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THE EFFECT OF CORTICAL LESIONS ON AUDITORY AND VISUAL DISCRIMINATION BEHAVIOR IN MONKEYS [1]

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Introduction

There has been much recent interest in the sensory specific functioning of the posterior "association" areas of the monkey cerebral cortex (Blum, Chow and Pribram, 1950; Chow and Hutt, 1953). A number of studies have offered evidence that subdivisions of the parieto-temporo-preoccipital cortex (PTO) play sensory-specific roles in discrimination behavior. Several studies have reported that the inferior temporal lobe functions uniquely in visual discrimination behavior (Mishkin and Pribram, 1954; Chow, 1951, 1954a). Still other studies have reported that the posterior parietal area functions uniquely in somesthetic discrimination behavior (Pribram and Barry, 1956; Wilson, 1957; Ettlinger and Wegener, 1958; Ettlinger, Morton and Moffett, 1966). An attempt to delineate a similar portion of the PTO cortex that is uniquely involved in auditory discrimination behavior resulted in a failure to clearly demonstrate such a relationship (Weiskrantz and Mishkin, 1958). Later studies in our laboratory have yielded evidence which implicates the lateral surface cortex of the superior temporal gyrus in sound localizing behavior, but not in the type of auditory discrimination used in the study being reported here.

[1] This research was carried out while the author was a postdoctoral fellow of the National Research Council and the United States Public Health Service in the Laboratory of Neurophysiology, The Institute of Living, Hartford, Connecticut. Additional support for this research was received from the American Academy of Arts and Sciences, the Society of Sigma Xi and the United States Public Health Service (M-2459). During the writing of this report support was received from the National Science Foundation (NSF GB 2987) and the United States Public Health Service (NB 06587).

A number of studies have attempted to specify the nature of the involvement by comparing the visually guided behavior of animals having lesions of the primary visual projection system with the behavior of animals having removal of inferior temporal cortex. The general conclusion of the investigations is that the inferior temporal cortex is involved in visual discrimination behavior via intracortical connections with the primary visual system, possibly via transcallosal fibers (Mishkin, 1958; Ettlinger, 1959; Mishkin, 1962). The unique behavioral functions of the two systems are less clear, but the limited amount of evidence suggests that the primary visual system is more concerned with acuity functions while the inferior temporal system is more concerned with functions related to learning (Wilson and Mishkin, 1959; Weiskrantz and Cowey, 1963).

A question which naturally follows these findings asks whether the PTO cortex as a whole plays a role in the integration of sensory input from the separate sensory systems. The present study was designed to determine whether the PTO cortex has any unique role in the performance of discrimination tasks clearly dependent on both auditory and visual cues for correct solution. The question of a unique role is both an anatomical and behavioral one — unique as compared to the role of other areas of the cerebral cortex, sensory and nonsensory, and uniquely concerned with sensory integration as distinct from the known role of portions of the PTO cortex in visual discrimination behavior and the possible role of other portions in auditory discrimination behavior.

Methods

Subjects

The subjects in this experiment were 17 rhesus monkeys (*macaca mulatta*). All animals were preadolescent at the beginning of the experiment but all were adolescent before the end. The median length of time animals were subjects in the experiment was 18 months, with a range of 8 ½ months to 26½ months. All animals were untrained prior to the beginning of this experiment.

Apparatus

All training and testing was carried out in the two-choice discrimination apparatus shown in Figure 1. The monkey was restrained in a cubical cage (20 in. on each edge) and made its responses by reaching between the vertical bars making up the front wall of the cage. When in place in the testing apparatus, the test cage was surrounded by a plywood enclosure on the top and sides, and by a black curtain at the rear. The response required was a press against one of the two vertically hung response doors. The response doors were 18 in. apart and were hung behind 3 in. × 3 in. openings in a stationary vertical black panel. Following a correct response, the door, hinged from above, swung open and remained open to allow the monkey to retreive a shelled half peanut from a food well located behind and below the level of the door. Both food wells were baited

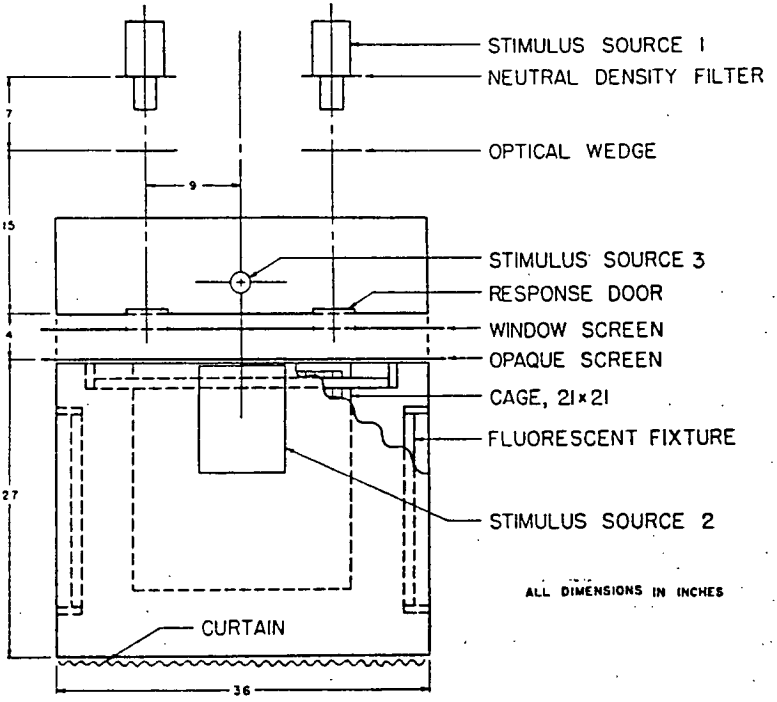

Fig. 1 — *Top view of the two-choice discrimination apparatus.*

and both doors were closed on every trial. The incorrect door was locked on each trial. Between trials the monkey was separated from the response doors by two vertically sliding screens. The screen closest to the monkey was solid plywood, while the other screen, also made of plywood, had windows which allowed full view of the response doors through clear plastic inserts but prevented responding. Between trials the monkey enclosure was only dimly illuminated with stray light from the projectors used to illuminate the stimuli. Patterned, colored or unpatterned plaques could be inserted in the hollow clear plastic and plate glass response doors for simultaneous visual discriminations. The doors were transilluminated from behind by separate 2 in. × 2 in. slide projectors.

Stimuli

The stimuli to be differentiated in the simultaneous visual discrimination tasks were contained in 3 in. × 4 in. translucent plaques. The patterns were solid black India ink drawings on onionskin paper and bound between two pieces of thin clear plastic. The color stimuli consisted of two sheets of colored translucent celluloid separated by ore or more sheets of onionskin paper and bound in plastic as already described. The amount of light transilluminating the response doors from the projectors was controlled by use of optical wedges and neutral density filters. The neutral density filters were contained in 2 in. × 2 in. slide mounts and were used only on the discrimination task which required that the two response doors be differentially illuminated.

