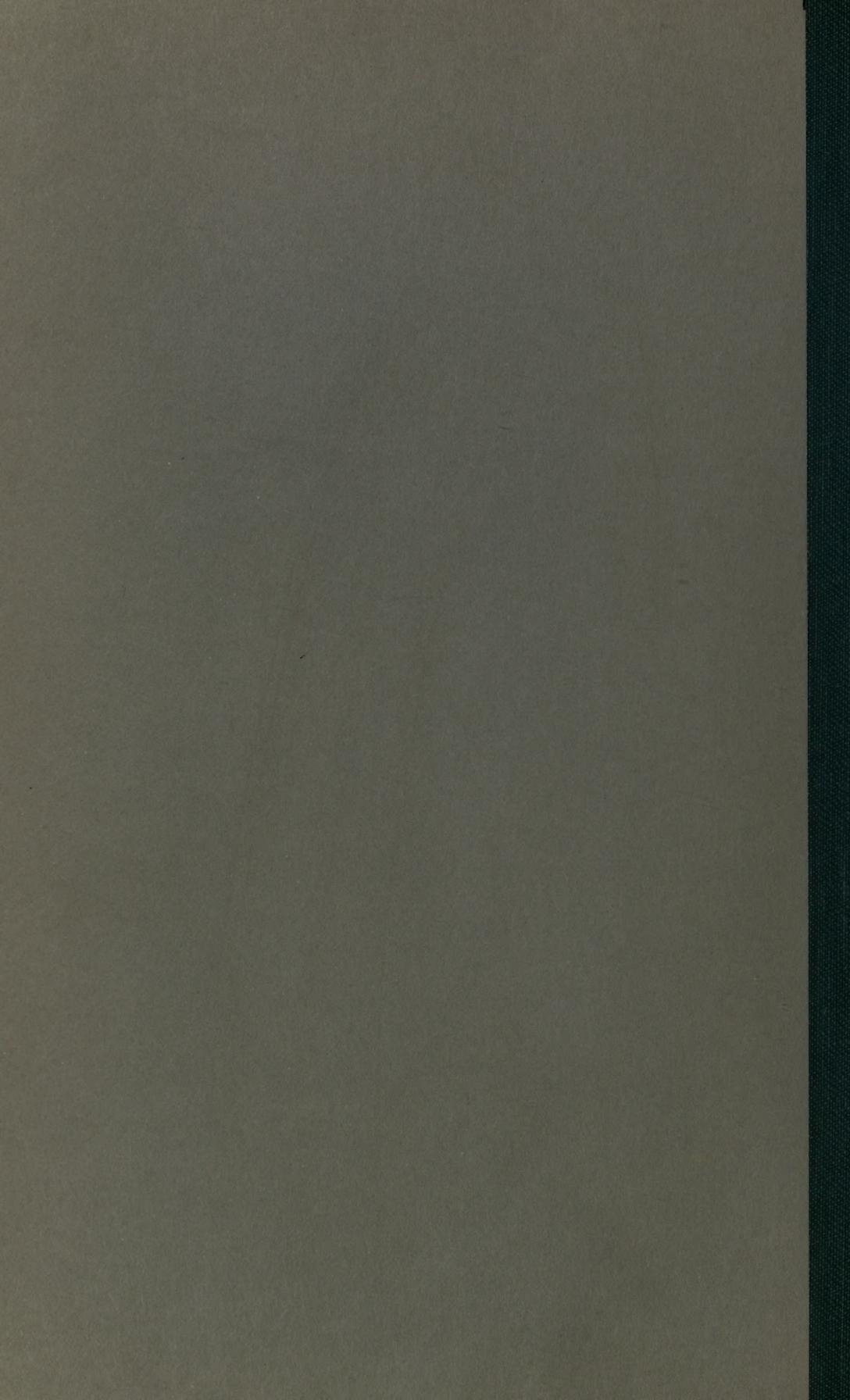




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## STUDIES OF CEREBRAL FUNCTION IN LEARNING<sup>1</sup>

### IX. MASS ACTION IN RELATION TO THE NUMBER OF ELEMENTS IN THE PROBLEM TO BE LEARNED

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THREE TEXT FIGURES AND FIVE PLATES

#### INTRODUCTION

Measurements of the influence of extent of cerebral lesion upon efficiency in various functions have given quite different results, according to the functions studied. In some cases there is clearly an all-or-nothing relation between some functional area and the capacity for performance; in others, a close relationship between surface extent of injury and degree of lowering of efficiency. The data thus far accumulated in quantitative studies are summarized in table 1. The constants given are based on error scores, where these are available, otherwise on trials for learning or relearning. The table shows a definite bimodal distribution of the constants. Six fall below 0.10, thirteen are above 0.50, and only three fall between these limits. Of these three (double platform box, difference threshold for two lights, and learning of a 1 cul de sac maze) the first reduces to zero when corrected for motor disorders, the second is approximately 0.50, and the third is based on a maze which is known on other grounds to be an unreliable measure of performance.

<sup>1</sup>This work was supported by a grant from the Otho S. A. Sprague Memorial Institute. We are indebted to Prof. L. L. Thurstone and to Mrs. Annette M. Wiley for advice and assistance in the statistical treatment of the data and to Dr. Margaret Frank for assistance in the training of animals and the preparation of material for histological study.

