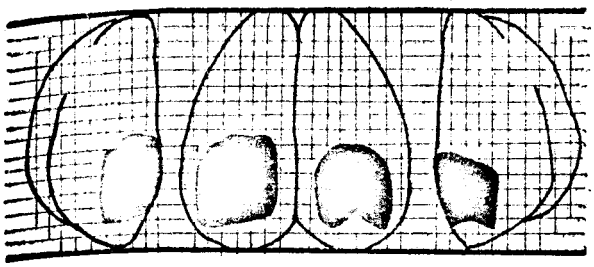

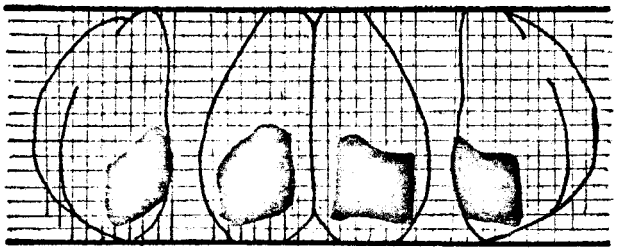

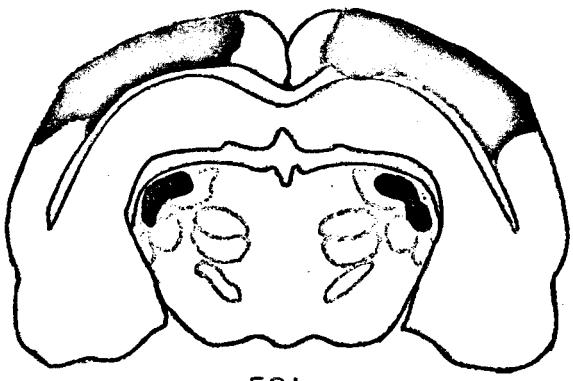

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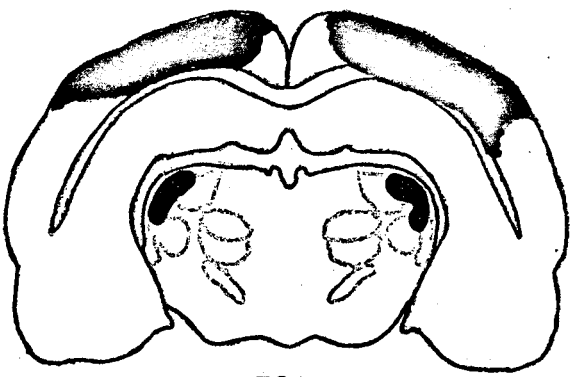

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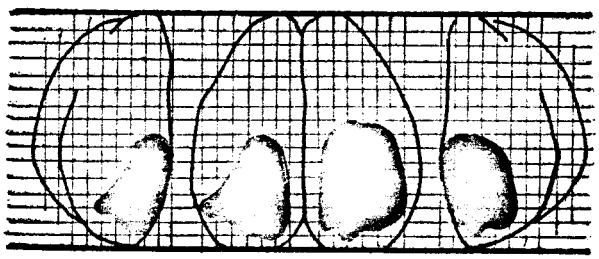

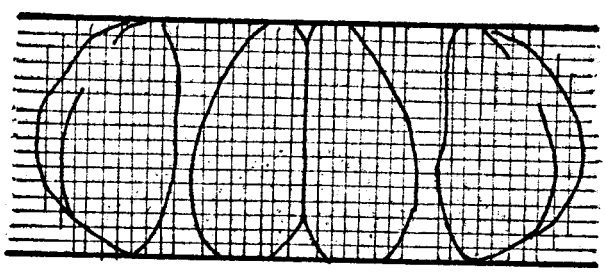

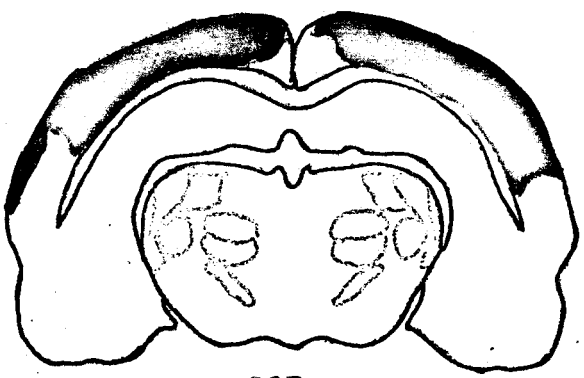

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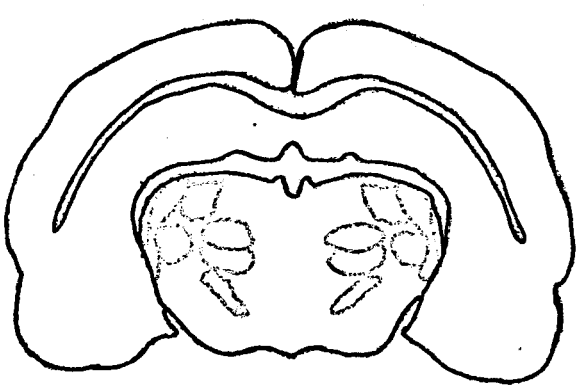

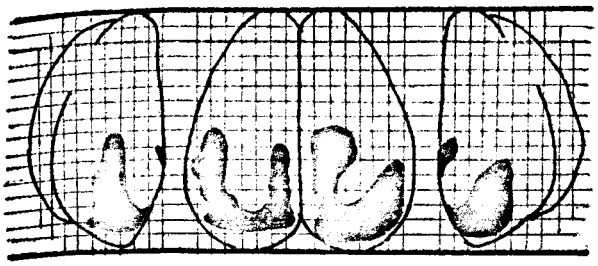

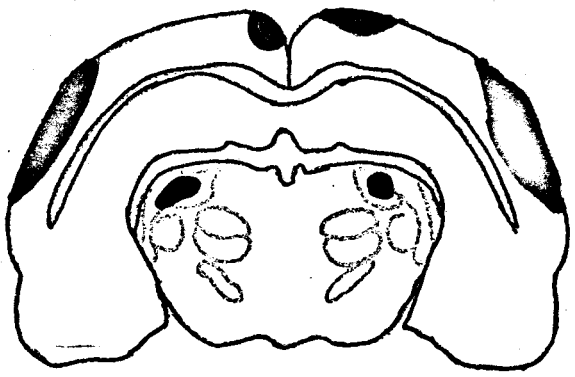

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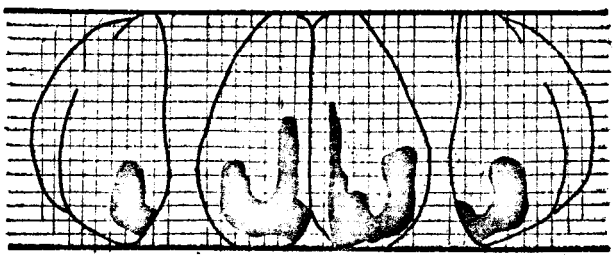

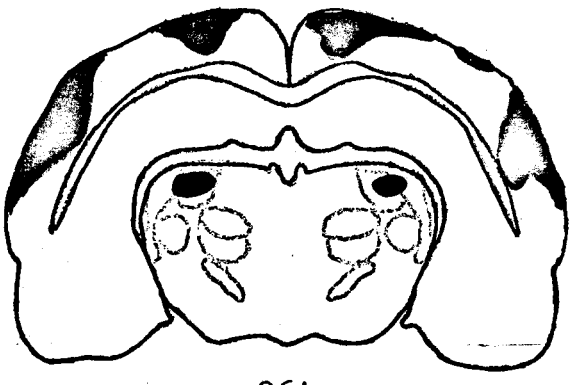

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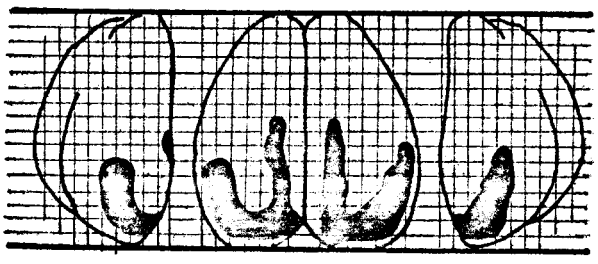

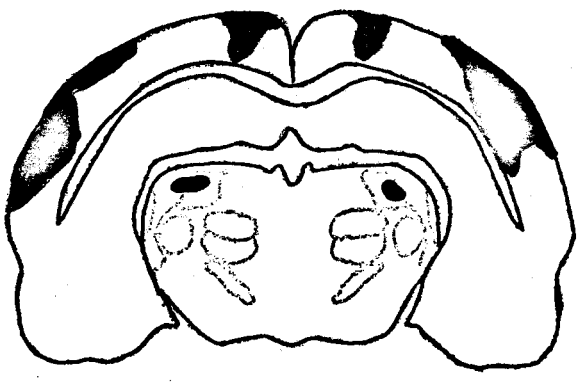

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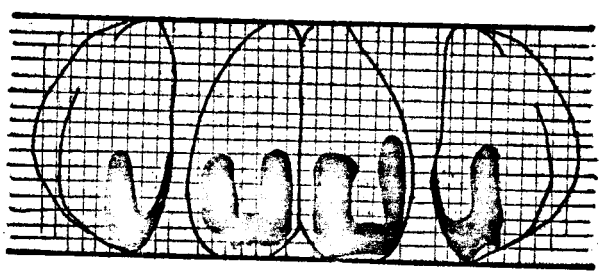

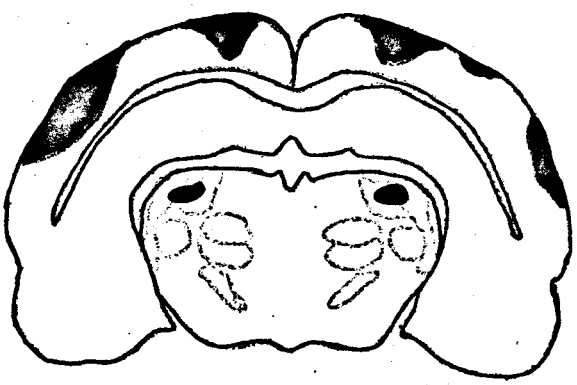

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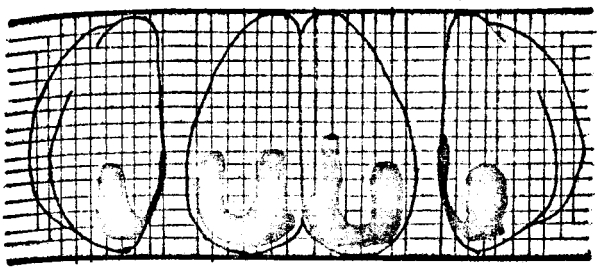

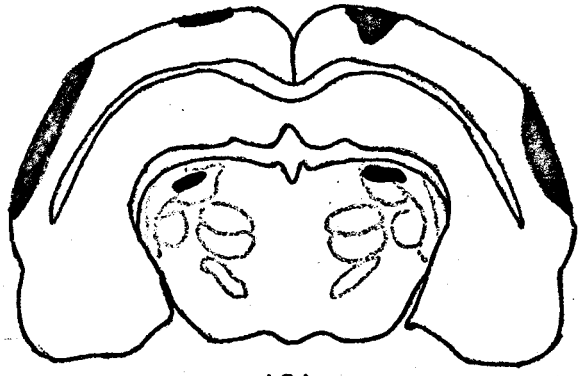

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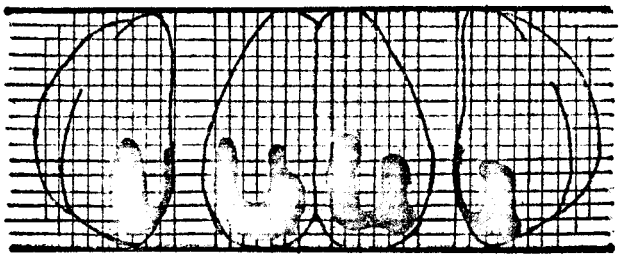

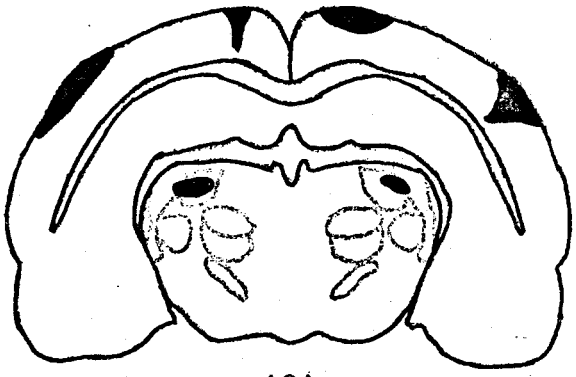

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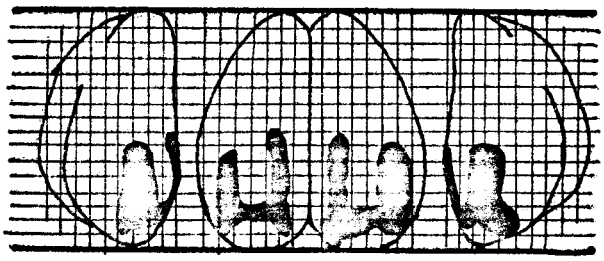

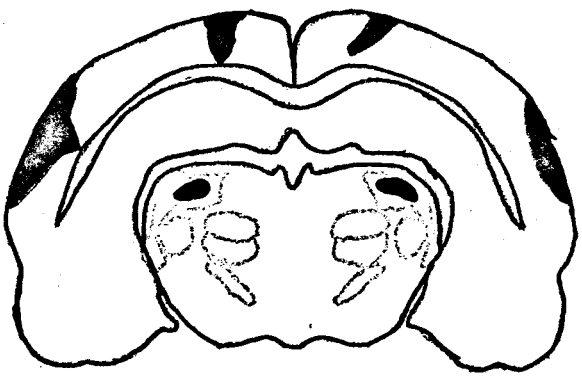

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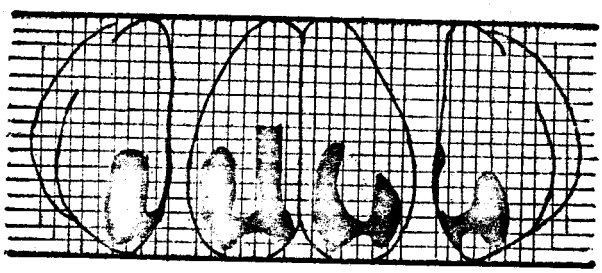