The stimuli to be differentiated in the successive discriminations were presented from stimulus source 2 (Figure 1), stimulus source 3, and the flourescent lamps located inside the animal enclosure. Stimulus source 3 consisted of a 25-watt lamp mounted on the top of the apparatus. Stimulus source 2 was either a lamp box or a sound panel. The lamp box contained a 150-watt lamp and a 6-watt lamp and had a 6 in. × 8 in. milk glass window through which the animal enclosure could be illuminated by one or the other of the two lamps. The sound panel was a piece of masonite on which were mounted a 4 in. loudspeaker and a 6 volt buzzer. When in place, the lamp box or sound panel rested over an opening cut in the top of the animal enclosure and located 3 in. above the center of the testing cage. The

fluorescent lamps were three 20 watt blue tubes mounted in the positions indicated in Figure 1.

Behavioral tasks

All animals learned all of the following discriminations in the order in which they are here described. Prior to learning the discriminations each animal was trained to open the response doors and retrieve the peanut from the food well. In all cases the positive stimuli were counter-balanced between animals. Throughout all of discriminations the order of the correct response door was varied randomly (Gellerman, 1933).

1. SIM-1, a simultaneous visual discrimination between horizontal and vertical black on white stripes (1/4 in. wide). On every trial each response door contained one of the two stimuli and the animal was to learn to consistently choose one of the two stimuli. The level of illumination for both patterns was maintained at approximately 7-foot candles.[2]

2. SIM-2, a simultaneous visual discrimination between a field of dots (Zip-a-Tone pattern No. 83) and a black on white plus sign (arms 1 in. × 1/4 in.). The level of illumination at the two response doors was maintained at approximatey 11-foot candles for both patterns.

3. SUC-1, a successive go-left, go-right visual discrimination. On trials when the animal enclosure was illuminated by the fluorescent lamps the animal was to choose the response door to the right (or left) of the midline. On trials when the enclosure was illuminated by the 25-watt lamp (stimulus source 3) the door on the opposite side of the midline was correct. For this discrimination as well as for the two following successive discriminations the response doors contained unpatterned plaques. The illumination level via the projectors and plaques was maintained at approximately 15-foot candles at both doors. The average level of illumination inside the animal enclosure during trials when the fluorescent lamps were on was 17-foot candles. When the 25-watt lamp was turned on the average level of illumination was 0.1 foot candle.

4. SUC-2, a successive go-left, go-right visual discrimination. On trials when the 150-watt lamp (stimulus source 2) illuminated the animal enclosure the animal was to choose the response door to the left (or right) of the midline. On trials when the 6-watt lamp was on, the opposite door

[2] All illumination readings were made with a Weston Illumination Meter, Model 756 with Viscor filter.

was the correct one. The average level of illumination in the animal enclosure when the 150-watt lamp was on, was 15 foot candles. When the 6-watt lamp was on the average level was 0.6 foot candles.

5. SUC-A, a successive go-left, go-right auditory discrimination. On trials when the low frequency-high intensity tone (stimulus source 2) was sounding the response door to the left (or right) of midline was the correct one. When the higher frequency-lower intensity tone was sounding the opposite door was the correct one. The sounds used were generated by a saw-tooth wave generator amplified and delivered to the 4 in. speaker mounted on the panel placed above the center of the testing cage. The low frequency sound was 260 cps and was presented at an average sound level of 100.7 db (re 0.0002 dynes/cm^2). The higher tone was 1725 cps and was presented at an average sound level of 79.8 db.[3]

6. VVC, a visual-visual conditional discrimination. On each trial the animal had to make a selective response to either an illuminated triangle or circle.[4] When the surround of the two shapes was red, one of the shapes was the positive cue, and when the surround was green the other shape was the correct one. The level of illumination at both response doors for all shapes and colors was maintained at an average of 5 foot candles.

7. AVC, an auditory-visual conditional discrimination. On each trial the animal had to make a selective response to one of two levels of illumination at the response doors. On each trial one response door was maintained at a level of 9.7 foot candles while the other was at a level of 1.0 foot candle. On those trials when a buzzer was sounding, one of the two brightnesses was the positive visual stimulus. On trials when the buzzer was not sounding the other level of brightness was the positive visual cue.

Sixteen of the monkeys were trained on the following tasks postoperatively. One of the normal control animals died before it could be trained on these tasks.

8. SIM-C, a simultaneous color discrimination. On each trial the animal was presented with a choice between a blue and a yellow plaque in the response doors. Intensity as a cue to correct responding was controlled

[3] The sound pressure values represent the averages of more than twenty readings taken from six different locations in the monkey cages. The ranges of readings were respectively 99 to 102 db and 74 to 84 db. All sound pressure readings were made with a General Radio Sound Level Meter, Type No. 1551A.

[4] The diameter of the circle was 1 in. The triangle was equilateral with a base of about 1 1/4 in. The areas of the circle and triangle were equal. Two sets of the different shape-color combinations were used in random order. The two sets differed in the amount of illumination they transmitted so that the intensity of the surround could not be used for successful performance of the discrimination.

by use of several blue and yellow plaques differing in the amount of light transmitted. These plaques were used in a randomly varied sequence.

9. VPC, a visual-pattern conditional discrimination. On each trial the animal had to make a selective response to an upright capital F or the same figure in an upside down orientation which can be visualized by rotating the F 180° in the plane of this page. When the stimuli were illuminated capital letters with a black surround, one of the letters was the positive cue. While on trials when the stimuli were black letters on an illuminated surround the other letter was the positive cue. With the plaques bearing black letters on an illuminated surround the average level of illumination at the response doors as 15 foot candles. When the plaques having illuminated letters on a black surround were in place the average level of illumination at the doors was 1 foot candle.

10. DA, a five-second spatial delayed alternation tasks. The response doors contained unpatterned plaques transilluminated at a level of 15 foot candles. On the first trial of each day's session both response doors were closed, unlocked and baited. After the free-choice trial which was always rewarded, the animal was required to alternate on successive trials between the doors to the left and right of the midline. If an error was made, correction trials were given until the correct response was made. The time between obtaining the reward following a correct response or making an incorrect response, and the raising of the screen to allow the next response was approximately 5 seconds.

11. RC, this task consisted of a series of 100 "no-cue" trials over a period of four testing sessions. The response doors contained unpatterned plaques transilluminated at a level of 15 foot candles. No known cues were given to indicate which of the response doors was unlocked on any given trial. The aim of this series of trials was to determine whether the very experienced monkeys had learned to make use of cues indicating the correct response door of which the experimenter was unaware.

Lesions and surgery

Four animals served as normal controls and underwent no surgical procedures. The cortical removals were in all cases bilateral and approximately symmetrical. Four animals underwent bilateral removal of parieto-temporo-preoccipital cortex (PTO). The area of the intended lesion is limited posteriorly by the lunate sulcus and anteriorly by the intraparietal sulcus. It extends to the dorsal edge of the hemisphere, and onto the ventral surface of the temporal lobe. Two of the monkeys survived a single-stage bilateral PTO removal and two others did not. Two additional animals underwent bilateral PTO removals in two stages separated by an interval of two weeks, during

which they were neither tested nor trained. Three animals underwent single stage bilateral removal of the prefrontal cortex (Frontal). The lesion was intended to be limited to the cortical projection of *n. medialis dorsalis*. It is limited posteriorly by the anterior banks of the superior and inferior limbs of the arcuate sulcus, the hypothetical extension of these limbs to the dorsal convexity of the hemisphere and the lateral orbital sulcus, and all lateral surface cortex anterior to these limits including the cortex in the depths of the principal sulcus.

