NON-EQUIPOTENTIAL CORTICAL FUNCTION IN MAZE LEARNING

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These experiments were designed to test the theory of equipotential cortical function in maze learning under conditions in which sensory cues are restricted to those arising from the animal's movements through the maze. Lashley's theory of equipotentiality rests on the assumption that the sensory projection-areas have no critical *sensory* function in the habit.[1] Lashley and Ball,[2] when they discovered that cervical spinal lesions which disturbed proprioceptive and tactual sensibility had no effect on, performance in a maze previously learned, concluded that the habit was completely independent of differential sensory stimulation. This conclusion was possible in view of evidence existing at that time which indicated that sensory cues from any modality other than the proprioceptive were unnecessary for retention of the maze habit.[3] It appeared then that maze-running is directed solely by some central nervous mechanism—that sensory stimulation elicits the initial response of running and determines the locus, but not the direction of turns in the maze. It was this autonomous central mechanism which Lashley thought he had interfered with when he found that lesions of the cortex retarded maze-learning and interfered with retention. Since no particular area of the cortex appeared to be more important than any other, he concluded that the various parts of the cortex are equipotential in their contribution to the central mechanism which controls the maze-habit.

Hunter criticized this view on the ground that the maze-habit had not been proven to be independent of sensory control.[4] Carr's experiments had shown that rotation of the maze after or during learning disrupts directional auditory cues.[5] Hunter's failure to obtain double alternation in his temporal maze meant that the rat could

* Accepted for publication August 23, 1951. This study, performed in the Psychological Laboratory of Brown University was directed by Professor W. S. Hunter.

[1] K. S. Lashley, *Brain Mechanisms and Intelligence,* 1929, 1-186.

[2] Lashley and J. Ball, Spinal conduction and kinesthetic sensitivity in the maze habit, *J. Comp. Psychol.,* 9, 1929, 71-106.

[3] J. B. Watson, Kinaesthetic and organic sensations: Their role in the reactions of the white rat to the maze, *Psychol. Monog.* 8, 1907, (no. 33), 1-100.

[4] W. S. Hunter, A consideration of Lashley's theory of equipotentiality of cerebral action, *J. Gen. Psychol.,* 3, 1930, 455-468.

[5] H. A. Carr, Maze studies with the white rat: I. Normal animals, *J. Anim. Behav.,* 7, 1917, 259-275; II. Blind animals, *ibid.,* 7, 1917, 277-294.

not learn a series of responses without differential stimuli for each response.[6] It seemed likely from the evidence that all sensory modalities contributed about equally to the control of maze performance, and Hunter suggested that a lesion in any sensory projection area of the cortex would produce about the same deficit in sensory function, giving the appearance of equipotentiality.

Lashley's rebuttal re-emphasized the sensory controls in his experiments,[7] one of which is particularly significant. When *blind* rats learned a maze, subsequent removal of occipital cortex produced serious deterioration of the habit. It is evident from this result that the visual area of the cortex has an important nonvisual function in retention of the maze habit. Lashley maintained that equipotentiality holds for the non-sensory function of sensory areas.

Later experiments on sensory control of the habit supported Hunter's theory of multiple control. Casper showed that the surgical elimination of *either* visual or olfactory receptors produces no deficit in the retention of a previously acquired maze-habit, a significant deficit follows the elimination of both.[8] This finding indicates that sensory control is multiple. In a control experiment Casper found that rotating the maze and interchanging its units between trials produced the same level of performance in normal animals as did the use of the stationary maze with rats who were blind, deaf, and anosmic. Honzik found that rotation of the maze disturbs directive auditory cues from outside and impairs performance when the influence of vision is decreased (as when the rats are blinded or an alley-maze is used).[9]

Other evidence indicates that lesions of dorsal spinal tract have little effect on behavior cued to proprioceptive stimulation; this evidence invalidates the assumption of Lashley and Ball that they were eliminating proprioceptive cues by making such lesions. It is probable that proprioceptive cues are routed around these lesions. Ghiselli was unable to find any changes in the rat's discrimination of inclined planes after dorsal hemisection of the spinal cord.[10] Brown obtained identical results for linear distance-discrimination.[11]

These considerations lead to the conclusion that a lesion in a sensory projection area may have a two-fold effect on retention: (1) The lesion destroys sensory discrimination mediated by that area and hence reduces retention to the extent that the habit depends solely on this discrimination. (2) The lesion eliminates any non-sensory function of the area in retention, *i.e.* the effect is similar to that demonstrated by Lashley for the

[6] Hunter, The temporal maze and kinesthetic sensory processes in the white rat, *Psychobiol.*, 2, 1920, 1-18.

[7] Lashley, Cerebral control versus reflexology: A reply to Professor Hunter, *J. Gen. Psychol.*, 5, 1931, 3-20.

[8] B. Casper, The normal sensory control of the perfected double-alternation spatial-maze habit in the albino rat, *J. Genet. Psychol.*, 43, 1933, 239-292.

[9] C. H. Honzik, The sensory basis of maze learning in rats, *Comp. Psychol. Monog.*, 13, 1936 (no. 64), 1-113.

[10] E. E. Ghiselli, The effects of lesions in the spinal cord on the ability of the rat to discriminate differences in inclined planes, *J. Comp. Psychol.*, 22, 1936, 319-323.

[11] C. W. Brown, Spinal lesions and distance discrimination, *J. Comp. Psychol.*, 33, 1942, 305-314.

visual areas. It is impossible to determine how much of a given lesion-produced deficit in retention is due to interference with sensory function and how much to non-sensory function unless we know the contribution of the sense in question to the control of performance. Under the ordinary conditions of multiple sensory control of maze-performance, we cannot evaluate the contribution of non-sensory functions of projection-areas. The purpose of the present experiments was to test Lashley's theory by using a maze devised by Hunter which can be run only on the basis of kinesthetic cues.[12] Hunter blinded his rats and also rotated the maze and interchanged its units before each trial. A simple alternation maze is easily learned under these conditions. This learning is due to the constant association of the stimulation resulting from a right turn with the following response of turning left and vice versa, these cues being the only ones remaining constant from trial to trial. It is probable that these kinesthetic cues are both tactual and proprioceptive.