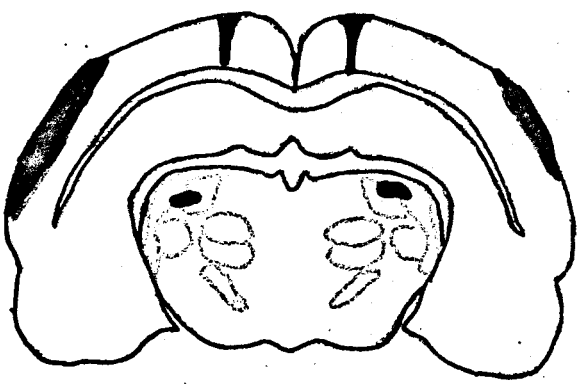

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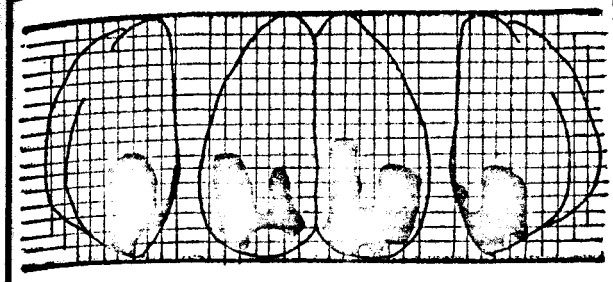

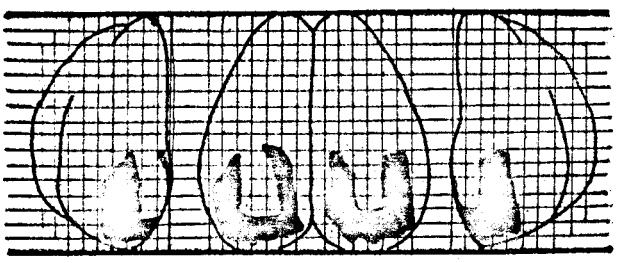

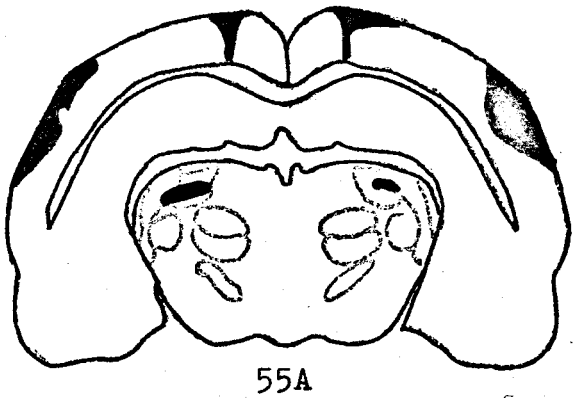

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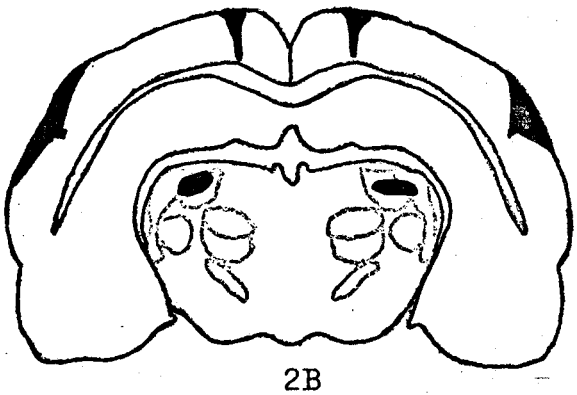

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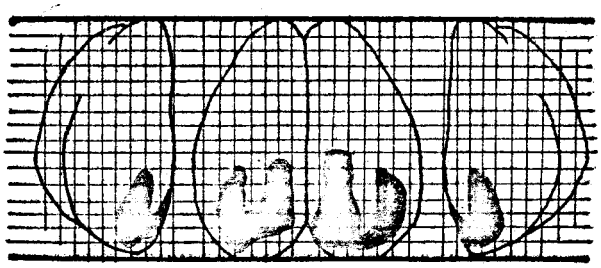

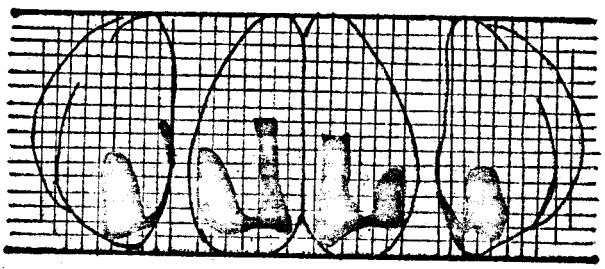

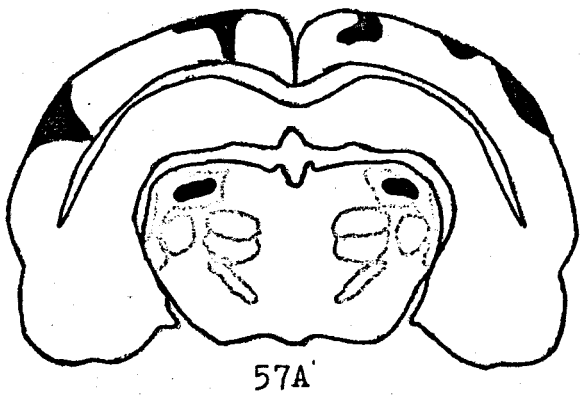

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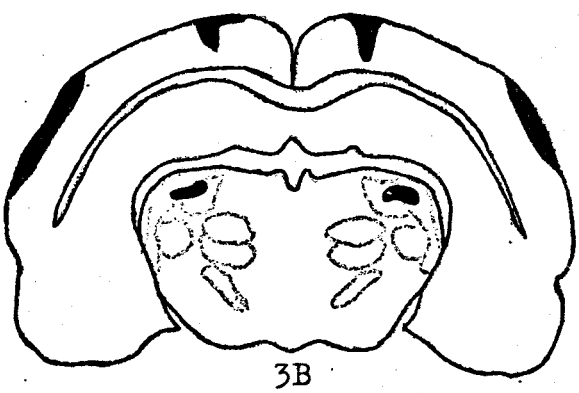

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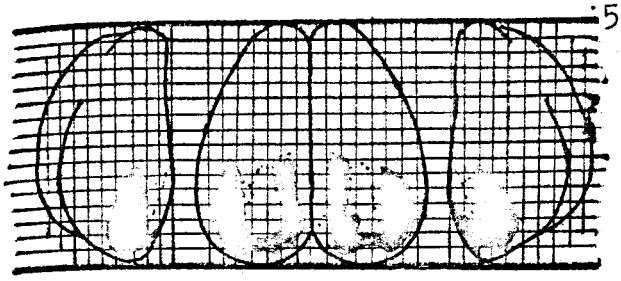

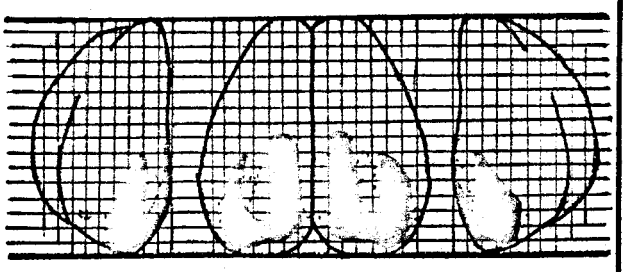

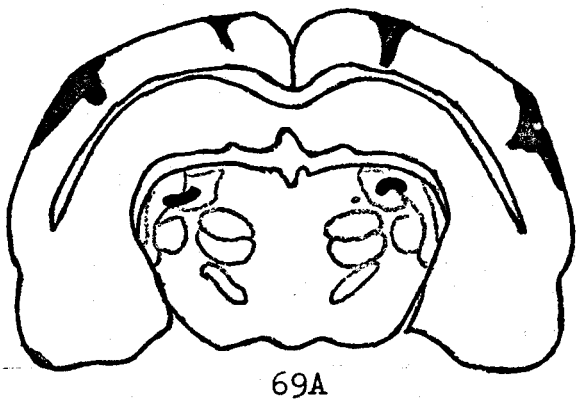

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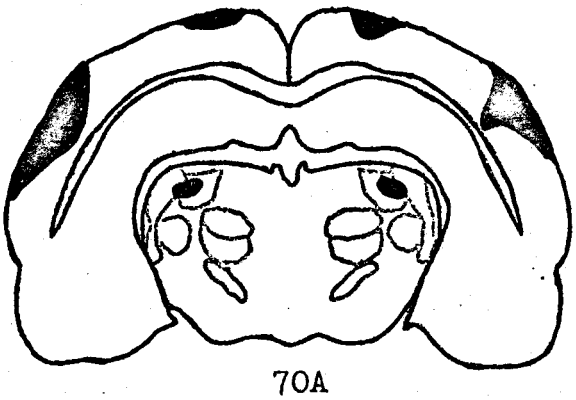

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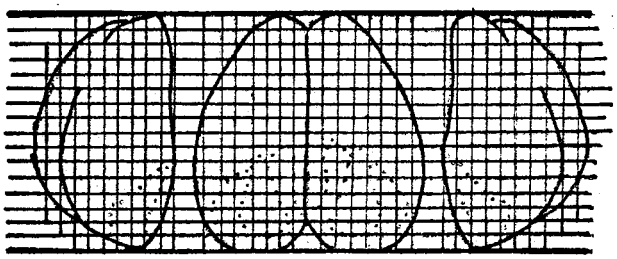
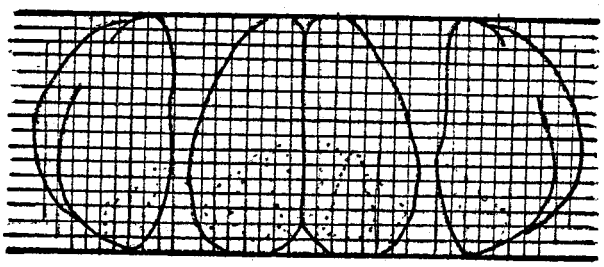
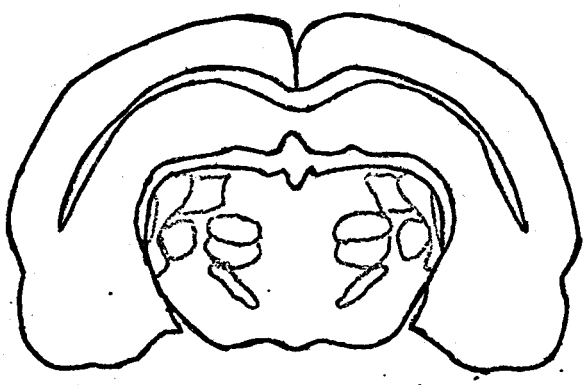
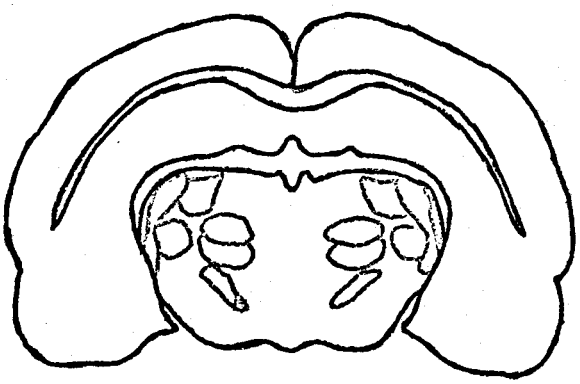








Appendix 2

Appendix 2 includes the raw data, summary statistics, statistical analysis, lesion descriptions, and lesion diagrams for the subjects in Experiment 2.

Raw Scores

Pre-treatment Data

subject	black-white total trials	black-white total errors	pattern total trials	pattern total errors
normal subjects:				
29A	0	0	59	29
30A	0	0	85	34
21A	4	2	102	36
28A	0	0	69	32
34A	15	7	82	33
44A	0	0	78	27
46A	12	4	45	12
54A	19	11	73	36
37A	22	8	90	27
59A	0	0	112	43
total posterior subjects:				
62A	1	1	66	31
71A	0	0	54	21
98A	18	5	75	32
78A	42	16	84	38
82A	32	17	65	23
83A	12	5	42	14
84A	6	3	51	30
87A	0	0	104	41
88A	12	4	93	39
89A	7	3	67	21
2C	25	12	101	38

partial posterior subjects:

64A	19	7	67	31
65A	21	6	32	9
8B	0	0	75	30
74A	2	1	43	15
75A	0	0	126	54
9B	9	2	2	1
77A	12	7	75	36
81A	18	4	64	28
85A	0	0	113	62
86A	13	6	84	43
90A	24	14	65	30
91A	0	0	84	33

Post-treatment Data

subject	black-white total trials	black-white total errors	pattern total trials	pattern total errors
normal subjects:				
29A	0	0	0	0
30A	18	10	17	7
21A	0	0	0	0
28A	0	0	26	8
34A	0	0	0	0
44A	14	6	19	8
46A	4	2	32	14
54A	0	0	11	10
37A	10	7	0	0
59A	0	0	12	4
total posterior subjects:				
62A	32	14	300	118
71A	12	7	300	126
98A	14	10	300	154
78A	35	14	300	138
82A	41	20	300	172
83A	0	0	300	164
84A	16	4	300	151
87A	17	12	300	147
88A	21	5	300	182
89A	45	16	300	137
2C	18	7	300	159

partial posterior subjects:

64A	18	7	53	27
65A	4	1	22	8
8B	6	3	42	25
74A	19	5	40	19
75A	0	0	34	12
9B	24	12	23	11
77A	31	14	54	27
81A	41	17	59	31
85A	15	4	97	43
86A	11	5	25	7
90A	18	10	24	9
91A	17	4	55	25

Descriptive Statistics

Pre-treatment Data

group	black-white total trials	black-white total errors	pattern total trials	pattern total errors
normal control	M = 7.2 SD = 8.4	M = 3.2 SD = 3.9	M = 79.5 SD = 18.6	M = 30.9 SD = 7.8
total posterior	M = 14.0 SD = 13.3	M = 6.0 SD = 5.9	M = 72.9 SD = 19.6	M = 29.8 SD = 8.6
partial posterior	M = 9.8 SD = 8.9	M = 3.9 SD = 4.1	M = 69.2 SD = 31.9	M = 31.0 SD = 16.6