Three animals sustained single stage bilateral removal of the primary auditory cortex and surrounding lateral surface temporal lobe and parietal lobe cortex (Auditory). The intended lesion was essentially the cortical area described by Pribram, Rosner and Rosenblith (1954) as being the area activated by auditory stimulation. It was to include the primary auditory cortex of the superior temporal plane, adjacent areas of the insula and parietal operculum, both banks of the inferior one-half of the intrapariental sulcus and the posterior bank of the inferior one-third of the central sulcus. Three animals received single stage bilateral removal of all striate cortex except the foveal representation (Talbot and Marshall, 1941). The anterior extent of the intended lesion stops short of the posterior bank of the lunate sulcus, and a rough triangle of cortex in the anterior and ventral portion of the lateral striate cortex was spared. On the medial surface the occipital-polar gyrus was stripped of cortex and the cortex of the banks and depths of the extreme and the calcarine sulci was removed.

All surgery was performed under aseptic conditions using barbiturate anesthesia. Animals had a minimum two-week recovery period following surgery. Two animals which received the large auditory area removal required longer recovery periods because the lesions invaded the face and upper extremity areas of the somatic sensory cortex. The efficiency of the motor behavior involved in reaching, grasping and eating was impaired. The recovery periods for these two animals were respectively 17 days and 21 days.

Procedure

Individual training trials included the following sequence of events. The trial began when the opaque vertical screen was raised followed in approximately 5 seconds by the raising of the window

screen. The trial was terminated after the animal made an incorrect response or made a correct response and retrieved the peanut from the food well. Correction responses were never allowed and correction trials were given only in the delayed alternation task. However, correction trials on DA are not counted in determining the number of trial to criterion or failure. The problem of nonresponding within a few seconds following removal of the second screen was rare and not persistent. Rarely did the length of a trial exceed 30 seconds and usually it was over in half this time. The intertrial interval averaged 20 seconds for most of the discriminations. All correct responses were rewarded with a shelled half peanut. Animals received 30 trials per session, six sessions per week. They were trained to a criterion of 90 correct responses in 100 consecutive trials on all tasks except the Random Control task.

On the conditional discriminations (VVC, AVC, VPC) animals first learned to choose one of the visual stimuli under one of the conditional contingencies to criterion performance level. They then learned to choose the other visual stimulus under the second conditional contingency to criterion performance. Then followed a series of mixed trials in which the conditions determining which of the two visual cues was the correct one on any given trial were varied randomly. Mixed trial were given until each animal attained the criterion performance level.

After being trained to open the response doors and retrieve the peanut from the food well each animal learned behavioral tasks 1 through 7 as described above. They then underwent the already described surgical procedures followed by a two-week recovery period, or if designated as normal control animals received a two-week rest. Following the rest or recovery period, all animals were tested and, when necessary, retrained to criterion on the preoperative sequence of discriminations. On tasks which were failed postoperatively, animals received at least as many trials postoperatively as they had had preoperatively. Following completion of testing on the preoperatively learned tasks most of the animals were trained on behavioral tasks 8 through 11. Upon completion of task 11 animals were anesthetized the brains were perfused with saline followed by 10% Formalin, the brains were removed from the skull dehydrated and embedded in celloidin.

Histology

Celloidin sections were cut at 50 microns and four sections were saved from each ten sections. For all brains one set of sections was stained with thionin, and for most brains the adjacent sections were stained with hematoxylin. The lesions were reconstructed from enlarged tracings of the microscopic sections, and the degeneration in the thalamus was plotted by the usual techniques.

Results

Anatomical

A systematic series of stained section tracings, cortical surface reconstructions, and sketches of thalamic degeneration for one animal from each lesion group are presented in Figures 2 - 5. Significant deviations from the brains illustrated will be noted where appropriate. The cortical lesions for each animal were reconstructed from enlarged tracings of every twentieth 50 micron section. The surface reconstructions illustrate the extent of the cortical removals, and the section tracings the depth of the removals. Thalamic degeneration determinations where made after inspection of every fifth or tenth stained section, although the illustrations do not include all the sections inspected. The thalamic degeneration depicted is limited to the nuclear masses corresponding to the intended cortical lesions. Occasionally there was cortical damage resulting in degeneration in other nuclei. These degeneration effects will be noted in the text. The identifying abbreviations for the thalamic nuclei follow the nomenclature suggested by Olszewski (1952).

Striate lesions. Figure 2 illustrates the cortical lesion and consequent thalamic degeneration for monkey 330. The pattern and extent of medial surface removal was very similar for all three of the animals in this group. The lateral surface striate cortex removal in monkey 330 was significantly smaller than that in the other animals of this group. In the latter two animals the cortical removals came closer to the lunate sulcus, and spared a smaller triangle of ventro-anterior striate cortex. Thalamic degeneration in the brains of the monkeys not illustrated was more extensive than in monkey 330. The density of gliosis was higher and degeneration was present in those portions

of the lateral geniculate nucleus which were spared in sections 470-510 of monkey 330.

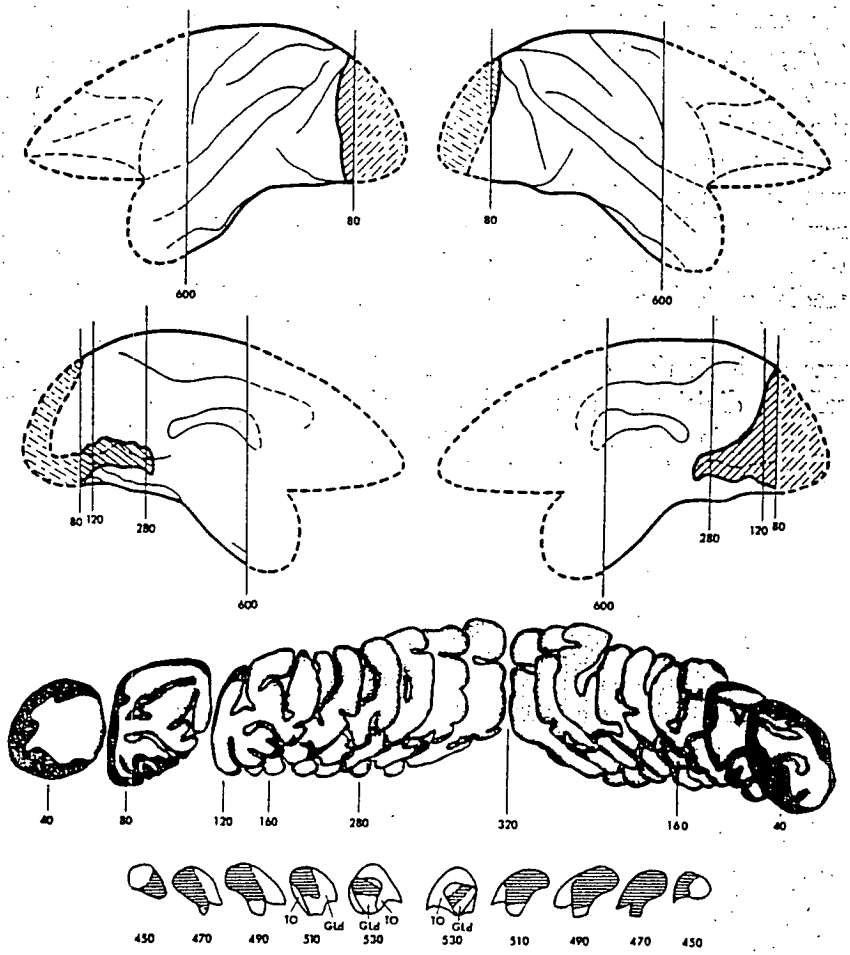

Fig. 2 — *Reconstruction of the cortical removal, representative sections and extent of thalamic degeneration for monkey 330. Cross hatching in cortical reconstructions and thalamic nuclei indicate extent of cortical removal and resultant thalamic degeneration in nucleus of primary interest.* Abbreviations: TO - *optic tract;* GLd - *lateral geniculate nucleus;* MD - *dorsal medial nucleus;* Pcn - *paracentral nucleus;* Cif - *central nuclei;* Pul.i,l,m - *inferior, lateral, medial pulvinar. (In Figures 2-5 the degeneration is plotted on nuclear outlines drawn at a scale approximately 2¼ times that for the reconstructions and sections.)*