Woolsey and Le Messurier,[13] using evoked potentials, have obtained evidence that tactual representation of the rat's body-surface on the cortex is orderly and delimited. This representation is referred to as somatic-sensory and divided into two areas, S I and S II. Woolsey has also localized the stimulable motor area, M, of the rat's cortex.[14] The somatic and motor areas together make up roughly the anterior 60% of the total cortex.[15] The dividing line between somatic and motor areas runs diagonally (dorsal and caudal) from the rhinal indentation nearly to the midline, with its mid-point at level 6 on the standard Lashley diagram.

Evidence on localization of proprioceptive function in the rat's cortex is very fragmentary. Brooks has localized a cortical area which must be present for the performance of the placing and hopping reactions.[16] The area covers roughly the dorsal half of the motor area and the dorsal fifth of somatic area I. In the cat, Gay and Gellhorn have shown that the area of cortical discharge on proprioceptive stimulation is primarily located in the sensorimotor cortex on either side of the central sulcus.[17] In a few cats, responses were found in all areas except the extreme poles, but the excitation was maximal in the sensorimotor area.

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[12] Hunter, A kinesthetically controlled maze habit in the rat, *Science,* 91, 1940, 267-269.

[13] C. N. Woolsey and D. H. Le Messurier, Pattern of cutaneous representation in the rat's cerebral cortex, *Fed. Proc.,* 7, 1948, 137.

[14] Woolsey, The pattern of localization in the motor cortex of the rat, *Fed. Proc.,* 8, 1949, 82.

[15] Unfortunately Woolsey's work, which also includes localization of visual and auditory cortex, has not been published in detail. Dr. J. P. Zubek has translated Woolsey's data into map-form in connection with a study of cortical function in tactual discrimination soon to appear in the *J. Compar. & Physiol. Psychol.*

[16] C. M. Brooks, Studies on the cerebral cortex. II. Localized representation of hopping and placing reactions in the rat, *Amer. J. Physiol.,* 105, 1933, 162-171.

[17] J. R. Gay and E. Gellhorn, Cortical projection of proprioception in the cat and monkey, *Proc. Soc. Exper. Biol.,* 70, 1949, 711-718.

Throughout the present study it was assumed, for lack of better evidence, that the tactual and proprioceptive areas of the rat cortex are limited to areas S I, S II, and M. It was predicted that lesions in these areas would produce severe deterioration of the kinesthetic habit compared with that due to equal lesions elsewhere.

PROCEDURE

The general plan of experimentation was as follows. After rats had learned the kinesthetic maze, they were subjected to lesions in various parts of the cortex. After postoperative recovery, retraining in the maze provided a measure of the effect of the lesions on retention.

The *Ss* were *blinded* male albino rats 55 to 65 days old. Preliminary training consisted of ten runs, one per day, down a straight pathway constructed like the maze to be learned. The animals were deprived of food for 24 hrs. and the reward was a bit of wet mash in a goal box. After eating this food the rat was given 20 min. of supplementary feeding outside the maze. Training and postoperative retraining on the maze were conducted in exactly the same manner.

Three versions of the maze were used, an elevated maze with narrow paths (1¾ in. wide), another with wide paths (4 in.), and an alley-maze (4 in. wide). A given rat ran under only one of these conditions. Each maze consisted of 8 T-units arranged in a simple alternation pattern. It was constructed of linear segments which could be interchanged in position. True-path and cul distances were the same in each maze, the total true-path being about 17 ft. long. Each maze stood on a large platform which could be rotated in the room. Random interchange of units and rotation of the maze (45° clockwise) were carried out daily during maze training and retraining. Retracing was prevented except within a cul. An error was counted if the rat entered a cul to the extent of a full body length (excluding the tail). Pre-operative training continued to a criterion of 3 perfect trials in succession plus 7 additional trials. Behavior on the elevated maze runs was observed for evidence of incoördination.

Each rat was operated 12 to 24 hr. after its last acquisition trial. Lesions were made either by cautery or aspiration—results from the two methods did not prove to be significantly different. Three types of operation were performed: (1) removal of somatic and motor cortex (anterior lesions); (2) removal of posterior cortex outside the somatic areas (posterior lesions); (3) sham operation.

Ten days were allowed for post-operative recovery during which time the animal was maintained on the 24-hr. hunger rhythm. After the recovery period, the rats were retrained to criterion on the maze. After relearning the animals were killed and maps of the lesions were made. Mapping consisted of transferring the limits of the lesion as seen on the gross brain to the standard Lashley diagram using proportional dividers. Twelve lesions which appeared to involve subcortical damage were also mapped from histological sections. The average error between the two methods of mapping was ±2.6% of the neocortex. The average algebraic error was 0.28% of the neocortex. The percentage of neocortex destroyed was computed for each animal excluding areas above the corpus callosum in the lateral views.

The measures obtained from the maze data were number of preoperative trials

to criterion and number of postoperative trials to criterion. The basic measure of postoperative deterioration was obtained by subtracting preoperative trials to criterion from postoperative. This deterioration score was distributed fairly symmetrically, whereas the original maze-data were skewed high. All of the *t*-tests reported below on mean deterioration scores were confirmed by using Festinger's d-test on the original data.[18]

RESULTS

(1) Ineffectiveness of posterior lesions: (a) Elevated mazes. The results show that posterior lesions have no effect on retention of the habit of

TABLE I

EFFECT OF LOCUS OF LESION ON MAZE-HABITS

Maze	Lesion group	No. Ss	Mean % lesion	Median trials to criterion		Mean deterioration score
				pre-op	post-op	
Narrow elevated	anterior	5	21.7	4	19	12.00
	posterior	4	23.9	8	6	−0.75
	sham	8	—	3.5	2	−3.00
Wide elevated	anterior	15	15.9	8	8	3.67
	posterior	25	17.4	6	4	−1.60
	sham	21	—	5	3	−1.09
Alley	anterior	10	23.0	11.5	7.5	2.40
	posterior	9	23.6	11	5	0.78