Post-treatment Data

group	black-white total trials	black-white total errors	pattern total trials	pattern total errors
normal control	M = 4.6 SD = 6.5	M = 2.5 SD = 3.6	M = 11.7 SD = 11.2	M = 5.1 SD = 4.8
total posterior	M = 22.8 SD = 13.1	M = 9.9 SD = 5.7	M = 300.0 SD = 0.0	M = 149.8 SD = 18.5
partial posterior	M = 17.0 SD = 11.0	M = 6.8 SD = 5.1	M = 44.0 SD = 20.7	M = 20.3 SD = 10.8

Statistical Analysis

Pre-treatment Data

black-white total trials:

	normal	total posterior	partial posterior
number of subjects above median	4	6	7
number of subjects below median	6	5	5

$X^2 = 0.82$, df = 2, $p > 0.05$

black-white total errors:

	normal	total posterior	partial posterior
number of subjects above median	4	6	6
number of subjects below median	6	5	6

$X^2 = 0.49$, df = 2, $p > 0.05$

pattern total trials:

	normal	total posterior	partial posterior
number of subjects above median	6	5	5
number of subjects below median	4	6	7

$X^2 = 0.82$, df = 2, $p > 0.05$

pattern total errors:

	normal	total posterior	partial posterior
number of subjects above median	6	5	6
number of subjects below median	4	6	6

$X^2 = 0.49$, df = 2, $p > 0.05$

Post-treatment Data

black-white total trials:

	normal	total posterior	partial posterior
number of subjects above median	1	8	8
number of subjects below median	9	3	4

$X^2 = 10.01$, df = 2, $p < 0.01$

black-white total trials: two group comparisons

normal - partial posterior

	normal	partial posterior
number of subjects above median	2	9
number of subjects below median	8	3

$X^2 = 6.60$, df = 1, $p < 0.02$

partial posterior - total posterior

	partial posterior	total posterior
number of subjects above median	6	6
number of subjects below median	6	5

$X^2 = 0.18$, df = 1, $p > 0.05$

black-white total errors

	normal	total posterior	partial posterior
number of subjects above median	3	8	5
number of subjects below median	7	3	7

$X^2 = 4.21$, df = 2, $p > 0.05$

pattern total trials

	normal	total posterior	partial posterior
number of subjects above median	0	11	5
number of subjects below median	10	0	7

$X^2 = 21.33$, df = 2, $p < 0.001$

pattern total trials: two group comparisons

normal - partial posterior

	normal	partial posterior
number of subjects above median	2	9
number of subjects below median	8	3

$X^2 = 6.60$, df = 1, $p < 0.02$

partial posterior - total posterior

	partial posterior	total posterior
number of subjects above median	1	11
number of subjects below median	11	0

$X^2 = 19.33$, df = 1, $p < 0.001$

pattern total errors

	normal	total posterior	partial posterior
number of subjects above median	0	11	6
number of subjects below median	10	0	6

$X^2 = 21.00$, df = 2, $p < 0.001$

pattern total errors: two group comparisons

normal - partial posterior

	normal	partial posterior
number of subjects above median	2	9
number of subjects below median	8	3

$X^2 = 6.60$, df = 1, $p < 0.02$

partial posterior - total posterior

	partial posterior	total posterior
number of subjects above median	1	11
number of subjects below median	11	0

$X^2 = 19.33$, df = 1, $p < 0.001$

Histology

62A: In the left hemisphere all of area 17 was included in the lesion. In the right hemisphere the lesion included all but the most posterior portion of area 17. Area 18 was damaged only in its lateral portion in both hemispheres. The very posterior portion of area 18a remained intact in both hemispheres. There was no damage to structures below the corpus callosum in either hemisphere. Degeneration was evident throughout the left pars dorsalis and in the dorsal-lateral portion of the pars posterior in the left hemisphere. Degeneration appeared throughout all but the dorsal tip of the pars dorsalis and in the dorsal-lateral portion of the pars posterior in the right hemisphere.

71A: In both hemispheres all of area 17 was included in the lesion. In the left hemisphere all of area 18a was included in the lesion. In the right hemisphere only the very posterior portion of area 18a remained intact. In both hemispheres only the medial portion of area 18 remained intact. In the right hemisphere the lesion was unusually shallow frequently not reaching the level of the corpus callosum. There was no damage to structures lieing below the corpus callosum in either hemisphere. In both hemispheres degeneration appeared in all but the dorsal tip of the pars dorsalis and in the dorsal-lateral portion of the pars posterior.

98A: In both hemispheres the lesion included all but the posterior portion of area 17. In both hemispheres the medial portion of area 18 remained intact. The very posterior portion of area 18a remained intact in both hemispheres. There was no damage to structures below the corpus callosum in either hemisphere. In both hemispheres degeneration was found throughout the pars dorsalis and in the dorsal- lateral portion of the pars posterior.

78A: In the right hemisphere all of area 17 was included within the lesion. In the left hemisphere the posterior portion of area 17 remained intact. In both hemispheres the medial portion of area 18 remained intact. In both hemispheres only the posterior pole of area 18a remained intact. There was no damage to structures below the corpus callosum in either hemisphere. In both hemispheres degeneration was found throughout the pars dorsalis and in the dorsal- lateral portion of the pars posterior.

82A: In the left hemisphere all of area 17 was included in the lesion. In the right hemisphere all but the posterior-medial section of area 17 was included in the lesion. In both hemispheres only the medial portion of area 18 remained intact;

the intact portion of area 18 was slightly larger in the right hemisphere. In both hemispheres only the very posterior section of area 18a remained intact. There was no damage to structures beneath the corpus callosum in either hemisphere. In the right hemisphere degeneration was found throughout the pars dorsalis and in the dorsal-lateral section of the pars posterior. In the left hemisphere degeneration was found throughout the pars dorsalis, except in its dorsal pole, and in the dorsal-lateral portion of the pars posterior.

83A: In each hemisphere a small portion of the posterior-medial part of area 17 remained intact. In both hemispheres the medial portion of area 18 remained intact. In both hemispheres the posterior portion of area 18a remained intact. Subcortical damage was nonexistent. Degeneration was found throughout the pars dorsalis in each hemisphere. Degeneration was present in the dorsal-lateral portion of the pars posterior in each hemisphere.

84A: In the right hemisphere the lesion included all of area 17; in the left hemisphere the posterior-medial portion of area 17 remained intact. In both hemispheres the medial portion of area 18 was undamaged. In the left hemisphere the dorsal-medial portion of area 18a remained intact. In the right hemisphere the posterior portion of area 18a remained intact. Subcortical damage was nonexistent. Degeneration was found throughout the pars dorsalis in each hemisphere; degeneration was found in the dorsal-lateral area of the pars posterior in each hemisphere.

87A: In both hemispheres the posterior portion of area 17 was not included in the lesion. In both hemispheres damage was limited to the lateral portion of area 18. In both hemispheres the posterior portion of area 18a remained intact. In both hemispheres degeneration was found throughout the pars dorsalis and in the dorsal-lateral portion of the pars posterior. There was no damage to structures lieing below the corpus callosum.

88A: In the left hemisphere the posterior portion of area 17 remained intact; in the right hemisphere the lesion included all of area 17. In both hemispheres damage was limited to the lateral portion of area 18; damage to area 18 was more extensive in the right hemisphere. In both hemispheres the posterior portion of area 18a remained intact. In both hemispheres degeneration was present throughout the pars dorsalis and in the dorsal-lateral area of the pars posterior. There was no damage to structures lieing below the corpus callosum.

89A: In both hemispheres the posterior portion of area 17 was not included in the lesion. In both hemispheres the lesion was

limited to the lateral portion of area 18. In both hemispheres the posterior portion of area 18a was not included in the lesion. In both hemispheres degeneration was present throughout the pars dorsalis and in the dorsal-lateral area of the pars posterior. Subcortical damage was nonexistent.

2C: In each hemisphere a small portion of the posterior part of area 17 remained intact. In both hemispheres the lesion included only the lateral portion of area 18. In both hemispheres the posterior portion of area 18a was left essentially intact. Problems with the staining of the tissue made it impossible to accurately determine the extent and location of thalamic degeneration. There was no damage to structures lieing below the corpus callosum.

64A: In both hemispheres a large percentage of the cells in area 17 remained intact. The lesion was limited to the lateral portion of area 18 in both hemispheres. The lesion included all but the posterior portion of area 18a in both hemispheres. In both hemispheres degeneration was limited to the dorsal-lateral area of the pars posterior and a small area in the medial portion of the pars dorsalis. Subcortical damage was nonexistent.

65A: In both hemispheres damage to area 17 was limited to its posterior portion. The lesion was limited to the lateral 2/3 of area 18 in both hemispheres. The damage to area 18a included all but the posterior portion in each hemisphere. In both hemispheres degeneration was found in the dorsal-lateral area of the pars posterior and the medial portion of the pars dorsalis. There was no damage to structures lieing below the corpus callosum.

8B: In both hemispheres most of area 17 remained essentially intact; damage to area 17 was limited primarily to the posterior portion. In both hemispheres the damage to area 18 was limited to the lateral 2/3 of that area. The damage to area 18a in both hemispheres was extensive except for the posterior portion. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior and the medial area of the pars dorsalis; degeneration was more extensive in the left hemisphere. Subcortical damage was nonexistent.

74A: In both hemispheres most of area 17 remained essentially intact; damage to area 17 was limited primarily to the posterior portion. Damage to area 18 was limited to the lateral 2/3 in both hemispheres. In both hemispheres the damage to area 18a was extensive except for the posterior portion. Problems with the tissue stain made it impossible to determine the extent and

location of thalamic degeneration. There was no damage to structures lieing ventral to the corpus callosum.

75A: In both hemispheres most of area 17 remained intact; damage to area 17 was limited primarily to the posterior portion. In area 18 damage was limited to the lateral 2/3 in both hemispheres. In both hemispheres the damage to area 18a was extensive except for the posterior portion where damage was light. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior and in the medial area of the pars dorsalis. There was no damage to structures lieing below the corpus callosum.

9B: In both hemispheres there was rather heavy damage to the posterior portion of area 17; the anterior portion of area 17 remained essentially intact in both hemispheres. In both hemispheres damage to area 18 was restricted to the lateral 2/3. In both hemispheres only the posterior portion of area 18a escaped extensive damage. In both hemispheres degeneration was evident in the lateral portion of the pars posterior and the medial area of the pars dorsalis. Degeneration was more extensive in the right hemisphere. There was no subcortical damage.

77A: In both hemispheres most of area 17 remained intact; damage to area 17 was limited primarily to the posterior portion. In both hemispheres damage to area 18 was limited to the lateral 2/3. In both hemispheres only the posterior portion of area 18a escaped extensive damage. In both hemispheres degeneration was evident in the lateral area of the pars posterior and the medial area of the pars dorsalis. There was no damage to structures lieing below the corpus callosum.

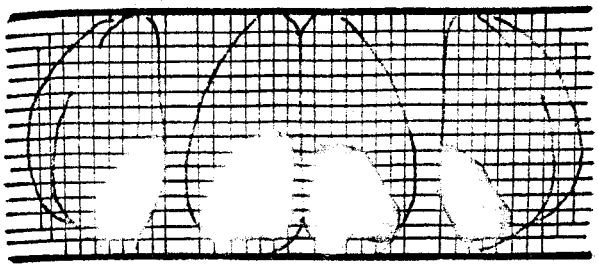
81A: In both hemispheres damage to area 17 was limited primarily to the posterior extent. In both hemispheres damage to area 18 was limited primarily to the lateral portion. In both hemispheres the damage to area 18a included all but the posterior portion. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior and in the medial area of the pars dorsalis. There was no damage to structures lieing below the corpus callosum.

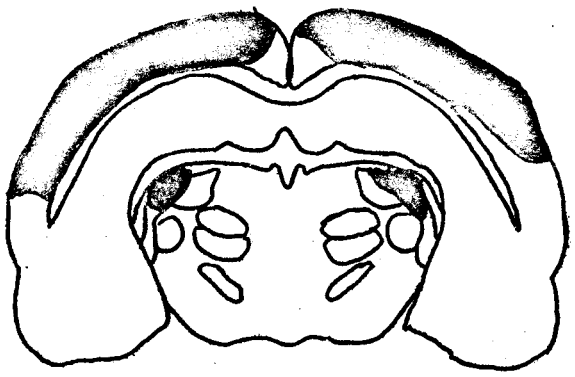
85A: In both hemispheres damage to area 17 was limited primarily to the posterior portion. In both hemispheres the lesion in area 18 was limited to the lateral portion. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior and the medial portion of the pars dorsalis. Subcortical damage was not present.

86A: In both hemispheres damage to area 17 was minor and limited to the posterior portion. In both hemispheres damage to area 18 was limited to the lateral portion. In both hemispheres the lesion included all but the posterior portion of area 18a. Degeneration was heavier in the right hemisphere; in both hemispheres degeneration was evident in the dorsal-lateral portion of the pars posterior and the medial portion of the pars dorsalis. There was no damage to structures lieing below the corpus callosum.

90A: In both hemispheres damage to area 17 was minor and limited to the posterior portion. In both hemispheres damage to area 18 was limited to the lateral portion. In both hemispheres the lesion included all but the posterior portion of area 18a. In both hemispheres degeneration was evident in the lateral portion of the pars posterior and the medial portion of the pars dorsalis. Subcortical damage was nonexistent.

91A: In both hemispheres damage to area 17 was limited primarily to the posterior portion. In both hemispheres damage to area 18 was limited to the lateral 2/3. In both hemispheres the lesion included all but the posterior portion of area 18a. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior and the medial portion of the pars dorsalis. Subcortical damage was nonexistent.