Auditory lesions. Figure 3 depicts the cortical lesion and resultant thalamic degeneration for monkey 294. The lateral surface portion of

the lesion in this monkey included 100% of the intended cortex in both hemispheres. The primary auditory projection area on the superior temporal plane extends from the hypothetical downward extension of the central sulcus for about 10 mm in a dorso-caudal direction (Wegener, 1964b). In the monkey brain illustrated approximately 20% of the projection area was spared in both hemispheres. Much of the superior temporal plane cortex anterior to the primary projection area was also spared. In the other two animals of this group the removal of the projection area cortex was more nearly complete. The resultant thalamic degeneration was similar for all three monkeys. Most of the degeneration was in the parvocellular division of the medial geniculate nucleus. In all cases there was little or no degeneration in the most caudal fourth of the nuclei. In each monkey there was an indication of some degeneration in portions of the magnocellular division. In two of the animals there was degeneration extending into the suprageniculate nucleus. In all three animals there was bilateral degeneration in the pulvinar.

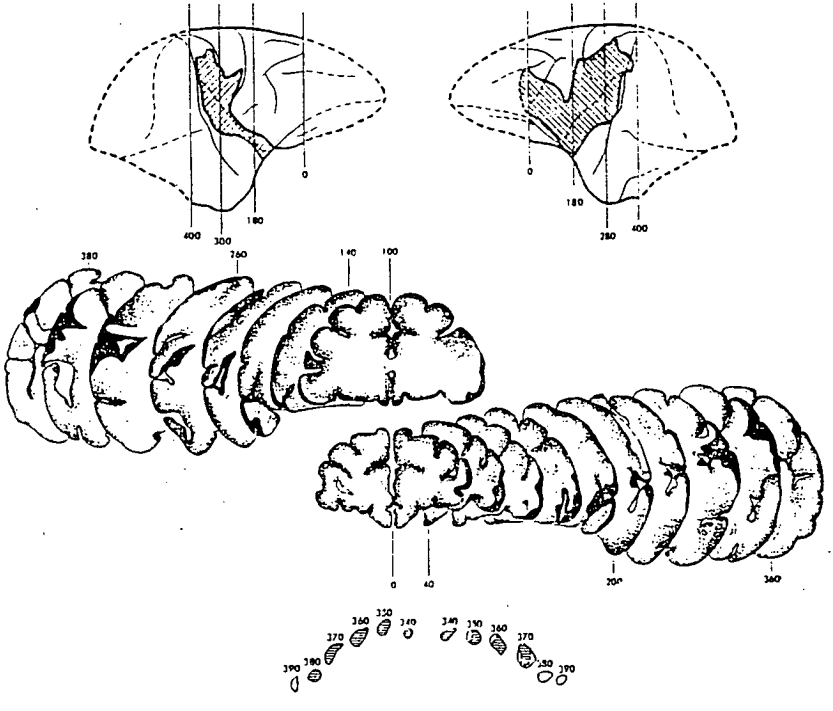

Fig. 3 — *Reconstruction of the cortical removal, representative sections and extent of thalamic degeneration for monkey 294. The thalamic nucleus shown is the medial geniculate nucleus. (See Figure 2 legend for key.)*

Frontal lesions. Figure 4 illustrates the extent of cortical removal for one of the animals in this group (# 339). For all the monkeys in this group the lesions were approximately as intended. Only in the left hemisphere of monkey 339 did the cortical removal extend posterior to the arcuate sulci. This latter removal was rather superficial in contrast to the depth of the lesion anterior to the sulci. The extent and pattern of thalamic degeneration was very similar in all the monkeys of this group. Throughout the rostro-caudal extent of medialis dorsalis the degeneration was restricted to the parvocellular division of the nucleus. There was evidence of degeneration in nucleus paracentralis in two of the monkeys.

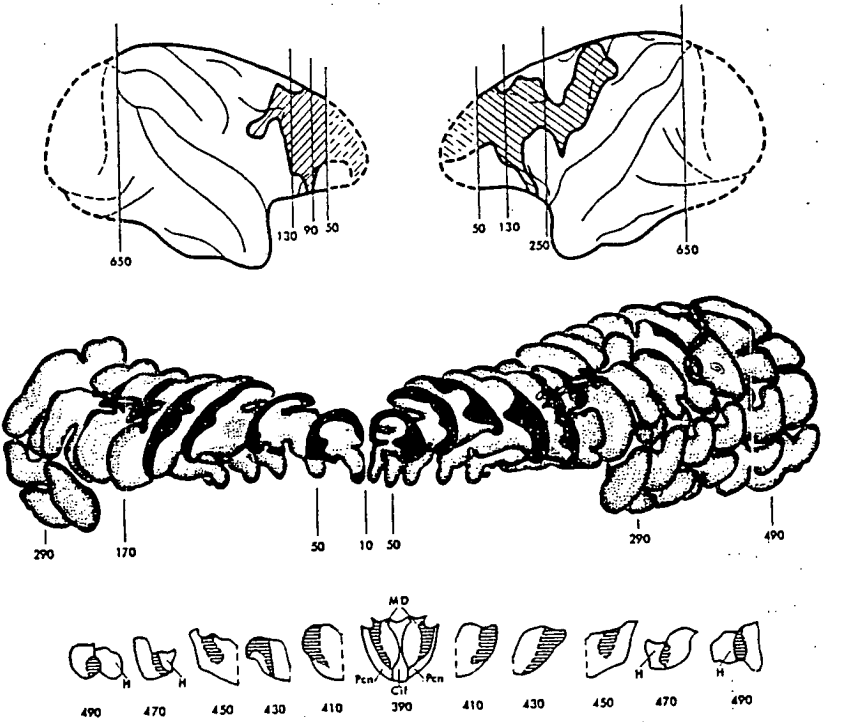

Fig. 4 — *Reconstruction of the cortical removal, representative sections and extent of thalamic degeneration for monkey 339. (See Figure 2 legend for explanation of abbreviations and key.)*

PTO lesions. Figure 5 shows the cortical lesion and resultant thalamic degeneration for monkey 293. The cortical lesion in this animal includes over 90% of the area of the intended lesion, sparing

ıly a small region of the anterior marginal gyrus in the left hemisphe-
:. For the other three animals of this lesion group the lateral surface
sion ranged from 75% to 95% of the intended removals. In five
: the six hemispheres not illustrated, there was a sparing of the
ıterior third of the inferior temporal gyrus and in two of the six
:mispheres sparing of portions of the anterior marginal gyrus. In
ıe of the four monkey brains there was complete sparing of the
:ntral surface of the temporal lobe. In three of the four animals of
ıis group the cortical removal extended into the striate cortex,
ıually the damage was limited to the dorsal third of the most anterior
ıird of the occipital lobe.