TABLE II

THE SIGNIFICANCE OF DIFFERENCES BETWEEN MEAN DETERIORATION SCORES

	Groups compared					
	Anterior vs. sham		Posterior vs. sham		Anterior vs. posterior	
Maze	*t*	P	*t*	P	*t*	P
Narrow elevated	4.82	.01	0.52	.70	2.98	.05
Wide elevated	2.00	.05	0.22	.80	1.81	.10
Alley	—	—	—	—	1.00	.40

running the elevated mazes. For both the narrow and wide mazes the mean deterioration-scores of animals with posterior lesions are close to those of the sham operants (Table I). The *t*-tests (posterior vs. sham, Table II) show that the differences are not significant. Table III also shows that there is no relation between extent of posterior lesions and deterioration of the habit in the wide elevated maze.

(*b*) *Alley-maze.* In the alley-maze there is no relation between extent

[18] L. Festinger, The significance of difference between means without reference to frequency distribution function, *Psychometrika,* 11, 1946, 97-105.

of posterior lesion and postoperative loss of the habit (Table III). This does not necessarily mean that the lesions had no effect. It is possible that removal of some particular, restricted area of posterior cortex caused loss of the habit, but an inspection of the individual lesions and behavioral data shown in Fig. 1 does not indicate that such was the case. Unfortunately, there are no results on this maze for sham operants, but the smaller lesions of the group can be considered control cases against which the larger lesions are evaluated by the correlation procedure. The near-zero

TABLE III

RANK-ORDER CORRELATIONS BETWEEN PERCENT OF LESION AND DETERIORATION SCORE

Maze	Anterior lesions				Posterior lesions			
	rho	P	Range of % lesion	No. Ss	rho	P	Range of % lesion	No. Ss
Wide elevated	+.11	>.05	5.7–38.1	15	−.03	>.05	5.8–41.6	25
Alley	+.77	<.01	4.4–36.0	14	−.16	>.05	12.8–55.0	11

coefficient obtained is taken to mean that posterior lesions have no effect on the alley maze habit.

There is another possibility which would invalidate this inference from the obtained correlation. If removal of a given small amount of posterior cortex should act to produce maximal loss of the habit obtainable through posterior lesion, a zero correlation between extent of lesion (beyond this small, functionally maximal lesion) and loss of the habit would be expected. The smallest of the posterior lesions tested on the alley-maze is 12.8% of the total cortex and one cannot therefore overlook this possibility. No evidence for the assumption is to be found in the literature, however, and the negative results with posterior lesions for the elevated maze-habit suggests that it may be discounted.

(2) Effects of anterior lesions: (a) Elevated mazes. In both types of elevated mazes, anterior lesions produced more loss of the habit than did either sham operations or posterior lesions of equal extent (Table I). The *t*-tests in Table II show that all of the differences are statistically significant except that between anterior and posterior effects for the wide maze which reached the 10-% level. Although anterior lesions are effective in producing deterioration of the habit, there is no relation between the extent of deterioration and the mass of tissue removed (Table III). This finding suggests that the effect of anterior lesions is due to the removal of some specific area of cortex. An analysis of the behavior on the wide maze suggested that the results might be attributed to incoördination result-

ing from the destruction of the motor area. Evidence of incoördination usually appeared when the rat was attempting to short-cut the turn at a choice-point; a slip of the inside hind leg led to a turn in the opposite direction (into the cul) as the leg was pulled back to footing on the pathway. Recovery of footing usually did not occur until the leg was stretched out on the pathway dorsal surface down.

This type of incoördination was noted on 17 of the 359 postoperative trials given on the *wide* elevated maze. Of these 17, 14 cases occurred in animals with anterior lesions (rats 63, 67, 77, 97, 104, 106, and