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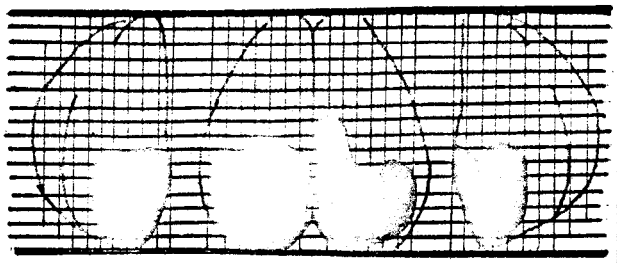

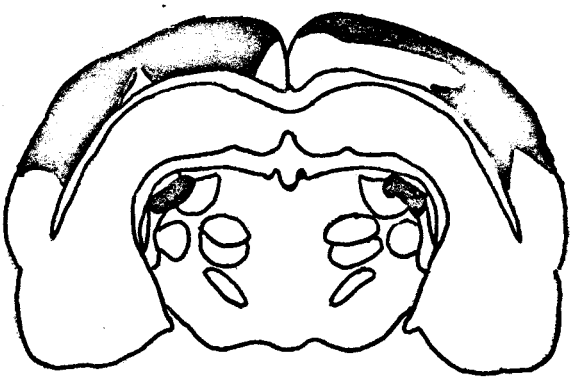

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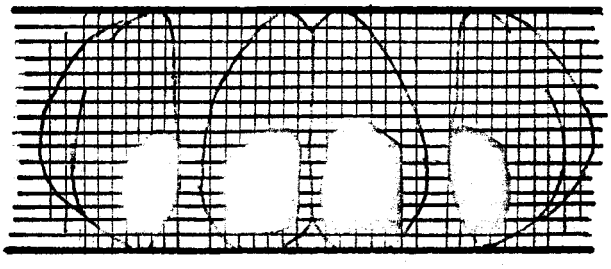

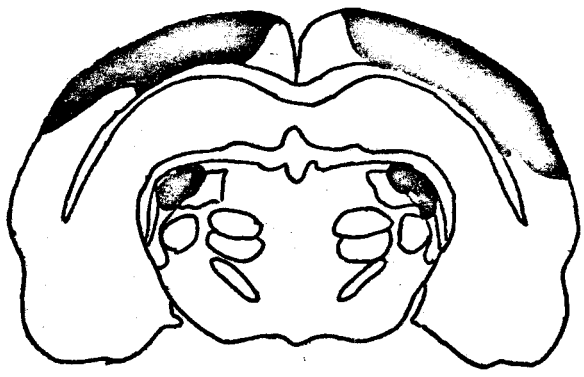

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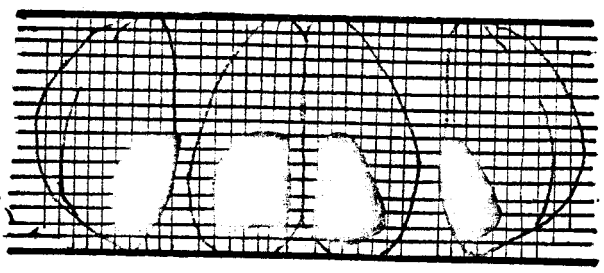

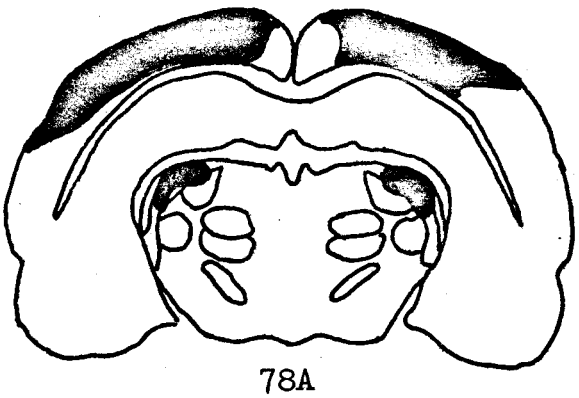

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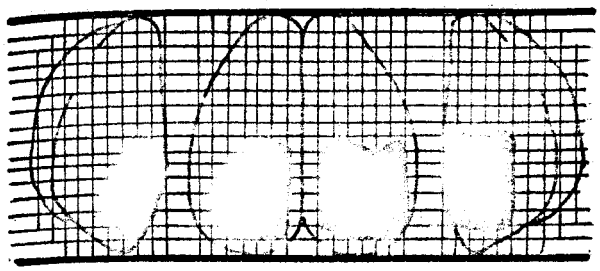

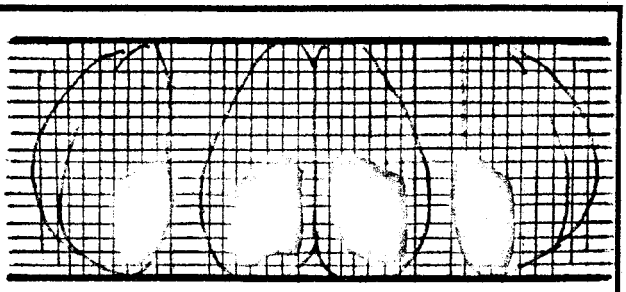

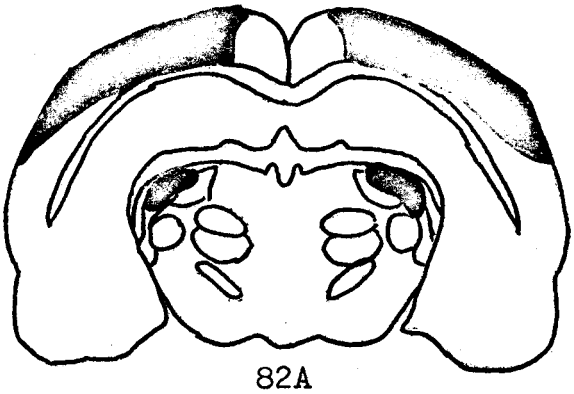

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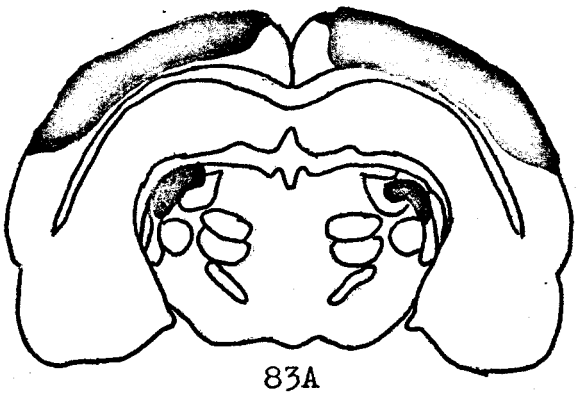

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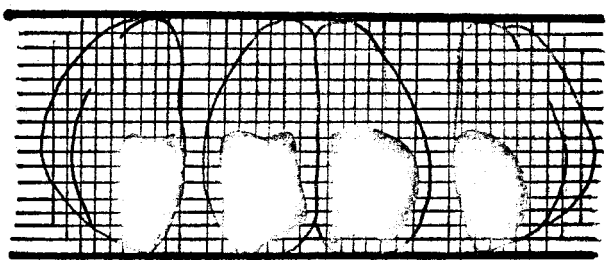

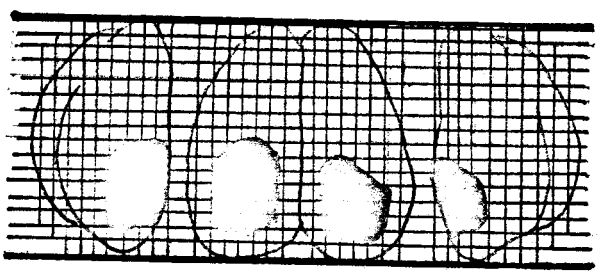

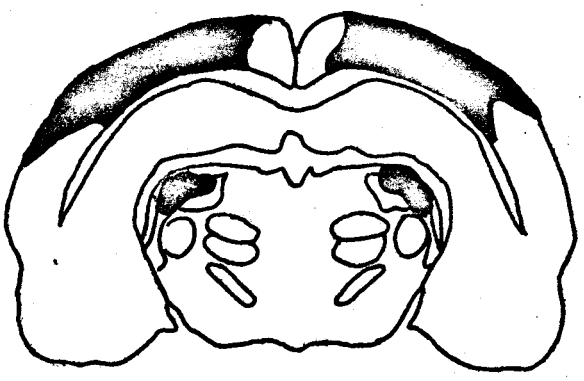

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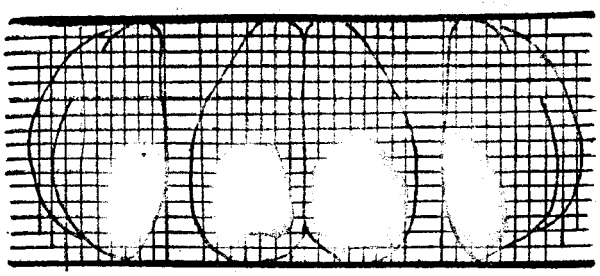

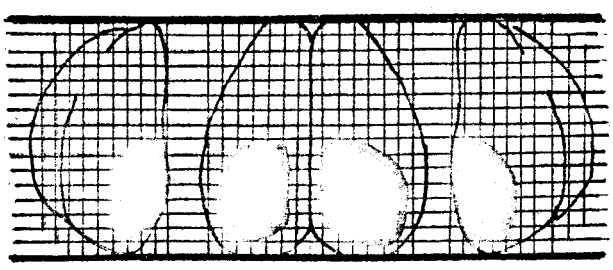

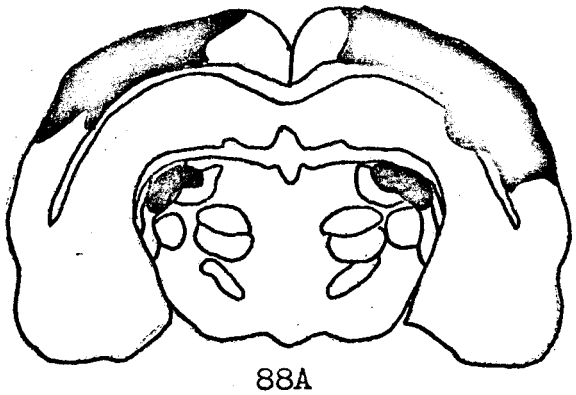

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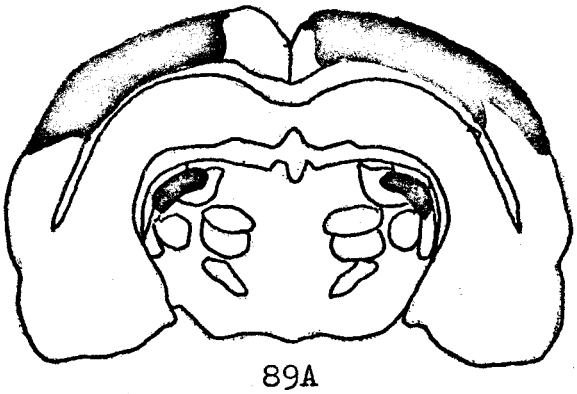

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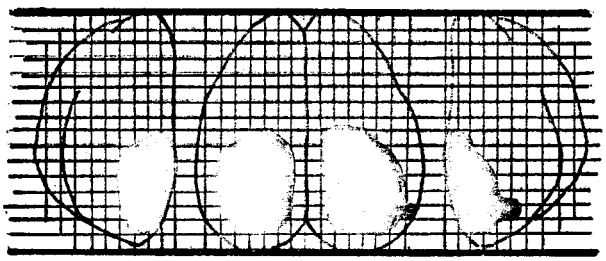

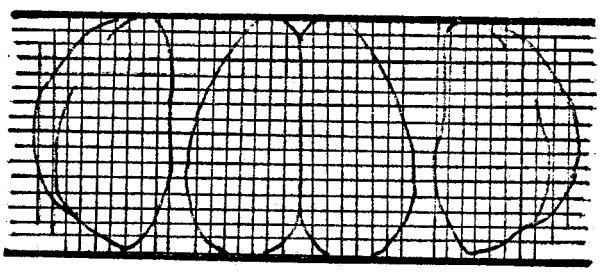

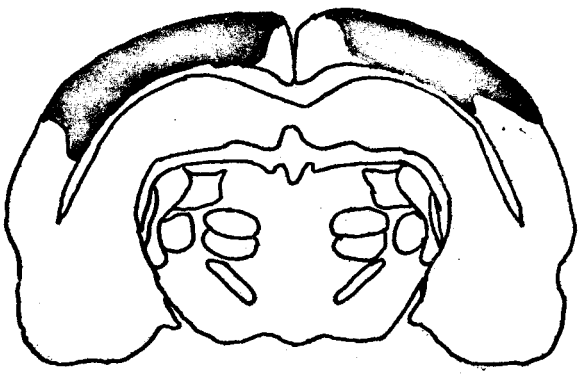

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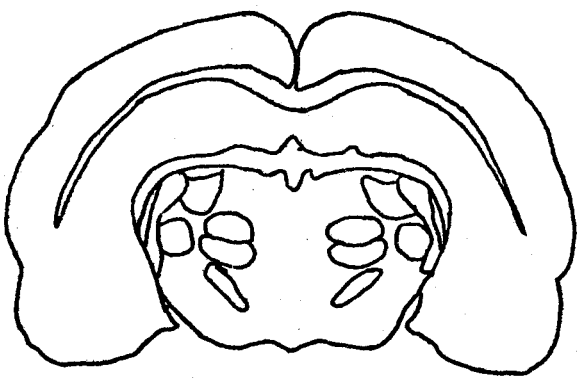