The thalamic degeneration in the pulvinar was usually greatest
: the dorso-lateral portion of *pulvinaris inferior* and ventro-lateral
ılvinaris lateralis. There was also some degeneration in *pulvinaris*
edialis but it tended to be less profound than that located more
terally and ventrally, and was less consistent from animal to animal.
ı the three monkeys having cortical lesions which invaded the
riate cortex there was degeneration in the medial and central sectors
: lateral geniculate nuclei, involving all layers.

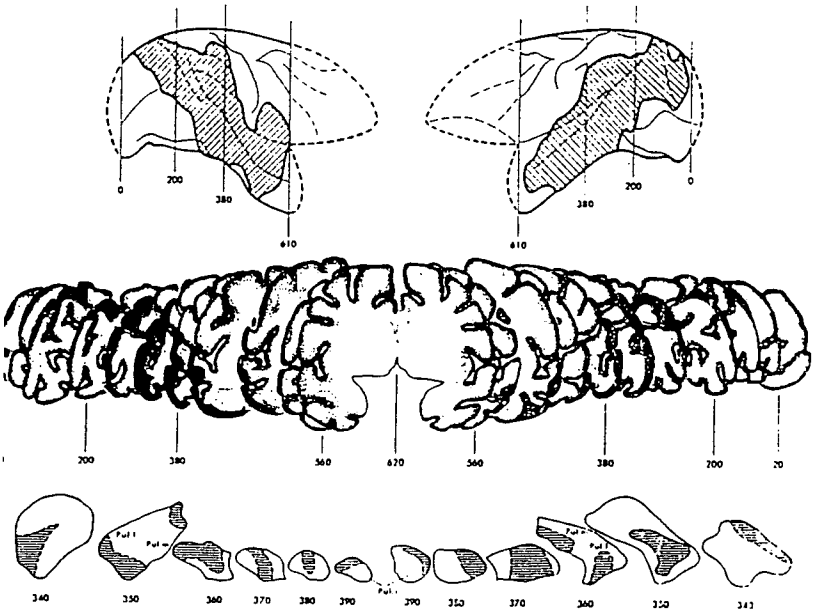

Fig. 5 — *Reconstruction of the cortical removal, representative sections and extent thalamic degeneration for monkey 339. (See Figure 2 legend for explanation of breviations and key.)*

Behavioral

The preoperative learning data are summarized in Table I. These data give a rough indication of the relative difficulty of these discrimination tasks when learned in the sequence used in this study. The relative level of difficulty for SIM-1, SUC-2 and SUC-A is similar to that obtained in another study when the remaining tasks were not included in the sequence (Wegener and Stamm, 1966). The range is included to indicate the wide individual differences which the lesion effect must override.

TABLE I

Preoperative Learning

	Number of Trials and Errors to Criterion													
	SIM-1		SIM-2		SUC-1		SUC-2		SUC-A		VVC		AVC	
	T	E	T	E	T	E	T	E	T	E	T	E	T	E
(N=17)														
Mean	88	41	18	13	38	20	160	65	856	376	511	194	1382	573
Median	80	39	10	11	40	22	100	41	790	421	380	145	1090	472
Range Low	10	11	0	1	0	5	0	5	343	188	203	84	550	252
Range High	220	106	90	36	80	29	690	272	1540	585	1880	660	3373	1541

The postoperative performance of the animals is summarized in Table II. The entries in this table are savings indices based respectively on trials to criterion performance and errors through criterion. In both cases the savings index (S. I.) is calculated by subtracting the postoperative score from the preoperative score and dividing this difference by the sum of the pre- and postoperative scores. This measure of the relative effect of the lesions was used as an attempt to attenuate the effects of interindividual differences in discrimination learning and performance for purposes of postoperative comparison.[5]

The differences between savings indices for the different lesion

[5] Animals were not matched for preoperative learning ability. Because of inter-problem learning variability it would have been very difficult to justify any of the possible bases for forming matched groups. There was overlapping of number of trials to criterion for each group with every other group on all preoperative discrimination tasks.

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groups were evaluated by use of the Wilcoxon sums test (one-tailed). Each lesion group was separately assessed against every other group of animals, normals as well as brain-damaged. Separate analyses were made for number of error and number of trials. In most cases when significant differences were revealed by use of one measure they were also revealed by the other. Exceptions to this statement will be noted.

It should be noted that in most cases the level of significance of the differences between groups was limited by the small number of animals in each group. Thus it was possible to attain a significance level of .025 between the normal control and the PTO groups (N= 4), while it was impossible to attain a level of less than .05 when any of the other groups (N = 3) was one of the two being assessed.

SIM-1. The animals with PTO lesions exhibited a significant relearning deficit on this task compared to both normals (p= .025) and animals with lesions elsewhere in the brain (p= .05). The analysis by errors revealed that the frontal operates made significantly more errors than the normal controls (p= .05).

SIM-2. The analysis in terms of trials to criterion did not reveal any significant differences in retention performance between any of the groups of animals. The animals with auditory lesions tended to make more errors than the normals (p= .10) and the PTO animals made significantly more errors postoperatively than normal controls (p= .025).

SUC-1. The analysis by trials to criterion showed that animals with frontal lesions required significantly more trials than normal control animals (p= .05). In addition, animals with frontal lesions tended to require more trials than animals with lesions of the auditory cortex (p= .10). The error analysis reveals the same significant differences between animals with frontal lesions and animals with auditory lesions as well as with normal controls. The animals with PTO lesions had a significantly higher error score than normals (p= .025), as did also the group of animals with auditory lesions (p= .05).

SUC-2. Both the analysis by trials and that by errors reveal that the animals with PTO lesions exhibited significant relearning deficits compared to the normal control animals (p= .025) and as compared to each of the other brain-damaged groups (p= .05).

SUC-A. The pattern of significant differences is the same for both trial and error analyses on this task. Animals with frontal or auditory area lesions performed more poorly postoperatively than the normal control animals ($p = .05$), as did also the animals with PTO lesions ($p = .025$). In addition, the animals with auditory lesions showed a greater deficit than each of the other brain-damaged groups ($p = .05$).

VVC. The analysis by trials revealed that normal control animals required fewer trials to reattain criterion performance levels than animals with striate, auditory, or frontal lesions ($p = .05$) as well as fewer than those with PTO lesions ($p = .025$). The PTO animals tended to require more trials to reattain criterion (or failed to do so) than animals with frontal lesions ($p = .10$) and required significantly more trials than did animals with lesions of the auditory cortex ($p = .05$). The analysis by errors revealed the same pattern of tendencies and significant differences as that indicated for trials. In addition, the error scores for animals with auditory area lesions tended to be higher than those of the normal control animals ($p = .10$).

AVC. Both trial and error analyses showed that animals with auditory lesions performed more poorly than the normal controls and the frontal animals ($p = .05$). They also tended to require more trials to reattain criterion levels of performance than did animals with striate lesions ($p = .10$). Animals with PTO lesions tended to require more trials and make more errors post-operatively than the normal control animals ($p > .05 < .10$).