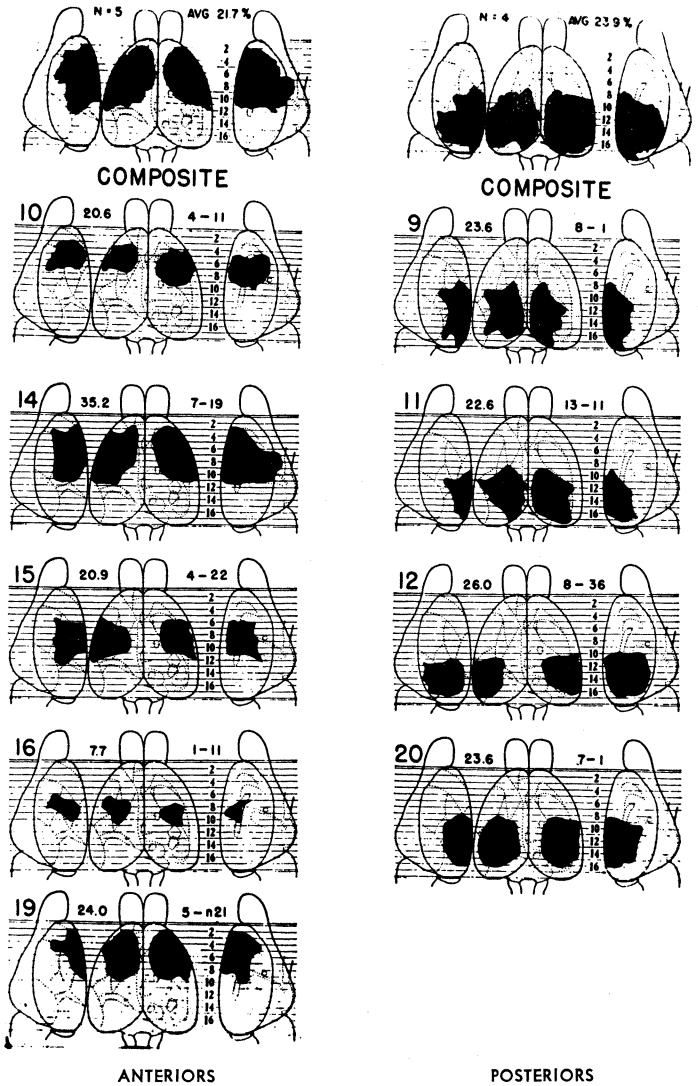
TABLE IV

THE EFFECTS OF MOTOR AND SOMATIC LESIONS (ON THE WIDE ELEVATED MAZE) COMPARED WITH POSTERIOR CONTROLS

Type of lesion	No. Ss	Mean % lesion	Median trials to criterion		Mean deterioration score	t	P
			pre-op	post-op			
Motor	4	21.1	7	15.5	8.25	2.70	.02
Posterior	10	24.9	7	3	−3.60		
Somatic	8	14.6	8	8	1.75	.12	1.00
Posterior	13	14.4	5	4	1.31		

107); the remaining three cases were due to cases of posterior lesion (rats 75 and 105). A chi-square test on this distribution of incoördinated trials between anterior and posterior groups gave a *P*-value of less than 0.01. In other words, the appearance of incoördination was significantly associated with anterior lesions. The post-operative records of the seven incoördinated animals with anterior lesions also played a large part in raising the mean deterioration-score for the anterior group. All seven animals had deterioration-scores greater than zero and these animals comprised 70% of the anterior cases with positive scores. A chi-square test within the anterior group on the frequencies of normal and incoördinated rats falling above and below zero-deterioration gave a *P*-value of 0.08 when corrected for continuity.

A further analysis on the effects of lesions in areas S I and S II versus those of lesions in area M indicates that the deteriorating effect of anterior lesions is due to invasion of the motor areas. Eight cases of lesion largely within the somatic areas (rats 58, 63, 65, 67, 72, 78, 79, and 107) were compared as a group with posterior cases chosen to match the somatic group in extent of lesion (rats 56, 57, 59-62, 70, 71, 76, and 88-91). Four cases of lesion of the motor area (rats 77, 97, 104, and 106) were compared with posteriors (rats 59, 71, 82-84, 95, 96, 102, 103, and



NARROW ELEVATED MAZE

FIG. 1A. MAZE-DATA AND MAPS OF CORTICAL DESTRUCTION

The number to the left of the dorsal view is the percentage of cortex destroyed; the first number at the right is the number of pre-operative trials to criterion and the second number is the number of post-operative trials to criterion. The letter 'n' before the number of postoperative trials indicates that the rat did not reach criterion in that number of trials.

105). The results are presented in Table IV, from which two facts are evident: (1) motor lesions are significantly more effective in producing deterioration of the habit than posterior lesions of approximately equal

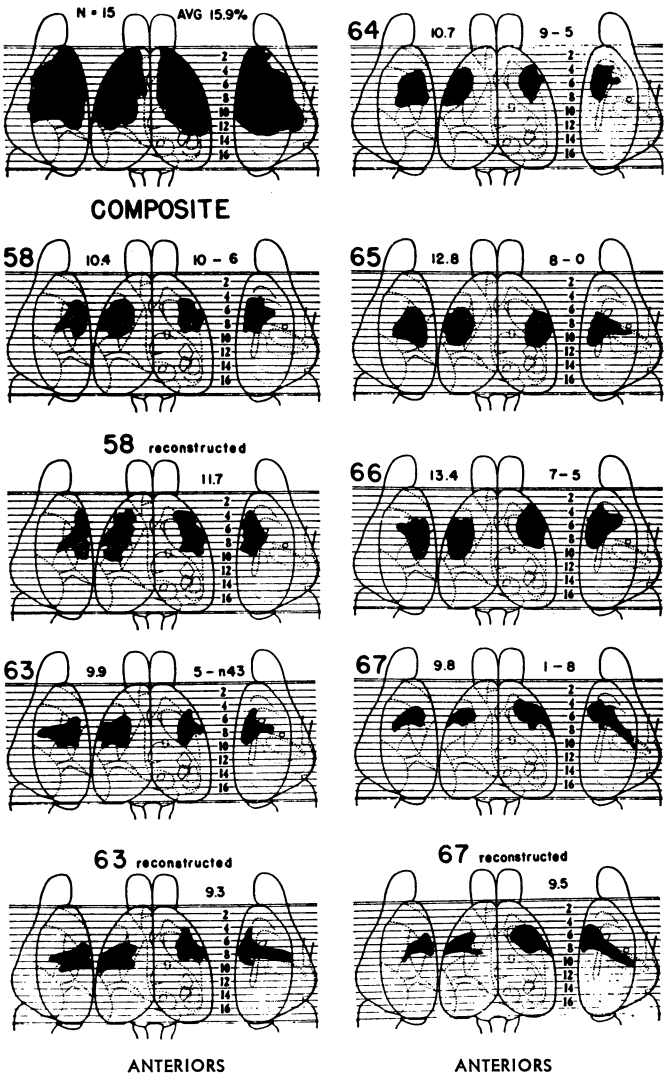
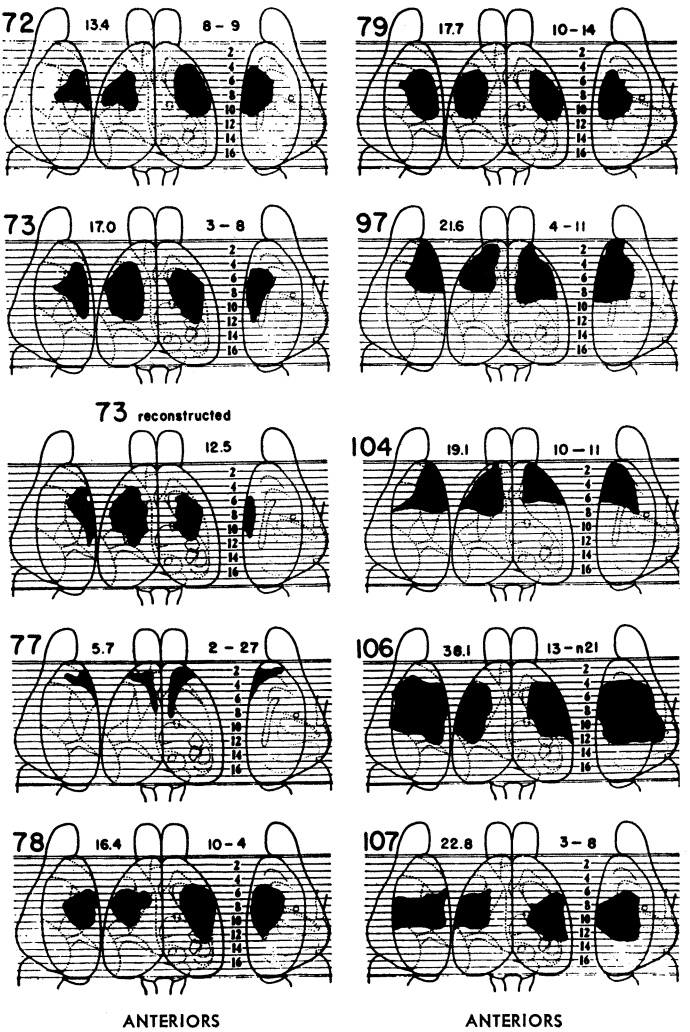