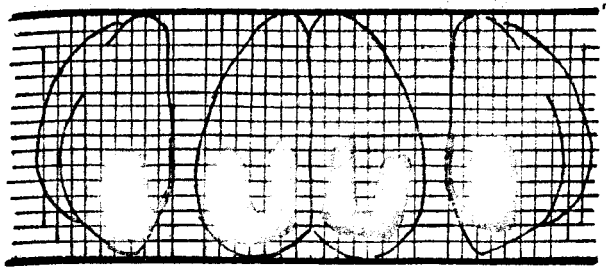

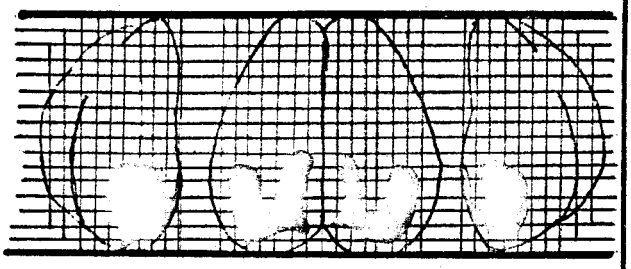

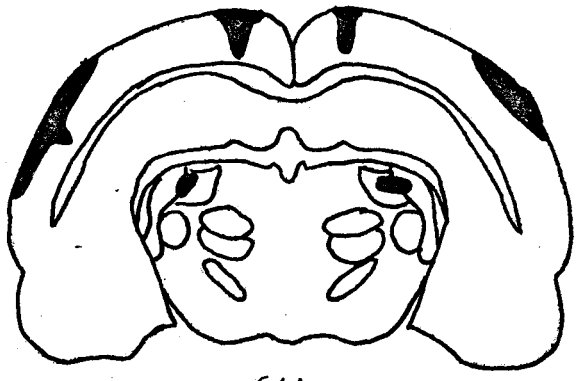

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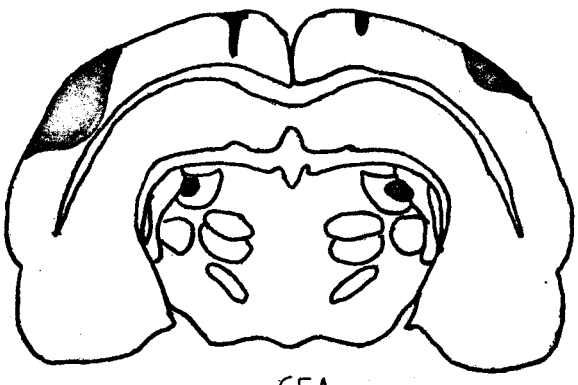

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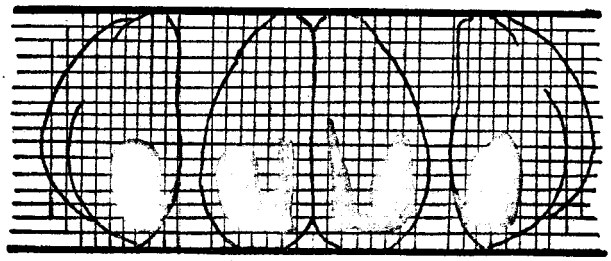

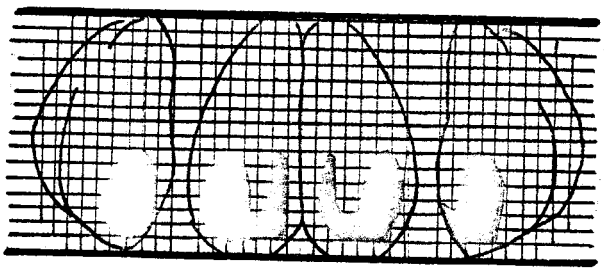

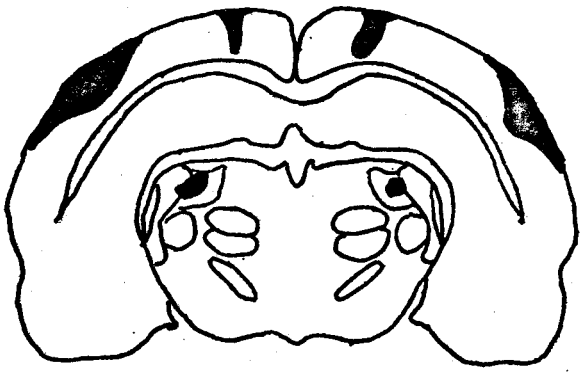

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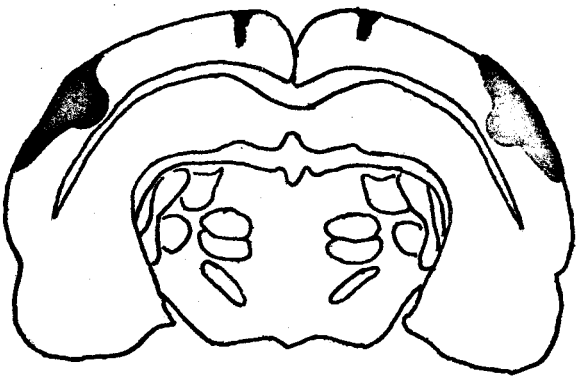

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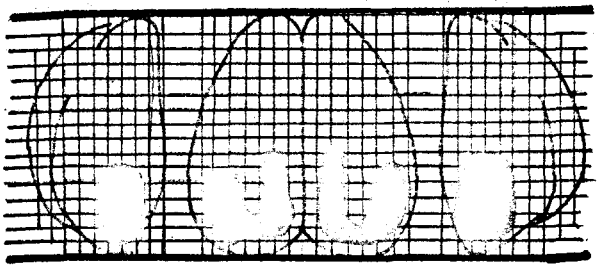

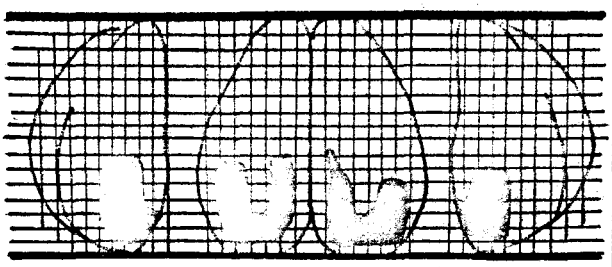

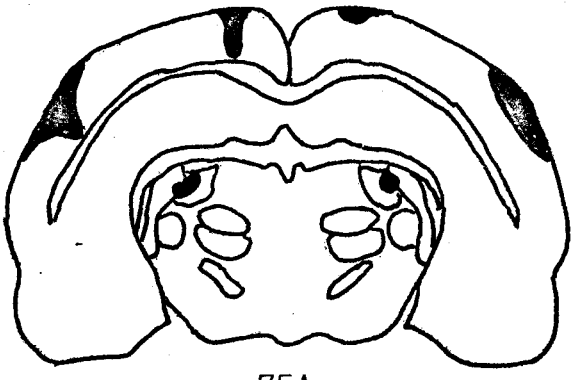

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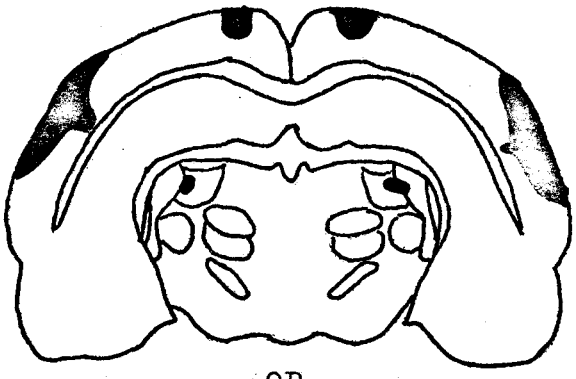

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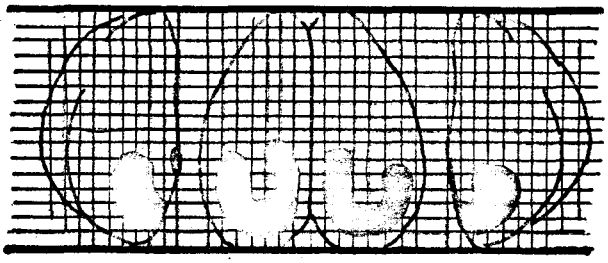

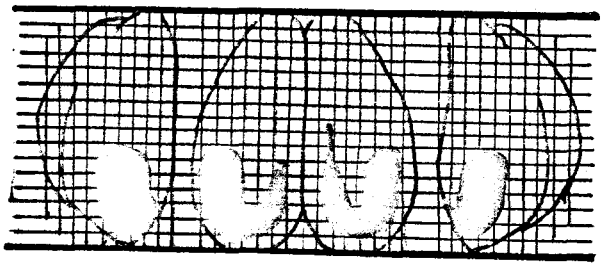

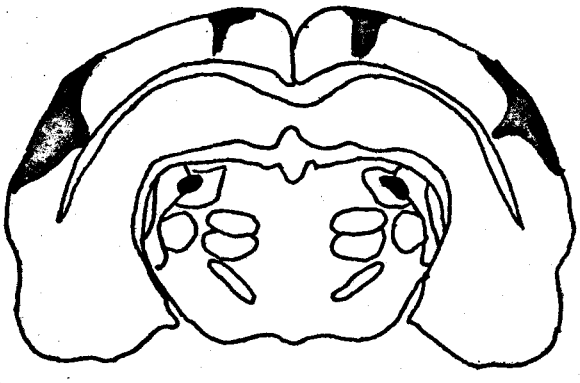

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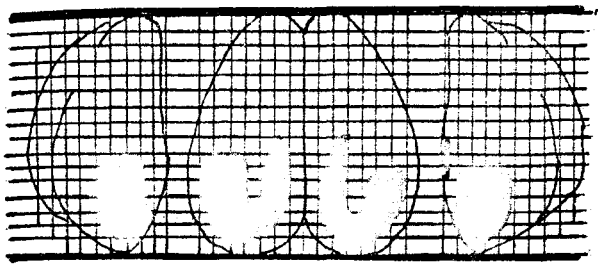

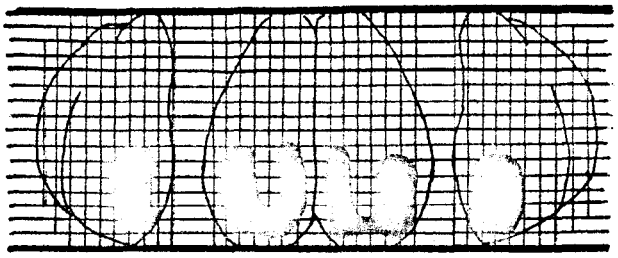

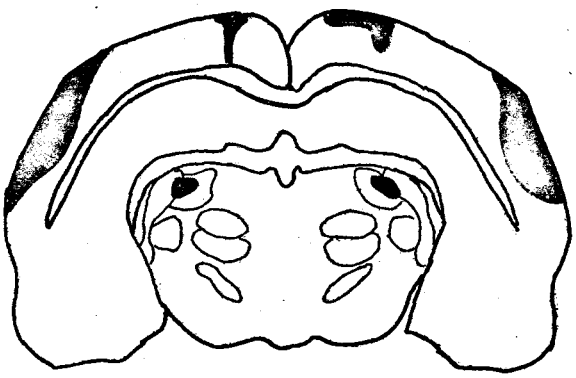

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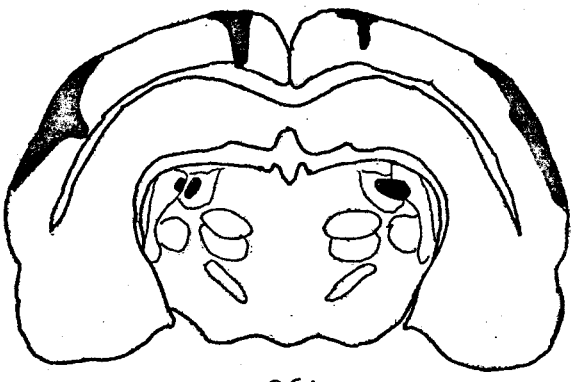

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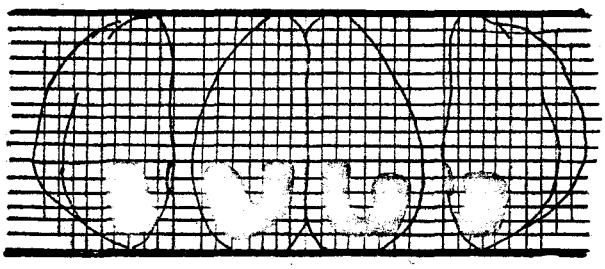

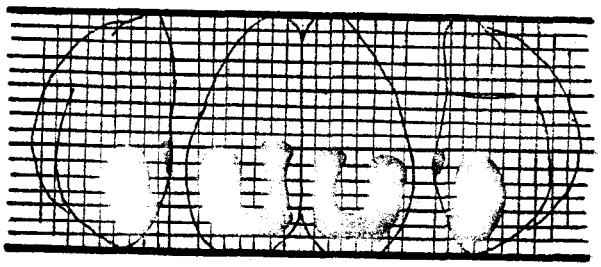

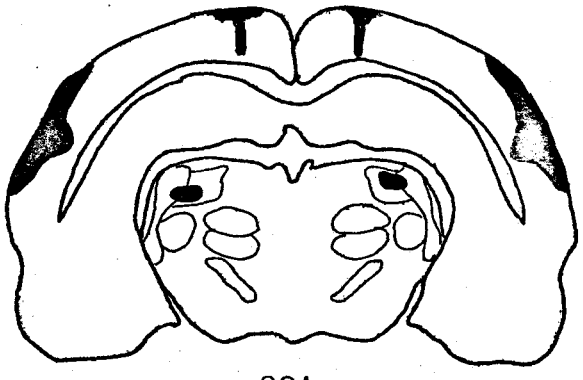

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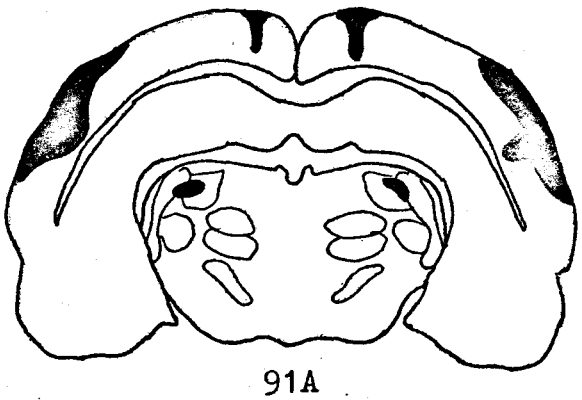


91A

Appendix 3

Appendix 3 includes the raw data, summary statistics, statistical analysis, lesion descriptions, and lesion diagrams for the subjects in Experiment 3.