The postoperative learning data obtained from 16 of the monkeys are presented in Table III and can be summarized as follows. All normal and brain-damaged animals learned discrimination SIM-C in fewer than ten trials and with an average of 6 errors. There was no difference in speed of learning between any of the animal groups. The visual pattern conditional discrimination (VPC) proved to be a difficult discrimination for both normal and brain-damaged animals. The mean number of trials to criterion for the ten monkeys which learned this discrimination was 1357. Three of the four PTO animals failed to learn this task in 1500 or more trials. One of three animals in each of the frontal and striate animal groups also failed, the former received 1500 trials, the latter 4444 trials. Otherwise there were no gross differences in the learning rates of the animals from the different groups.

TABLE III

Postoperative Learning

	SIM-C T	SIM-C E	VPC-1[a] T	VPC-1[a] E	VPC T	VPC E	DA T	DA E	RC Errors in 100 trials
Normals (3)						(2)[b]			
Mean	0	8.3	408	140	937	296	264	117	49
Median	0	8	430	150			390	121	50
Low	0	7	260	88	600	171	11	10	48
Range High	0	10	534	183	1275	422	390	219	50
Frontal (3)						(2)			
Mean	3	6.3	691	227	1062	368	530[d]	199	54
Median	1	4	494	201					54
Low	0	3	460	199	650	274			53
Range High	9	12	1120	275	1474	462			55
Auditory (3)						(3)			
Mean	0	5.3	603	224	1269	469	127	72	53
Median	0	5.7	590	206	1240	454	130	64	53
Low	0	4	390	175	870	365	70	56	51
Range High	0	6	830	290	1698	589	180	96	54
Striate (3)						(2)			
Mean	0	1.3	476	174	1442	462	228	83	52
Median	0	1	457	138			180	93	52
Low	0	0	200	99	840	315	144	60	48
Range High	0	4	770	285	2045	609	360	97	54
PTO (4)						(1)			
Mean	0	6	1260[c]	336[c]	2876	892	219	82	54
Median	0	7.5	—				216	81	53
Low	0	0	—				93	59	51
Range High	0	9					350	107	57

a - VPC-1. The first step in learning the complex visual pattern conditional discrimination.

b - The numbers in parentheses refer to the number of animals for which data is being reported; if it is different than the numbers in parentheses adjacent to the animal group labels, the number is given in the body of the table.

c - Three animals failed to learn VPC-1 in 1500 trials each.

d - Two frontals failed to learn in over 1000 trials each.

The spatial delayed alternation task was failed by two of the three frontal animals in over 1000 trials. The third frontal animal learned to perform this task in 530 trials. This performance, while unusual for a frontal, required more trials than those needed by any of the thirteen normal and brain-damaged which also learned this task.

There were no significant differences in rate of learning this task between any of the other groups of animals.

The performance of all animals on the Random Control series of trials was not different than what would be expected on a chance basis (mean number of errors = 52; range 48-57). However, all animals with brain lesions made significantly more "errors" in 100 trials than did the normal control animals (p. = .05).

Discussion

The preoperative learning data cannot be compared directly with those obtained in other studies because of several features of the procedure used in the present study. Compared to the studies using the Wisconsin General Testing Apparatus (WGTA) the primary differences were in the use of transilluminated visual stimuli instead of plaques; the use of response doors containing the stimuli rather than plaques to be displaced; and the use of an additional (window) screen separating the animal in the testing cage from the response manipulanda. The effect of these differences in apparatus cannot be assessed for most of the discriminations used in this study (SIM - 1, SIM-2, SUC-A) since comparable tasks have not been reported for the WGTA. However, there are some roughly comparable data for tasks VVC and AVC. In all cases the training procedures and criteria used, differed from those used in the present study. For VVC the number of trials and errors to criterion in a number of studies (Blum, Chow and Pribram, 1950; Chow, 1951, 1952) were of the same order of magnitude as those reported for the seventeen monkeys of the present study. Several studies report learning scores on an auditory-visual conditional task (Evarts, 1952a, 1952b; Chow, 1954). The number of trials and errors to a higher performance level (i. e., criterion) for the monkeys of the present study were consistently lower than those reported in the other studies. This difference may be due solely to the sequence of steps in the training procedure rather than to the differences in the apparatus or cues used.

Striate lesion. The pattern of postoperative performance of the animals with this lesion was for the most part consistent with predictions based on earlier studies of the effects of partial lesions of the striate cortex (Klüver, 1937; Lashley, 1939; Settlage, 1939; Hahn, 1952). On the simple pattern, color, and level of illumination

discriminations these animals performed as well as did the normal control animals. These animals did have difficulty reattaining criterion levels of performance on the visual-visual-conditional discrimination. This complex visual discrimination had a number of different aspects, any one of which may hold the key to the deficient postoperative performance. It required that the animal make a shape discrimination, a color discrimination, a simultaneous discrimination, a successive discrimination, and a conditional response. It is also the most difficult visual discrimination which the animals were required to learn preoperatively.

The animals with striate lesions performed as well as the normal control animals on the preoperatively learned pattern discrimination (SIM-1 and SIM-2). In addition, all three striate animals learned the very difficult F versus F pattern discrimination which made up the first phase of the visual pattern conditional discrimination (VPC) as rapidly as did the normal control animals. Postoperatively the striate animals learned the blue-yellow color discrimination (SIM-C) as rapidly as did the normal control animals. The normal performance on such pattern and color discriminations shows that these animals were able to perform simultaneous shape and color discriminations. The postoperative performance of the striate animals on SUC-1, SUC-2 and SUC-A was not different from that of the normal controls. These findings suggest that the deficit on VVC was not due to impaired ability to make successive discriminations in general, or successive visual discriminations more specifically. The normal post-operative performance of the animals with striate lesions on the auditory-visual conditional discrimination suggests that the deficit on VVC is not attributable to either a decrease in ability to perform a conditional response in general, or to the general level of difficulty of the discrimination.

These findings indicate that the deficit is not due to a deficiency in sensory discrimination behavior or the ability to make the responses required for correct solution of VVC. Rather they point to a specifically visual deficit, and one which would appear to be related to the integration of two different types of visual input. This notion is supported by the performance of the animals on VPC. Although the striate animals learned the initial pattern discrimination as rapidly as normal control animals their performance on the task as a whole tended to be inferior to that of the normal controls. Whether this difficulty was due to the discrimination reversal aspects of the

conditional response, or to some other kind of visual integration is impossible to decide on the basis of the data of this study. King, Roberts and King (1963) in a brief report noted that squirrel monkeys with partial bilateral lesions of the striate cortex developed a discrimination reversal learning set as rapidly as normal control animals, while monkeys with prestriate lesions were significantly retarded in the formation of such sets.

It seems reasonable, on the basis of the foregoing analysis, to conclude that the striate cortex has at least two types of functions: 1) a sensory receptive function; and 2) a function best described as concerned with the integration of visual inputs of different kinds.

Auditory lesion. The animal in this lesion group displayed a significantly poorer performance than the normal control animals only on the two tasks requiring auditory discriminations, SUC-A and AVC. That the difficulty on SUC-A was not with either the successive nature of stimulus presentation or the conditional nature of the response is indicated by the essentially normal performance of these animals on SUC-1 and SUC-2. The deficient postoperative performance of these animals on AVC is also probably a simple auditory deficit. These animals exhibited essentially the same type of performance as that given by normal control animals on simultaneous visual discriminations (SIM-1, SIM-2, SIM-C), successive visual discriminations (SUC-1, SUC-2), a visual-visual conditional task (VVC) and the visual pattern conditional task (VPC). This latter task was of about the same difficultly level as the AVC task. These control results suggest that poor performance on AVC was probably a consequence of an auditory discrimination deficit.