FIG. 1B. MAZE-DATA AND MAPS OF CORTICAL DESTRUCTION

extent; and (2) somatic lesions are not significantly more effective than posterior lesions. It is certain from these results that the striking effect of anterior lesions tested on the elevated mazes is at least partially due to the disorienting effect of the motor symptoms which appear on the elevated pathways. It is possible that post-operative scores under these condi-



WIDE ELEVATED MAZE

FIG. 1C. MAZE-DATA AND MAPS OF CORTICAL DESTRUCTION

tions may be attributed both to motor symptoms and to lack of tactual and proprioceptive function. The use of the alley-maze eliminated all motor symptoms.

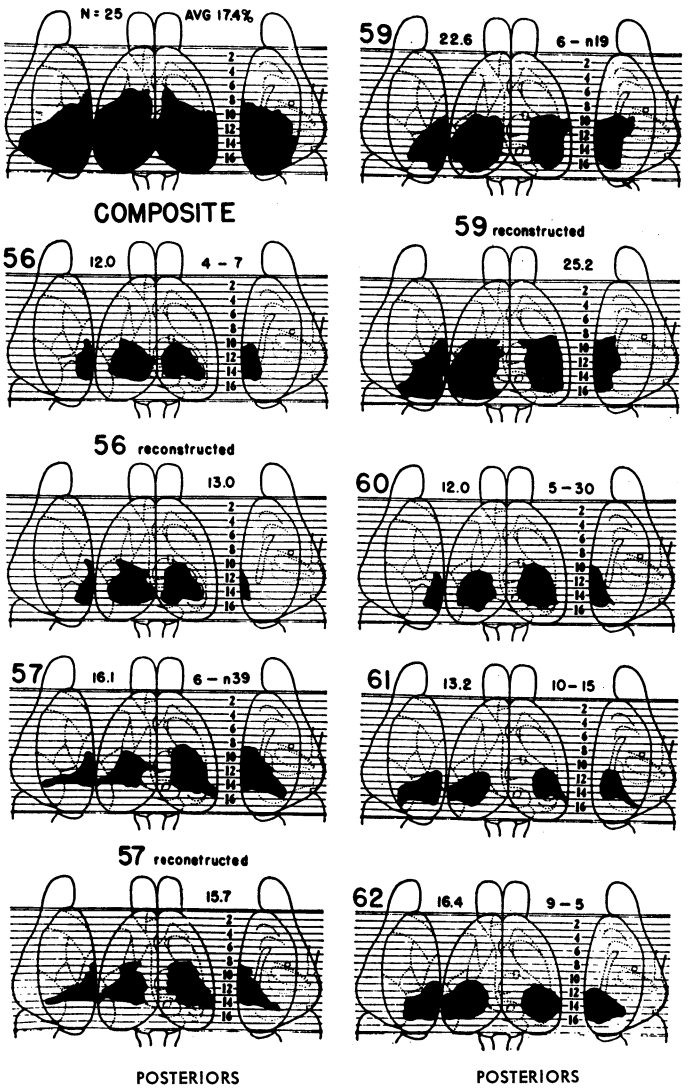


FIG. 1D. MAZE-DATA AND MAPS OF CORTICAL DESTRUCTION

(b) Alley-maze. On the whole, the anterior lesions were smaller than the posterior (Fig. 1, alley-maze), which made it necessary to match groups for extent of lesion before comparing them. The groups to be compared were formed by omitting the four smallest lesions from the anterior series (rats 33-35 and 51), and the two largest lesions from the posteriors (rats