Raw Scores

subject	black-white total trials	black-white total errors	pattern total trials	pattern total errors
normal subjects:				
20A	42	20	98	40
23A	38	18	115	43
32A	57	27	69	27
45A	90	48	120	42
47A	64	24	94	34
50A	28	12	83	35
60A	36	15	150	64
67A	42	18	80	29
79A	70	30	72	31
81A	68	31	88	39
total posterior subjects:				
93A	300	172	---	---
1B	287	138	300	179
12B	197	79	300	121
26B	300	134	---	---
66A	300	162	---	---
5B	296	117	300	108
14B	300	128	---	---
33B	234	79	300	196
44B	297	178	300	201
61B	300	152	---	---
partial posterior subjects:				
69A	88	47	287	108
94A	149	73	300	154
95A	185	69	300	193
96A	253	121	300	143
99A	112	62	248	155
7B	300	154	---	---

11B	75	38	257	164
30B	49	18	300	173
18B	98	31	300	139
22B	218	97	300	146
25B	145	91	297	167
40B	118	42	283	158

Descriptive Statistics

group	black-white total errors	black-white total trials	pattern total errors	pattern total trials
normal control	M = 24.3 SD = 9.9	M = 53.5 SD = 18.4	M = 38.4 SD = 10.0	M = 96.9 SD = 23.8
total posterior	M = 133.9 SD = 33.0	M = 281.1 SD = 34.0	M = 161.0* SD = 38.9*	M = 300* SD = 0.0*
partial posterior	M = 70.2 SD = 38.2	M = 149.2 SD = 72.8	M = 154.5** SD = 21.0**	M = 288.4** SD = 17.3**

Statistical Analysis

black-white total trials

	normal	total posterior	partial posterior
number of subjects above median	0	10	6
number of subjects below median	10	0	6

$X^2 = 20.0$, df = 2, $p < 0.001$

* Based on the 5 subjects which reached criterion within 300 trials on the black-white discrimination.

** Based on the 11 subjects which reached criterion within 300 trials on the black-white discrimination.

black-white total trials: two group comparisons

normal - partial posterior

	normal	partial posterior
number of subjects above median	1	10
number of subjects below median	9	2

$X^2 = 11.73$, df = 1, $p < 0.001$

partial posterior - total posterior

	partial posterior	total posterior
number of subjects above median	2	9
number of subjects below median	10	1

$X^2 = 11.73$, df = 1, $p < 0.001$

black-white total errors

	normal	total posterior	partial posterior
number of subjects above median	0	10	6
number of subjects below median	10	0	6

$X^2 = 20.0$, df = 2, $p < 0.001$

black-white total errors: two group comparisons

normal - partial posterior

	normal	partial posterior
number of subjects above median	1	10
number of subjects below median	9	2

$X^2 = 11.73$, df = 1, $p < 0.001$

partial posterior - total posterior

	partial posterior	total posterior
number of subjects above median	3	8
number of subjects below median	9	2

$X^2 = 6.60$, df = 1, $p < 0.02$

*pattern total trials

	normal	partial posterior
number of subjects above median	0	11
number of subjects below median	10	0

$X^2 = 21.00$, df = 1, $p < 0.001$

*pattern total errors

	normal	partial posterior
number of subjects above median	0	11
number of subjects below median	10	0

$X^2 = 21.00$, df = 1, $p < 0.001$

* The total posterior subjects were not included in the pattern discrimination analysis because there were to few of them to meet the expected frequency requirements of the median test. Only the 11 of the 12 partial posterior subjects that reached criterion on the black-white task were tested on the pattern task; hence only those 11 were included in the analysis.

Histology

93A: In both hemispheres all of area 17 was included in the lesion. In both hemispheres only the lateral portion of area 18 was included in the lesion. In both hemispheres all but the posterior portion of area 18a was included in the lesion. Degeneration was evident throughout the pars dorsalis and in the dorsal-lateral area of the pars posterior in both hemispheres. There was no damage to structures lieing below the corpus callosum.

1B: In both hemispheres the lesion included all of area 17. In both hemispheres all but a small portion of the medial part of area 18 was included in the lesion. In both hemispheres all of area 18a was included in the lesion. Degeneration was present throughout the pars dorsalis and in the dorsal-lateral area of the pars posterior. There was no damage to structures lieing below the corpus callosum.

12B: In both hemispheres the lesion included all but the very posterior tip of area 17. In both hemispheres the lesion was limited to the lateral 2/3 of area 18. In both hemispheres the lesion included all but the posterior portion of area 18a. In both hemispheres degeneration was present throughout the pars dorsalis and in the dorsal-lateral area of the pars posterior. There was no subcortical damage.

26B: In both hemispheres the lesion included all of area 17. In both hemispheres all but the medial portion of area 18 was included in the lesion. In the right hemisphere all but the posterior-medial portion of area 18a was included in the lesion. In the left hemisphere all but the posterior portion of area 18a was included in the lesion. In the right hemisphere degeneration was present throughout the pars dorsalis and in the dorsal-lateral area of the pars posterior. In the left hemisphere degeneration was evident throughout the medial 3/4 of the pars dorsalis and in the dorsal-lateral area of the pars posterior. There was no damage to structures below the corpus callosum.

66A: In both hemispheres the lesion included all but the posterior portion of area 17. In both hemispheres the lesion was limited to the lateral portion of area 18. In the right hemisphere the lesion included all but the posterior portion of area 18a. In the left hemisphere the lesion included all but the posterior portion and lateral tip of area 18a. In both hemispheres degeneration was evident throughout the pars dorsalis and in the dorsal-lateral area of the pars posterior. There was no damage to structures below the corpus callosum.

5B: In the left hemisphere the lesion included all of area 17. In the right hemisphere the lesion included all but the posterior portion of area 17. In both hemispheres the lesion included all but the medial portion of area 18. In both hemispheres the lesion included all but the posterior portion of area 18a. In both hemispheres degeneration was evident throughout the pars dorsalis and in the dorsal-lateral area of the pars posterior. There was no damage to structures below the corpus callosum.

14B: In the left hemisphere all but the dorsal-medial tip of area 17 was included in the lesion. In the right hemisphere all but the posterior portion of area 17 was included in the lesion. In the left hemisphere all but the posterior-medial tip of area 18 was included in the lesion. In the right hemisphere all but the medial portion of area 18 was included in the lesion. In both hemispheres all but the posterior portion of area 18a was included in the lesion. In the left hemisphere degeneration was evident throughout the pars dorsalis. In the right hemisphere degeneration was evident in all but the dorsal tip of the pars dorsalis. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior. Subcortical damage was limited to the dorsal tip of the left hippocampus.

33B: In the left hemisphere the lesion included all of area 17. In the right hemisphere the lesion included all but the posterior-medial portion of area 17. In both hemispheres the lesion included all but the medial portion of area 18. In the left hemisphere the lesion included all but the posterior portion of area 18a. In the right hemisphere the lesion included all but the posterior-medial portion of area 18a. In both hemispheres degeneration was evident throughout the pars dorsalis and in the dorsal-lateral area of the pars posterior. There was no damage to structures below the corpus callosum.

44B: In the left hemisphere the lesion included all but the posterior-medial portion of area 17. In the right hemisphere the lesion included all of area 17. In the left hemisphere the lesion included essentially all of area 18. In the right hemisphere the lesion included all but the posterior-medial portion of area 18. In both hemispheres the lesion included all but the posterior portion of area 18a. In both hemispheres degeneration was evident throughout the pars dorsalis and in the dorsal-lateral area of the pars posterior. There was no damage to structures lieing below the corpus callosum.

61B: In the left hemisphere the lesion included all of area 17; in the right hemisphere the lesion included all but the posterior tip of area 17. In both hemispheres the lesion

included all but the medial portion of area 18. In both hemispheres the lesion included all but the posterior portion of area 18a. In both hemispheres degeneration was evident throughout the pars dorsalis and in the dorsal-lateral area of the pars posterior. There was no damage to structures below the corpus callosum.

69A: In both hemispheres all but the posterior portion of area 17 remained intact. In both hemispheres the lesion included all but the medial portion of area 18. In both hemispheres the lesion included all but the posterior portion of area 18a. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior and the medial tip of the pars dorsalis. There was no damage to structures below the corpus callosum.

94A: In the right hemisphere only the posterior portion of area 17 was included in the lesion. In the left hemisphere the middle 2/3 of area 17 remained intact. In both hemispheres the lesion included all but the medial portion of area 18. In both hemispheres all but the posterior portion of area 18a was included in the lesion. In both hemispheres degeneration was evident in the dorsal-lateral portion of the pars posterior and in the medial tip of the pars dorsalis. There was no damage to structures below the corpus callosum.

95A: In the right hemisphere the middle 2/3 of area 17 remained intact; in the left hemisphere only the posterior portion of area 17 was included in the lesion. In both hemispheres only the lateral 2/3 of area 18 was included in the lesion. In both hemispheres all but the posterior portion of area 18a was included in the lesion. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior and the medial portion of the pars dorsalis. There was no damage to structures below the corpus callosum.

96A: In the right hemisphere essentially the entire area 17 remained intact; in the left hemisphere all but the posterior portion of area 17 remained intact. In both hemispheres the lesion included all but the medial portion of area 18. In both hemispheres the lesion included all but the posterior portion of area 18a. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior and the medial tip of the pars dorsalis. There was no damage to structures below the corpus callosum.

99A: In both hemispheres only the posterior portion of area 17 was included in the lesion. In both hemispheres the lesion included all but the medial portion of area 18. In both

hemispheres the lesion included all but the posterior portion of area 18a. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior and the medial tip of the pars dorsalis. There was no damage to structures below the corpus callosum.

7B: In the left hemisphere the lesion included the posterior 1/3 of area 17. In the right hemisphere area 17 remained essentially intact. In both hemispheres the lesion included all but the medial portion of area 18. In both hemispheres the lesion included all but the posterior portion of area 18a. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior and in the medial tip of the pars dorsalis. There was no subcortical damage.

11B: In both hemispheres only the posterior portion of area 17 was included in the lesion. In both hemispheres all but the medial portion of area 18 was included in the lesion. In both hemispheres only the posterior portion of area 18a remained intact. Degeneration in both hemispheres was evident in the dorsal-lateral portion of the pars posterior and in the medial tip of the pars dorsalis. There was no damage to structures below the corpus callosum.

30B: In the left hemisphere essentially the entire area 17 remained intact. In the right hemisphere the middle 1/2 of the striate area remained intact. In both hemispheres all but the medial portion of area 18 was included in the lesion. In both hemispheres all but the posterior portion of area 18a was included in the lesion. In the right hemisphere degeneration was evident in the medial tip of the pars dorsalis. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior. There was no damage to structures below the corpus callosum.

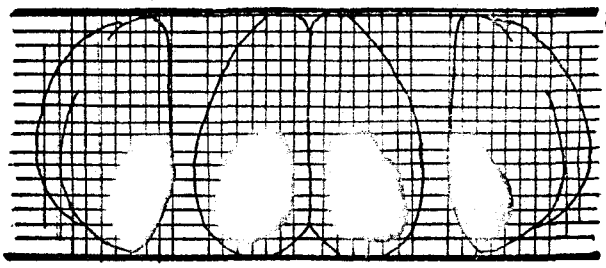
18B: In both hemispheres only the posterior portion of area 17 was included in the lesion. In the right hemisphere essentially all of area 18 was included in the lesion. In the left hemisphere only the lateral portion of area 18 was included in the lesion. In both hemispheres only the posterior portion of area 18a escaped damage. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior and in the medial portion of the pars dorsalis. There was no subcortical damage.

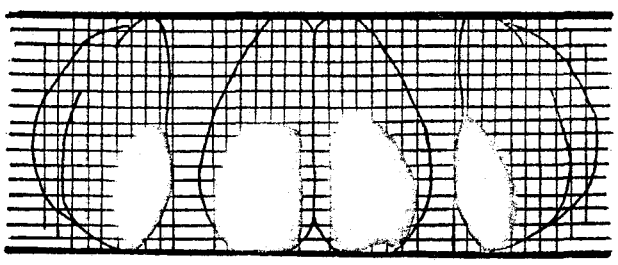
22B: In both hemispheres only the posterior portion of area 17 was included in the lesion. In both hemispheres all but the medial portion of area 18 was included in the lesion. In both hemispheres all but the posterior portion of area 18a was

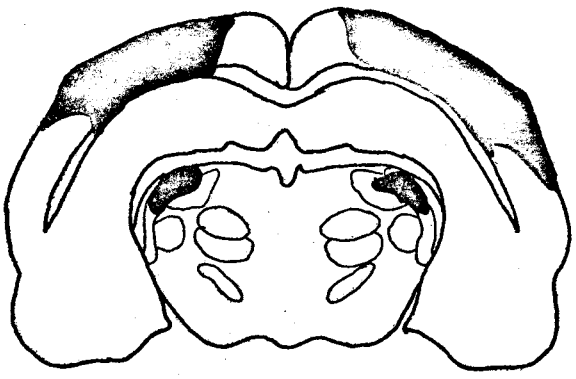
included in the lesion. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior and the medial portion of the pars dorsalis. There was no subcortical damage.

25B: In both hemispheres only the posterior portion of area 17 was included in the lesion. In both hemispheres only the medial portion of area 18 escaped damage. In both hemispheres the posterior portion of area 18a escaped damage. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior and in the medial portion of the pars dorsalis. There was no damage to structures lieing below the corpus callosum in either hemisphere.

40B: In both hemispheres all but the posterior portion of area 17 escaped damage. In both hemispheres all but the medial portion of area 18 was included in the lesion. In both hemispheres all but the posterior portion of area 18a was included in the lesion. In both hemispheres degeneration was evident in the dorsal-lateral area of the pars posterior and in the medial portion of the pars dorsalis. There was no damage to structures below the corpus callosum.