The nature of this auditory deficit has been analyzed elsewhere (Wegener, 1964). We can summarize that analysis in the following way. There is some evidence that the auditory cortex functions in auditory discrimination tasks not as a locus for primary detection, analysis and interaction, but rather by: 1) the mediation of the attention-getting properties of auditory stimuli; 2) the integration of the appropriate response to the auditory input; 3) the organization of auditory space; *or* 4) the interpretation of temporal sequence of auditory cues. The evidence does not allow a clear choice or elimination of any of these hypotheses, but it does favor an interpretation related to the interpretation of temporal sequence of auditory cues.

Frontal lesion. The pattern of deficits exhibited by the animals

with this lesion is both more extensive and involves a greater variety of tasks than that of either of the two lesion groups considered so far. The most useful comparisons are those to be made with the normal control animals. The frontal animals were significantly deficient compared to the normal control animals on discriminations SUC-1, SUC-A, VVC and DA.

Pribram and Mishkin (1955) reported that monkeys with bilateral antero-frontal lesions performed as well as normals on a successive conditional pattern discrimination which shared with SUC-1 the requirement of a response to the left or right of midline depending on which of two cues was displayed on any given trial. Whether the contradiction between the result reported by Pribram and Mishkin and the data of the present study is due to some of the differences in the details of the two discriminations, or some aspect of the training history of the animals is impossible to determine. Some recent data (Wegener and Stamm, 1966) in which the stimulus and response conditions were almost identical to those used in the Pribram and Mishkin study, also revealed that animals with bilateral frontal lesions have a significant deficiency in the ability to perform such simple successive, go-left, go-right discriminations. Ettlinger and Wegener (1958) reported data on the deficient performance of frontal operates on a test of spatial orientation which required a go-left, go-right response. The deficit is not a simple visual discrimination deficit since these animals performed as well as normals on SIM-1, SIM-2, SUC-2, SIM-C and VPC. All but one of these visual discrimination tasks is more difficult than SUC-1. Lashley (1948, 1950) has stated that lesions of the prefrontal cortex, as well as other regions, disrupt the ability of animals to perform conditional reactions. SUC-1 is a simple visual-motor conditional task, i. e. if the fluorescent lamps are on, go left (or right), if the incandescent lamp is on go to the door on the opposite side. If the deficit is one in ability to perform a conditional reaction it recovers rapidly, since the frontal animals performed as well as normal control animals on SUC-2, another visual-motor conditional task which followed SUC-1 in the testing sequence.

There have been a number of reports of deficits in the performance of a go, no-go auditory discrimination task for monkeys with frontal lesions (Weiskrantz and Mishkin, 1958; Gross and Weiskrantz, 1962; Battig, Rosvold and Mishkin, 1962; Gross, 1963; Symmes, 1968). In addition, Blum (1952) reported deficits following frontal ablations on an auditory quality-localization discrimination task. King,

Roberts and King (1963) reported deficient performance by squirrel monkeys with frontal lesions on a pitch-localization task. The deficit found in the present study on SUC-A adds another discrimination situation in which frontal animals display what appears to be an auditory deficit. The evidence has been assessed elsewhere in an attempt to specify the relationship between the frontal lobes and auditory discrimination behavior (Wegener, 1964). In summary it is not clear that the frontal deficit on auditory discrimination tasks is uniquely auditory or specific to the frontal lobes. If it is both of these, the evidence does not allow us to state whether the deficit is related to a perceptual factor such as attention, or to a deficiency related to the auditory response behavior of monkeys.

A failure to relearn a visual-visual conditional discrimination task has been reported for one frontal monkey by Warden, Barrera and Galt (1942). Other studies (Lashley, 1948; Chow, 1952) reported postoperative savings on such a discrimination following bilateral removal of the frontal eye fields. Wade (1952) reported significant postoperative deficits on a visual-visual conditional task following frontal lobectomy, lobotomy, or circumsection. The deficit on VVC exhibited by the animals in the present study cannot be attributed to an inability to perform the conditional response since prior to testing on VVC all animals had to relearn SUC-1, SUC-2 and SUC-A which are simple conditional tasks. And following the relearning of VVC all animals reattained criterion performance levels on AVC as rapidly as did the normal control animals. Nor can the deficit be related to overall task difficulty in any simple fashion since AVC is much more difficult than VVC, and SIM-1 is clearly more difficult than SUC-1. Earlier reports (Harlow and Dagnon, 1942; Brush, Mishkin and Rosvold, 1961) had established that monkeys with bilateral frontal lesions exhibited deficits in discrimination reversal learning. However, the deficient performance of the frontal monkeys in the present study on VVC cannot be traced solely to the discrimination reversal aspect of the problem. Rather the deficient performance is evidenced to about the same degree in the initial shape discrimination phase, the reversal phase and the series of mixed trials.

The pattern of failures within the sequence of tasks may hold a clue to the nature of the deficit. The deficits on SUC-1, SUC-A and VVC have a common feature in that each involved a definite change in either the manner of presentation, or type of stimuli to be discriminated, and in the cases of SUC-1 and VVC, a change in the nature

of the response requirements from the just preceding tasks. The successful retention performance of these animals on SUC-2 and AVC suggests that whatever deficiences are induced by the changes in method or mode of stimulus presentation and response requirement they are transient ones. It is as though the frontal animal is capable of adapting his behavior set to any mode of stimulus presentation or response requirements after an initial period of confusion. This confusion may be marked by a perseveration of the just preceding successful response, the maintenance of a stimulus preference, a reinforcement expectancy fixation, a regression to position habits, or some other unsuccessful mode of solution.

The suggestion of Mishkin (1964) that the failure of frontal operates on discrimination tasks can be traced to either the perseveration of central sets from just preceding experiences or from natural stimulus preferences does not fit all the data of the present study. In moving from SIM-2 to SUC-1 in this study, the mode of stimulus presentation is changed from simultaneous to successive and the stimuli to be discriminated are changed. This makes it difficult for the animals to exhibit any preference behavior either natural or acquired or as a carryover from SIM-2. It is also difficult to see how response perseveration could operate to cause the deficit on SUC-1. The motor response requirements are similar on both tasks, i. e. approach either the right or left door on each trial. Rather than the perseveration of a stimulus preference or a response set, the deficit is best described as a retarded rate of adaption to a changed manner of stimulus presentation.

The recent proposal reviving a modified short-term memory deficit hypothesis also has trouble explaining certain findings of the present study (Gross and Weiskrantz, 1964). It is difficult to understand how animals with non-functioning or inefficient short-term memory storage could learn both simple and difficult simultaneous visual discriminations as rapidly as normals. The postoperative learning of the yellow-blue color discrimination took place in all normal control and frontal monkeys in fewer than ten trials. And the rate of learning the initial shape discrimination portion of VPC was indistinguishable for the same two groups of animals.

The outcome of this analysis of the pattern of deficits displayed by with frontal lesions is unfortunately not an improvement over many previous conclusions. It suggests that the frontal deficit is neither solely a perceptual deficit nor a response deficit, but that it appears

in both aspects of discrimination performance (Wegener and Stamm, 1966).