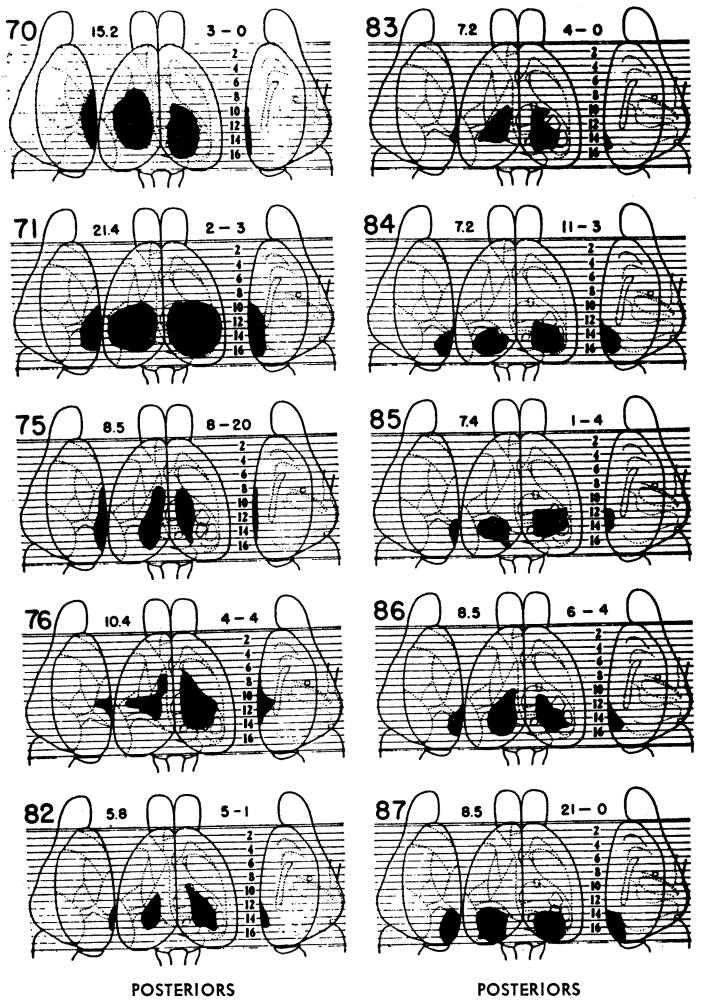


FIG. 1E. MAZE-DATA AND MAPS OF CORTICAL DESTRUCTION

115 and 116). The anterior group of 10 rats had an average lesion of 23.0% (ranging from 14.5 to 36%) and the posterior group of 9 rats had an average lesion of 23.6% (ranging from 12.8 to 42.1%).

There was a high positive correlation between extent of anterior lesions and deterioration-score (Table III, alley-maze), but a comparison of the post-operative records of rats having anterior lesions with those having

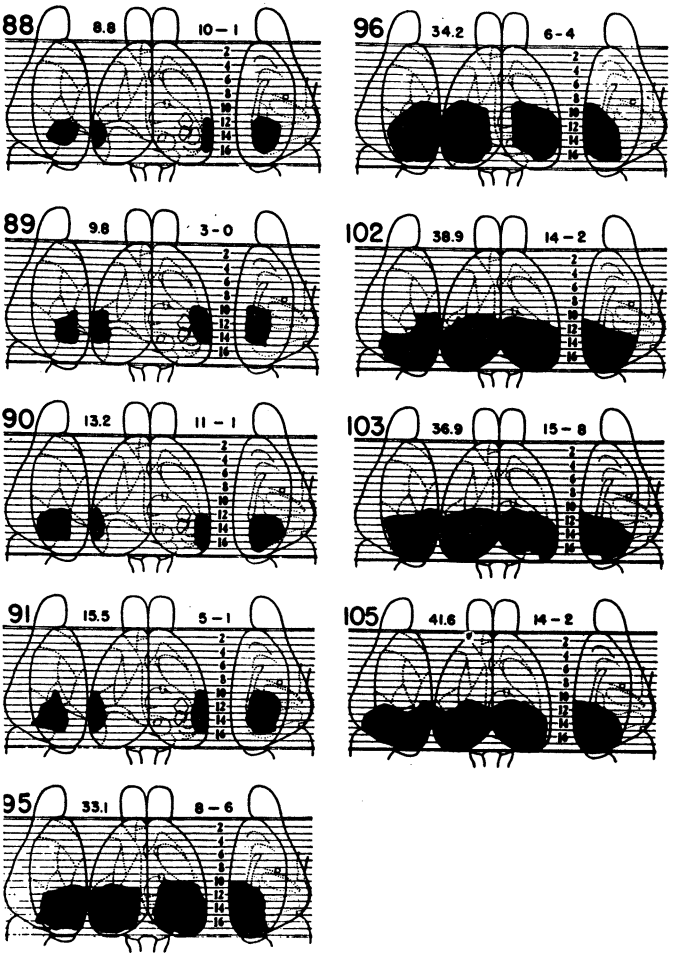
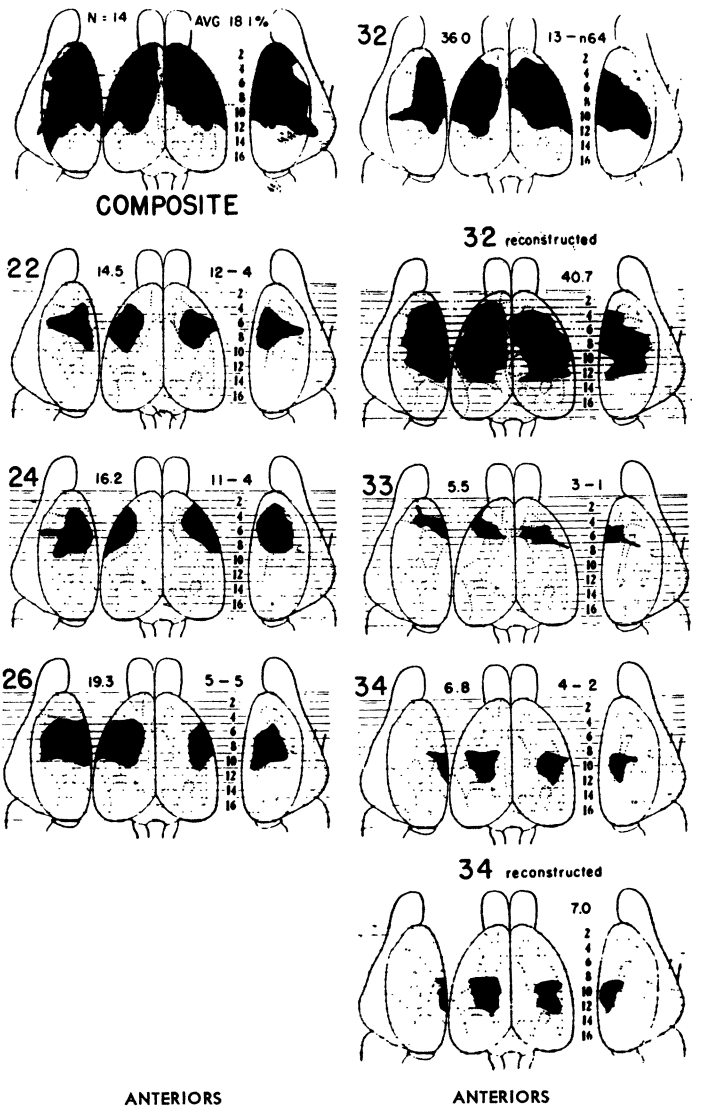