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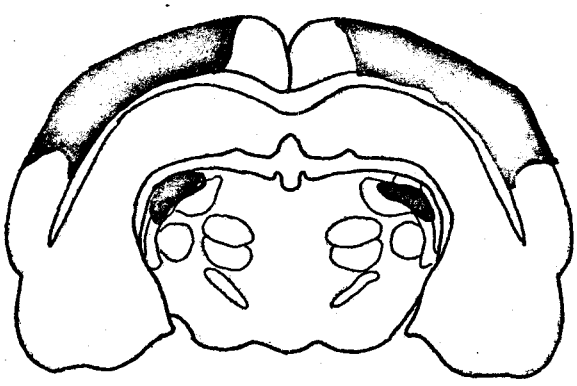

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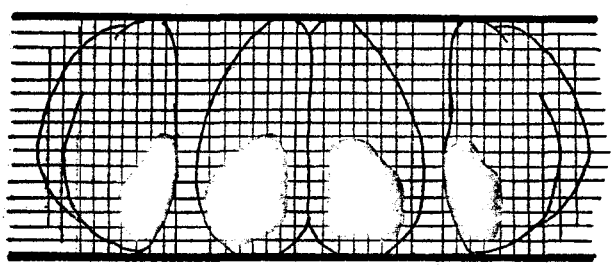

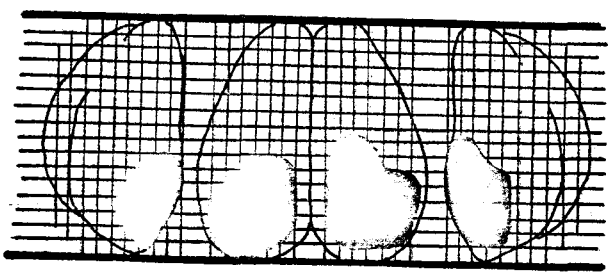

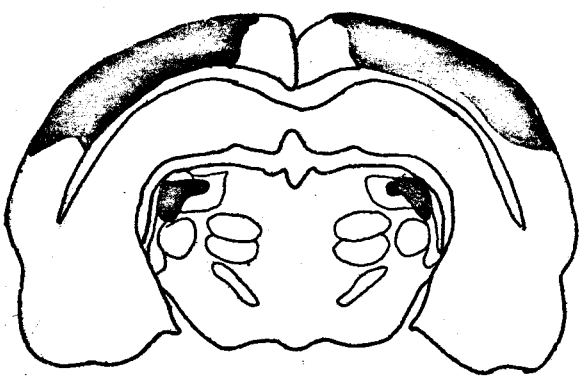

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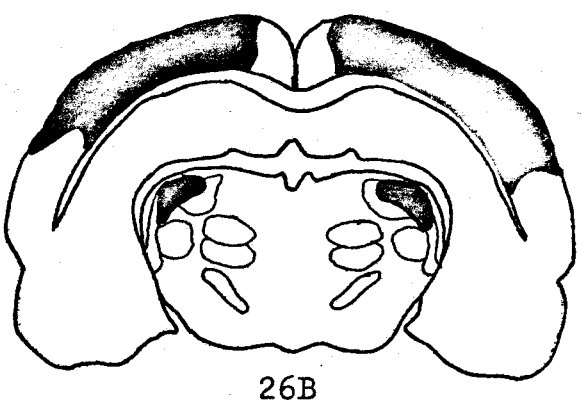

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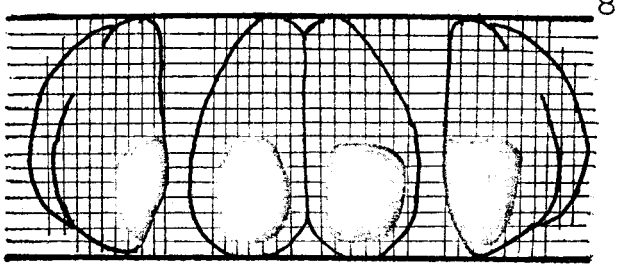

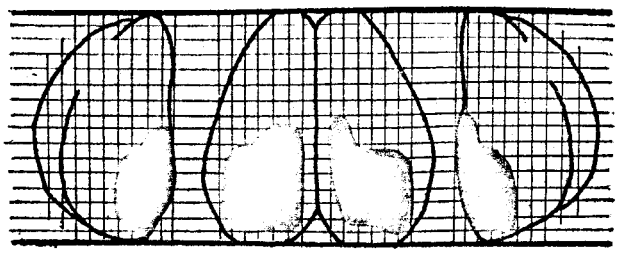

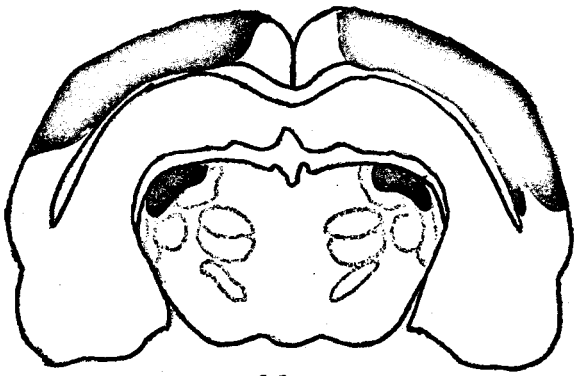

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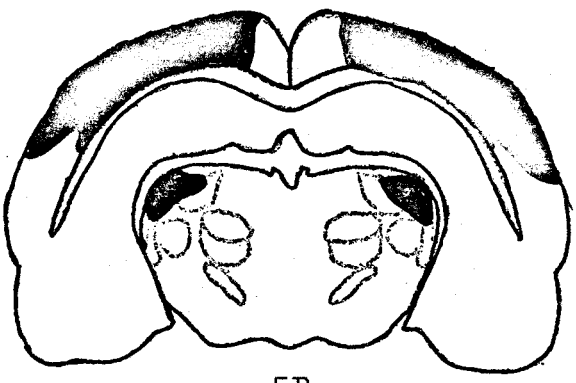

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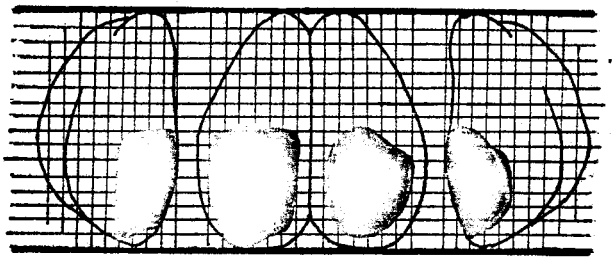

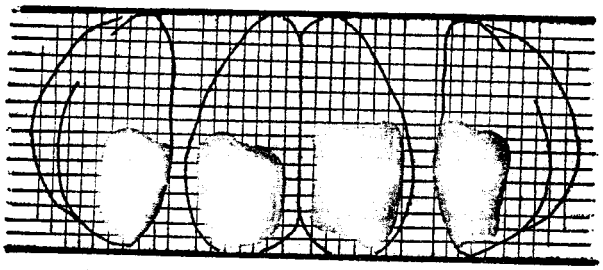

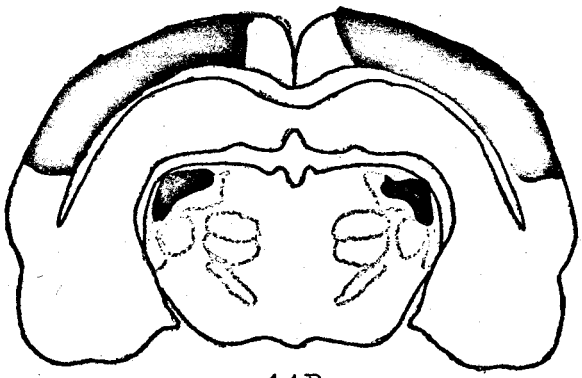

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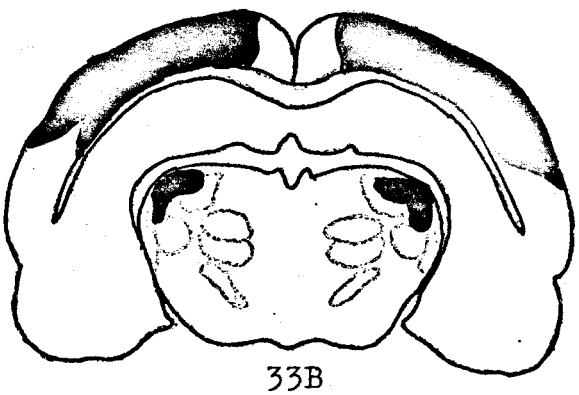

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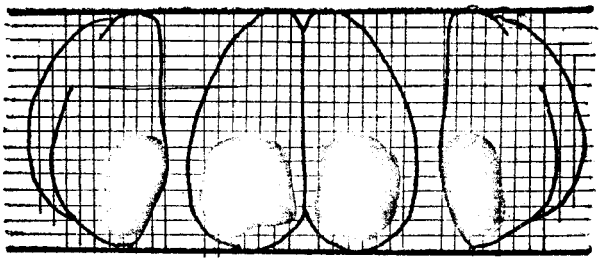

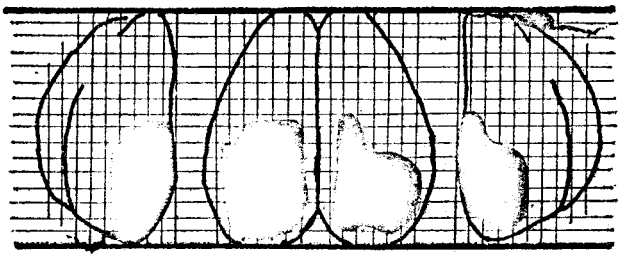

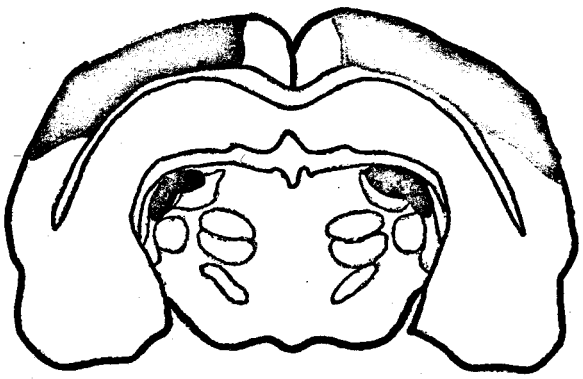

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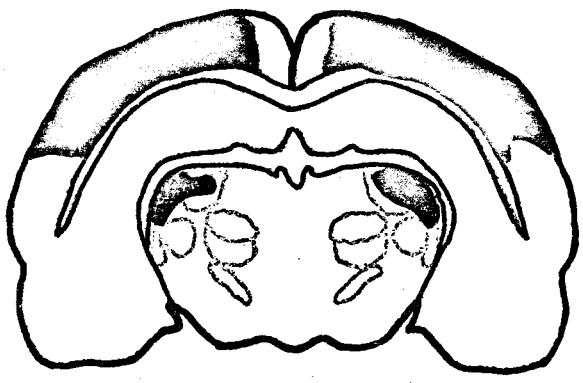

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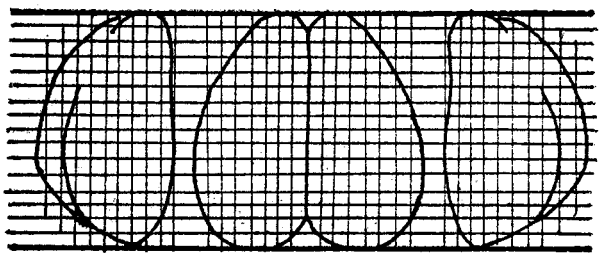

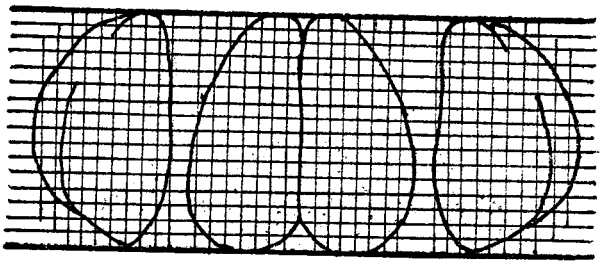

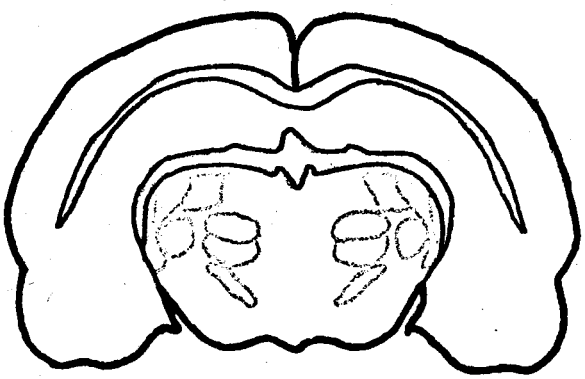

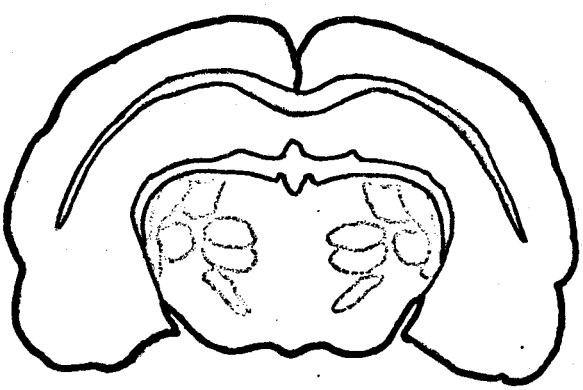

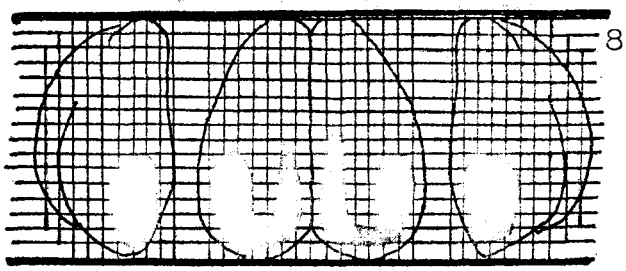

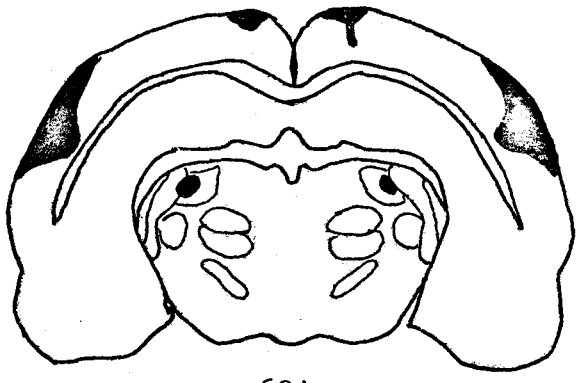

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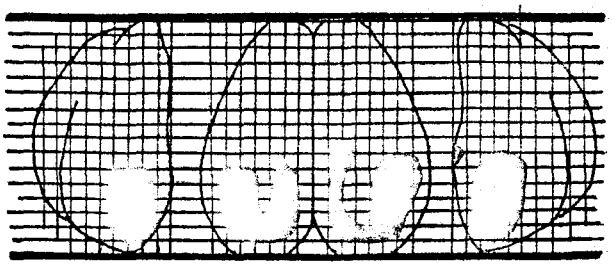