PTO lesion. The region of the brain included in this lesion is the one of initial interest in this study. It is this area with its sensory specific subdivisions which may serve as a region mediating the integration of sensory inputs from the separate sensory systems. It also includes the cortical areas homologous to those in the cat for which polysensory convergence has been reported (Buser and Borenstein, 1959; Dubner and Rutledge, 1964). The focus of interest then is on the auditory-visual conditional discrimination task. The performance of the animals with this lesion tended to be poorer than that of the normal control animals, but it does not quite attain acceptable levels of statistical significance ($p > .05 < .10$). This failure to attain statistical significance can be attributed to the relatively poor performance of one of the normal control animals (# 386).

The question remains then whether the trend toward deficient performance of these animals on AVC is related to some kind of failure in intermodal integration or to other types of malfunction. The pattern of performance by these animals on the other tasks makes it appear unlikely that the difficulty on AVC is one of intermodal integration. The simultaneous visual brightness discrimination which makes up the visual aspect of AVC would appear to have been too simple to have been the sole cause of the deficient performance. These same animals did exhibit a deficient performance on the more difficult pattern discriminations (SIM-1 and VPC), however their performance on SIM-C indicates that they can perform as well as normal control animals on some simple simultaneous visual discriminations.

The deficit these animals exhibited on VVC does not clarify the nature of the deficit on AVC. One of the four PTO animals failed to relearn the first phase of VVC, a simple triangle versus circle discrimination. The two additional animals which failed to relearn this task, failed on the second phase of the task. This latter failure could have been due to deficient performance on either the discrimination reversal or color differentiation aspects of the task. Two earlier studies (Chow, 1951; Blum, Chow and Pribram, 1950) reported color discrimination deficits following either temporal lobe or PTO removals. However, the normal postoperative performance of the PTO animals in the present study on a yellow-blue color discrimination, with

intensity controlled, suggests that the failure on VVC was not due to difficulty with making the red-green color differentiation. Brush, Mishkin and Rosvold (1961) have reported that monkeys with inferior temporal removals are deficient in discrimination reversal learning. It should be noted here that the one PTO monkey which relearned VVC (239) had a bilateral lesion which spared much of the ventral surface temporal cortex reported to be involved in visual discrimination behavior. Thus it seems probable that the failure on VVC was related to a deficit in visual discrimination reversal behavior.

The mild deficiency on AVC may be due to: 1) deficient performance on the auditory aspect of the intermodal task, 2) the discrimination reversal aspect of the task, 3) depressed efficiency in the performance of successive discriminations, or 4) lowered efficiency in the performance of tasks involving conditionality. The essentially normal performance of the PTO animals on SUC-1 limits the latter two possibilities to tasks more difficult than SUC-1 in both their successive and conditional aspects. Three of the four PTO animals registered their poorest postoperative performance on the second phase of AVC. This phase involves both visual discrimination reversal and a simple auditory discrimination. In view of the deficient performance of these animals on SUC-A, and the already cited evidence concerning the difficulty monkeys with inferior temporal removals have with visual discrimination reversals, it seems likely that the deficient performance of the PTO animals of this study was due to one or both of these two types of deficits rather than to a failure in intermodal integration.

Several conclusions can be drawn from the discussion of the findings reported in this study:

1) The striate region of the monkey brain probably has in addition to a specific receptive function, a more general function related to the successful utilization of more than one source of visual input in the same discrimination task.

2) The primary auditory cortex plus surrounding regions of the brain are probably involved in both simple and more complex auditory discrimination behavior but are not essential for simple auditory tasks or for complex tasks the auditory aspect of which is simple.

3) The prefrontal region of the monkey brain probably operates to mediate a function related to behavioral flexibility or adaptability to changes in either conditions of stimulation or response requirements.

4) The PTO cortex as a whole does not appear to play a unique role in the integration of sensory input from the visual and auditory systems as required in the behavior of this study. Its overwhelming involvement seems to be in tasks requiring the use of visual input, especially in the integration of more than one source of visual information.

Summary

Seventeen rhesus monkeys were trained on a series of seven visual and auditory discrimination tasks. Thirteen of these animals then received bilateral lesions of one of the following areas of the cerebral cortex: primary auditory area plus surrounding cortex; prefrontal cortex; striate cortex sparing the foveal representation; parieto-temporo-preoccipital cortex. Four monkeys served as normal control animals. Following recovery from surgery all animals were tested and retrained on the preoperatively learned tasks. Animals then learned four additional tasks. The brains were prepared and studied to allow the reconstruction of the cortical lesion and plotting of the consequent thalamic degeneration.

The pattern of behavioral changes for each of the lesion groups was analyzed and possible functions were suggested for each region. None of the cortical areas studied exhibited any unique function in the integration of sensory input from more than one sensory system.

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Lesion Group		SIM-1 T[2]	SIM-1 E[3]	SIM-2 T	SIM-2 E	SUC-1 T	SUC-1 E	SUC-2 T	SUC-2 E	SUC-A T	SUC-A E	VVC T	VVC E	AVC T	AVC E
Normal (4)															
Median		1.00	.82	1.00	.91	.80	.47	1.00	.67	.86	.82	.89	.82	.88	.63
Range	Low	1.00	.68	1.00	.75	.50	.27	1.00	.25	.77	.78	.73	.74	.53	.44
	High	1.00	.86	1.00	1.00	1.00	.70	1.00	1.00	.91	.84	1.00	.91	.94	.90
Frontal (3)															
Median		.97	.61[b]	1.00	.77	.31[b]	.14[b]	.40	.60	.70[b]	.72[b]	.59[b]	.49[b]	.74	.69
Range	Low	.20	.19	1.00	—.05	.14	—.08	.23	.24	.53	.55	—.25	—.17	.52	.46
	High	1.00	.63	1.00	.87	.40	.25	1.00	.85	.76	.78	.70	.51	.89	.84
Auditory (3)															
Median		.90	59	1.00	.71	.38	—.23[b]	.97	86	.01[b,c]	.09[b,c]	.74[b]	.73[b]	.19[b,d]	.33[b,d]
Range	Low	.69	.50	1.00	.33	—1.00	—.30	.37	.35	—.64	—.65	—.68	—.54	—.25	—.20
	High	1.00	.91	1.00	.76	1.00	—.03	1.00	.93	.41	.48	.82	.76	.34	.37
Striate (3)															
Median		.97	.57	.97	.36	.97	.29	.97	.67	.81	.81	—.27[b]	—.15[b]	.80	.69
Range	Low	.78	.22	.78	—.33	—.58	—.34	.76	.54	.66	.65	—.67	—.66	.21	.28
	High	1.00	.91	1.00	1.00	1.00	.48	1.00	.82	.87	.85	.71	.51	.85	.77
PTO (4)															
Median		—.73[a,e]	—.70	.75	.22[a]	.23	.04[a]	—.35[a,e]	—.28[a,e]	.63[a]	.70[a]	—.97[a,e]	—.64[a,e]	.22	.25
Range	Low	—1.00	—.89	—1.00	—.95	—.59	—.50	—.64	—.53	.46	.35	—1.00	—.76	—.68	—.60
	High	—.06	—.07	1.00	.36	1.00	.25	.16	—.09	.73	.71	.53	.65	.68	.59

1 - S.I. = $\frac{\text{Pre} - \text{Post}}{\text{Pre} + \text{Post}}$

2 - Based on # of trials excluding 100 criterion trials

3 - Based on # of errors including 100 criterion trials

Significantly different from: a - normals p = .025; b - normals p = .05; c - auditory p = .05; d - frontal p = .05; e - all other lesion p = .05.

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