FIG. 1F. MAZE-DATA AND MAPS OF CORTICAL DESTRUCTION

equal posterior lesions indicated that the anterior lesions were not as a group significantly greater in effect than posterior (Tables I and II). Again small anterior lesions may be taken as controls against which to



ALLEY-MAZE

FIG. 1G. MAZE-DATA AND MAPS OF CORTICAL DESTRUCTION

evaluate large anterior lesions. The high correlation, then, must mean that the larger anterior lesions (ranging from 14.5 to 36% were effective in producing deterioration of the alley-maze habit.

Discussion

In the light of previous experimental findings there are two aspects

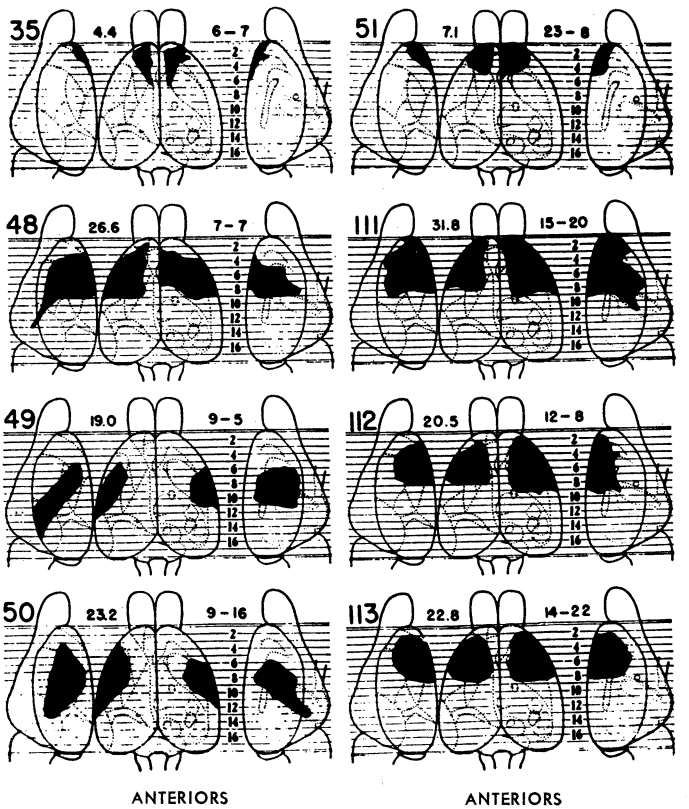


Fig. 1H. Maze-Data and Maps of Cortical Destruction

of the present results requiring explanation: (1) the failure to find *extreme* deterioration of the kinesthetic maze-habit after lesions in the anterior areas; and (2) the failure to confirm Lashley's finding that lesions in sensory areas having no sensory function in the habit nevertheless produce serious deterioration.

There seem to be three possible explanations of the relatively mild effect of anterior lesions on the kinesthetic maze habit. First, control of the sensory cues on the basis of which the habit was performed may not have completely eliminated all but tactual and proprioceptive cues. Con-

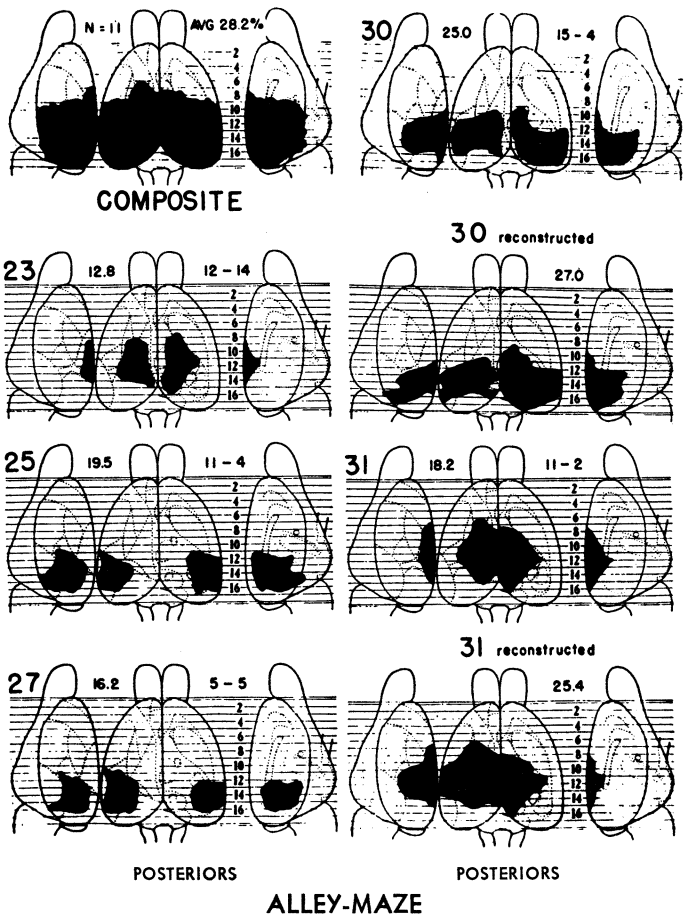
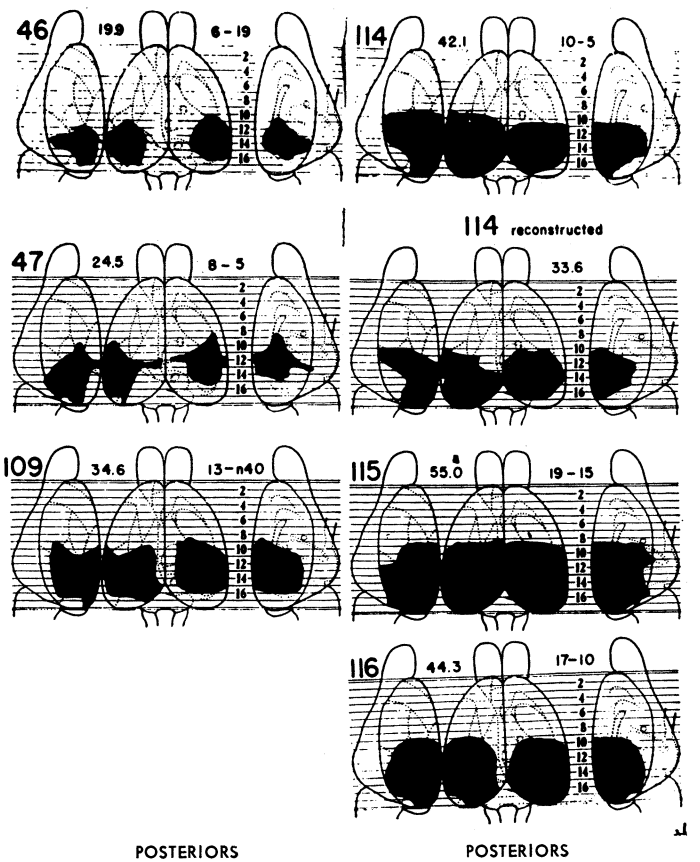