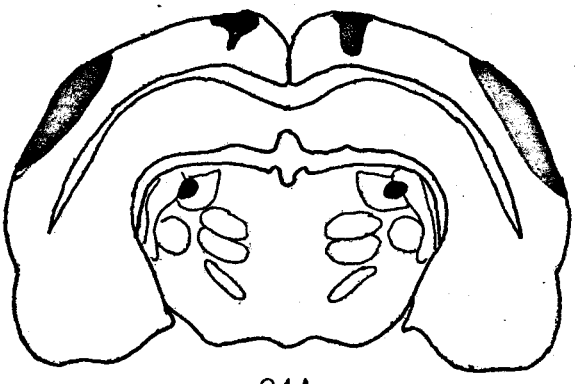

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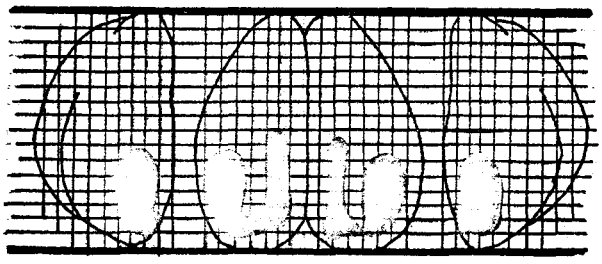

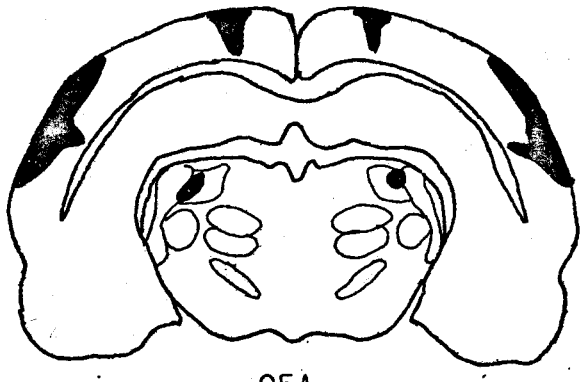

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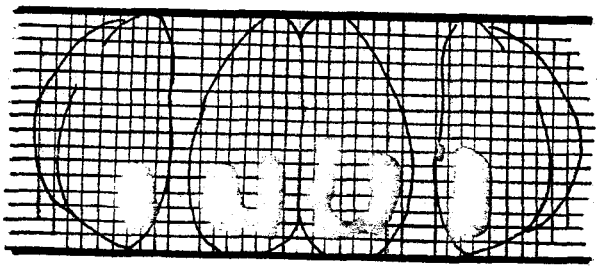

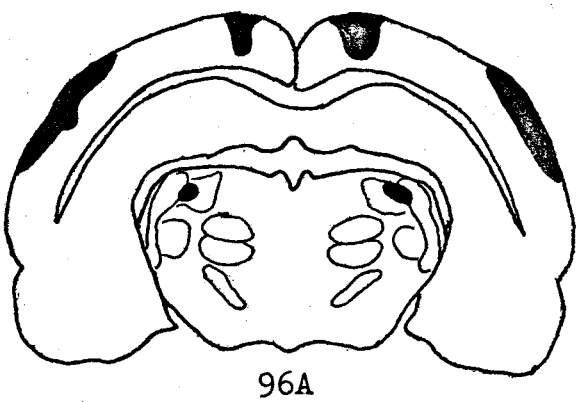

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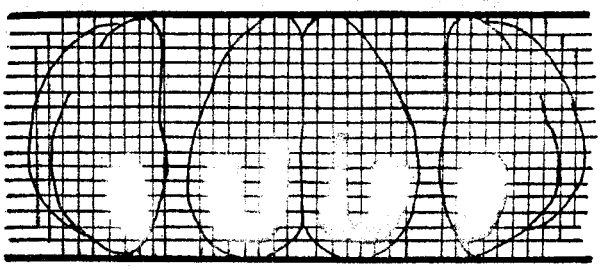

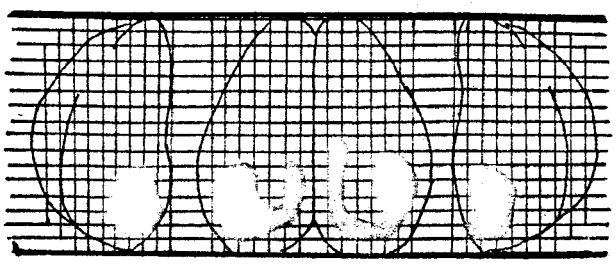

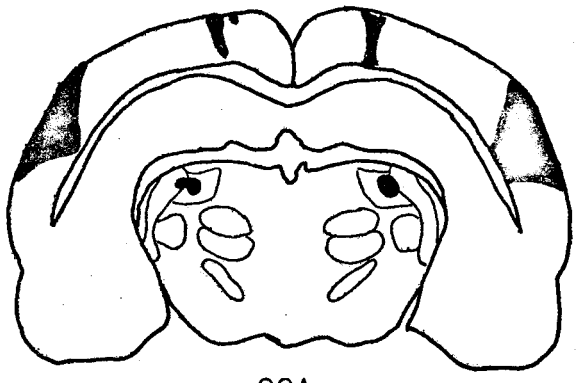

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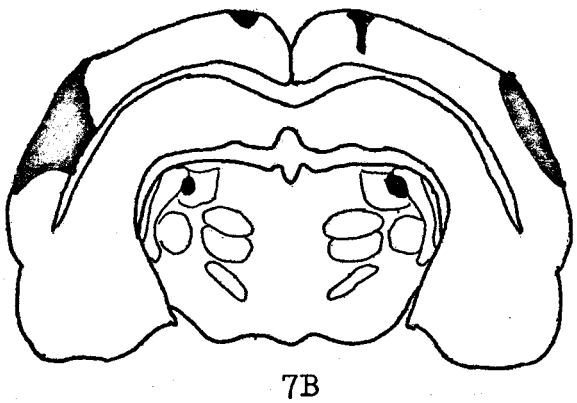

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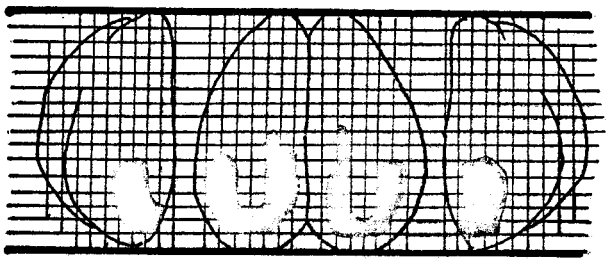

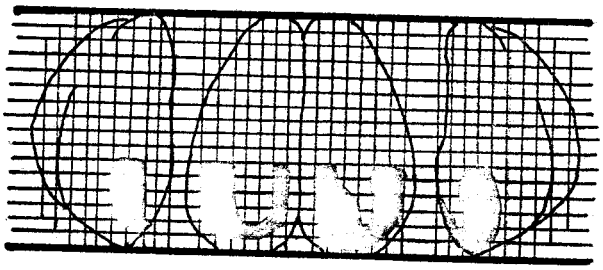

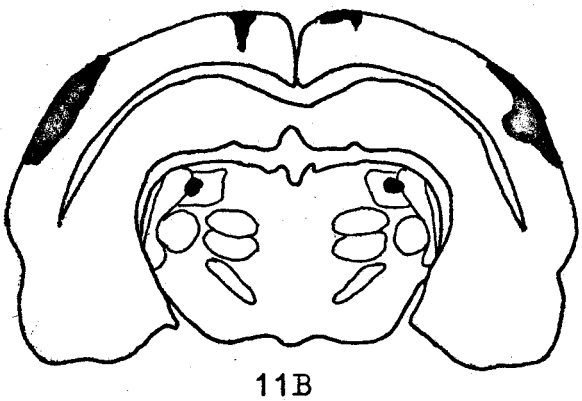

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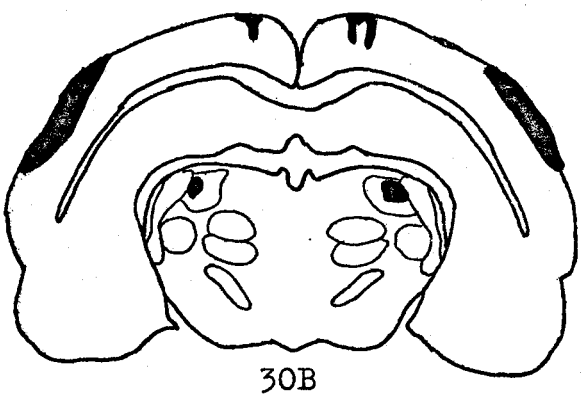

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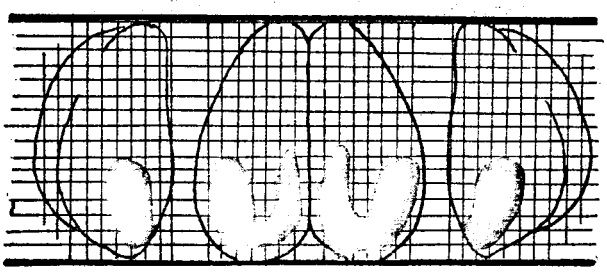

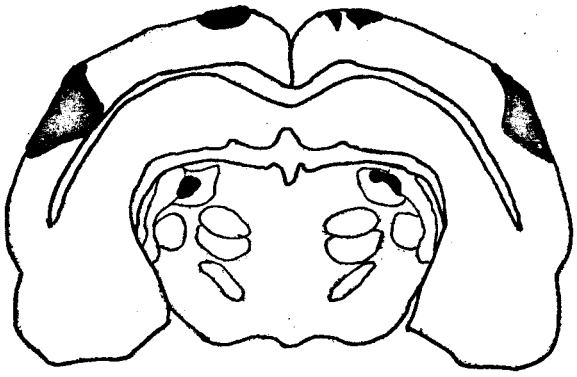

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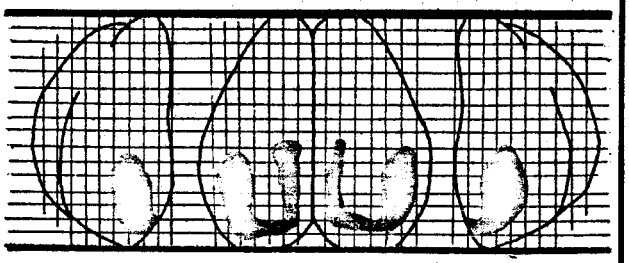

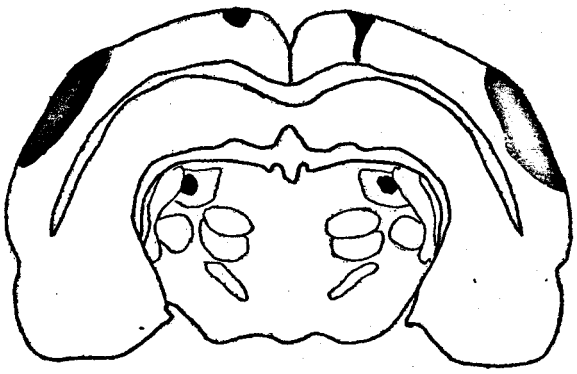

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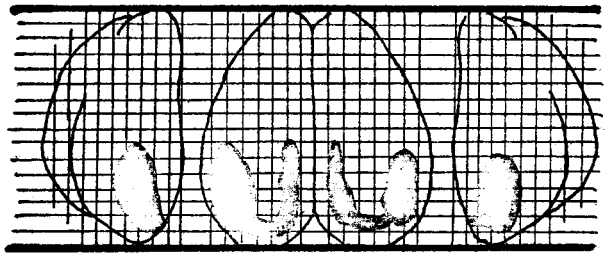

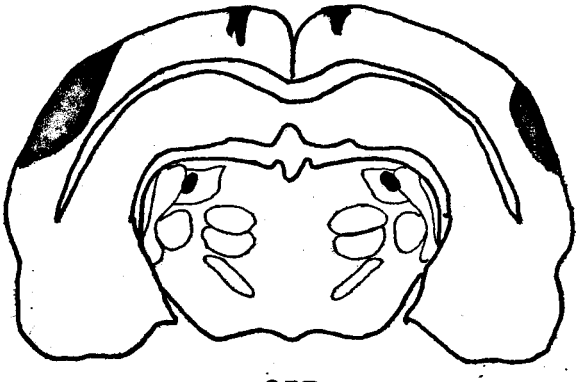

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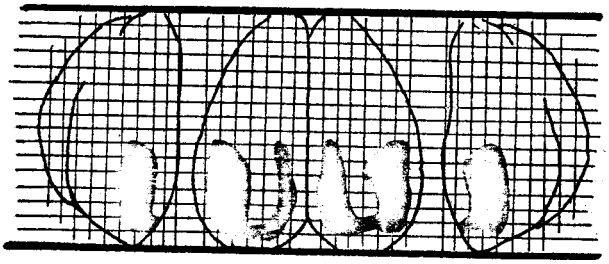

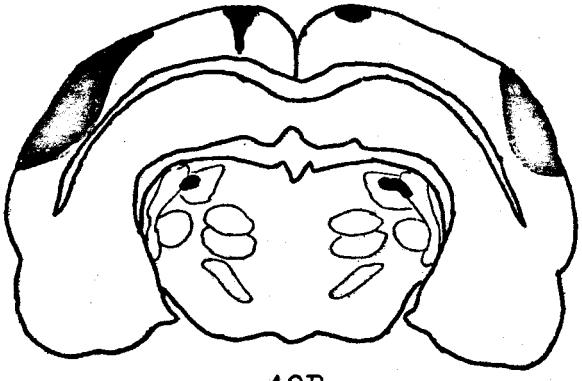

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APPROVAL SHEET

The Dissertation submitted by Stephen Milliser has been read and approved by members of the Department of Psychology.

The final copies have been examined by the director of the Dissertation and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the Dissertation is now given final approval with reference to content and form.

The Dissertation is therefore accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

May 17 1973
Date

Richard Maier
Signature of Advisor