FIG. 11. MAZE-DATA AND MAPS OF CORTICAL DESTRUCTION

sidering the rotation and interchange of the maze, and the use of blinded rats, it is difficult, however, to imagine what cues could have been operating other than those from the movements of the animal. There is evidence that, with blinded rats, rotation and interchange are adequate con-

trols of exteroceptive cues (Casper,[19] Hunter and Hall[20]).

A second possibility is that the maze habit, when learned to criterion, is not under the guiding control of sensory stimulation (Lashley and Ball[21]). If, however, the habit were under purely central control, there



ALLEY-MAZE

FIG. 1J. MAZE-DATA AND MAPS OF CORTICAL DESTRUCTION

[19] Casper, *op. cit.*, 289.

[20] W. S. Hunter and B. E. Hall, Double alternation behavior of the white rat in a spatial maze, *J. Compar. Psychol.*, 32, 1941, 253-266.

[21] Lashley and Ball, *op. cit.*, 71-106.

would be no reason, on present experimental evidence, to expect any localizable cortical function; it would be difficult to explain the fact that the larger anterior lesions in the present experiment produced loss of the habit in the alley-maze, except on the assumption that the wholly central control of the habit lies in this anterior region. Since there is no evidence for such localization in the literature of the cortical control of maze-performance, it cannot be considered a strong possibility.

A third explanation seems the most likely: that tactual and proprioceptive areas of the rat's cortex are well localized, but that few lesions of the anterior series in the alley-maze were large enough to invade the greater part of these areas. If proprioceptive function in the rat's cortex extends over a large part of the motor area, complete removal of tactual and proprioceptive areas would involve nearly the whole anterior half of the cortex. The average anterior lesion in our experiment was 23% of the cortex (ranging from 14.5 to 36%) and the average of the sensorimotor lesions was 27.5% (ranging from 20.5 to 36%). The correlation between extent of anterior lesion and amount of deficit in the alley-maze indicates that the larger lesions were the more effective in interfering with the habit. It is possible that this effect would be enhanced with even larger lesions.

The second surprising result of the investigation was the lack of any serious deterioration of performance on the elevated maze after lesions in posterior (non-somesthetic) areas of the cortex. Lashley has twice demonstrated that lesions of the striate area of the cortex interfere markedly with retention of an alley-maze habit learned *in the absence of vision.*[22] On the average, Lashley's posterior lesions were about the same size as the present ones. Lashley's maze III was shorter than those here used (7.7 ft. of true-pathway) and his training trials were massed at five per day. The correct pathway in the present pattern was a right-angled zig-zag pattern with eight turns and eight culs. Although Lashley's maze had eight culs, the correct path was serpentine, with five U-turns in simple alternation. Normal animals required an average of 19 trials to learn this maze; the median number of trials would probably have been about 25% lower. This value may be compared with a median of 11 trials in the present alley maze. It is apparent that the two maze situations are not comparable. Since, so far as sensory control is concerned, the two experiments

[22] Lashley, *Brain Mechanisms and Intelligence,* 1929, III. Studies of cerebral function in learning: XII. Loss of the maze habit after occipital lesions in blind rats, *J. Comp. Neurol.,* 79, 1943, 431-462.

were the same and should have produced the same results, it would seem that the discrepancy is due to differences in the mazes (alley vs. elevated) or in methods of training (massed vs. spaced practice). Only further experiment can decide this question.

The loss of the kinesthetic maze-habit after large anterior lesions and the lack of effect of posterior lesions are inconsistent with a theory of equipotentiality of function. The evidence obtained here lends direct support to Hunter's analysis of Lashley's equipotential results. The most general conclusion indicated by the present results is this: when the cues controlling a maze-habit are restricted to one or two modalities, the function of the cortex in the habit is restricted to the sensory projection areas of those modalities. This conclusion should be tested using larger lesions, other modalities, and different maze-patterns; and it should somehow be harmonized with Lashley's demonstration of the function of the visual projection area in a non-visual maze habit.

SUMMARY

Rats were trained on elevated and alley-mazes under considerations designed to permit learning only on the basis of tactual and kinesthetic cues. Retention was measured following cortical lesions. In general the results were: (1) posterior lesions had no effect; (2) the larger anterior lesions (somatic and motor areas) produced loss of the alley-maze habit; (3) anterior lesions of the motor area produced incoördinated running on the elevated mazes which resulted in slower post-operative learning. These results are not consistent with the theory of equipotential cortical function.