The bimodality of distribution of these constants is good evidence that the positive correlations are not merely the

TABLE 1

*The relation between extent of lesion and efficiency of performance in previous studies of the effects of cerebral injury. The constants are for error scores where these are available, otherwise for trials*

TASK	LOCUS OF LESION	COEFFICIENT OF CORRELATION	REFERENCE
Simple maze, P. R.	Frontal	0.00 <sup>1</sup>	Lashley and Franz ('17)
Double platform box, L.	Frontal	0.24 ± 0.15	Lashley ('20)
Double platform box, L., corrected for motor disorder	Frontal	0.00 <sup>1</sup>	Lashley ('20)
Delayed alternation, L.	Frontal	-0.02 ± 0.19	Loucks ('31)
Delayed alternation, P. R.	Frontal	0.54 ± 0.12	Loucks ('31)
Maze habit, 8 culs de sac, L.	Frontal	0.64 ± 0.08	Maier ('32 a)
Reasoning	Frontal	0.54 ± 0.09	Maier ('32 a)
Light-darkness discrimination, L.	Visual	0.08 ± 0.14	Lashley ('26)
Light-darkness discrimination, P. R.	Visual	0.72 ± 0.05	Lashley ('26)
Light-darkness discrimination, P. R., corrected for critical area	Visual	0.73 ± 0.08	Lashley ('32)
Light-darkness discrimination, P. R.	Visual	0.64 ± 0.10	Lashley ('30)
Discrimination two lights, L.	Visual	0.58 ± 0.10	Lashley ('30)
Discrimination two lights, P. R.	Visual	0.65 ± 0.10	Lashley ('30)
Discrimination two lights, difference threshold	Visual	0.49 ± 0.09	Lashley ('31 b)
Visual acuity and pattern vision	Visual	0.00 <sup>1</sup>	Lashley ('31)
Reasoning	Visual	0.75 ± 0.05	Maier ('32 b)
Discrimination, noise, P. R.	Auditory	0.61 ± 0.11	Wiley ('32)
Maze habit, 8 culs de sac, L.	All parts	0.86 ± 0.03	Lashley ('29)
Maze habit, 3 culs de sac, L.	All parts	0.65 ± 0.07	Lashley ('29)
Maze habit, 1 cul de sac, L.	All parts	0.30 ± 0.16	Lashley ('29)
Maze habit, 8 culs de sac, P. R.	All parts	0.51 ± 0.11	Lashley ('29)
Maze habit, 1 cul de sac, P. R.	All parts	0.00 ± 0.08	Lashley ('29)

L. = initial learning, P. R. = postoperative retention.

<sup>1</sup>Correlations not computed, but no suggestion of a correlation from inspection of the data.

result of some error in sampling, in which case they would show a normal distribution around zero, but express a genuine relation between the variables studied. All of the constants, however, have been determined from relatively small numbers

of cases, and, although they do attest the importance of the mass relationship, they give little further information concerning it. Several purely statistical questions concerning the relationship remain to be answered before we can make much progress in interpreting the functional significance of the correlations. These questions call for a larger mass of data than has been hitherto available and the primary aim of the present study is to obtain a large series of cases, which can be analyzed with some assurance of reliability.

#### *The continuity of the mass relationship*

The available evidence is not conclusive as to whether there is a continuous progression in the effects of cerebral lesions from the least to the greatest or whether there may be a critical amount of destruction below which injuries are relatively ineffective in producing deterioration. Several studies (Lashley, '26, '29, table 11; Maier, '32 a and b) have given indication of a sharp increase in the effectiveness of lesions at about 15 to 20 per cent destruction. This may be evidence for lower limit of extent of lesion necessary to produce significant symptoms, but the appearance of a sharp rise in effectiveness might also result, if the relation between extent of lesion and deterioration had a logarithmic or other accelerated form. Lashley ('26) found that the correlation ratio gave a higher value (0.84) for the relation between lesion and amnesia than did the correlation coefficient (0.72), indicating that the relationship is curvilinear, but with the small number of cases the difference between these constants was not statistically reliable. Thurstone ('33) has analyzed the data for maze III of Lashley's study ('29) and finds that the rate of learning is for this maze a function of the sixth power of the intact cortex. Maier ('32 a, b) has reported a very pronounced drop in efficiency in reasoning tests with lesions exceeding 18 to 20 per cent of the cortex.

None of these studies provides sufficient material to determine the reliability of the form of the function. Our first problem, therefore, has been to collect data on maze learning

after cerebral lesion for a larger number of cases, as a basis for a more reliable determination of the continuity and form of the relation between extent of lesion and efficiency in learning.

*The equivalence of different cortical areas for maze learning*

It seems quite clear from the results of Cameron ('28), Lashley ('29), Maier ('32), and Jacobsen<sup>2</sup> that injuries in any part of the cortex result in some retardation in the rate of learning the maze. The relative effects of injuries in different cytoarchitectural areas remain uncertain. Lashley ('29) attempted to measure the influence of injuries in different areas by comparing the numbers of errors made by animals with lesions in different parts of the cortex, and concluded that, within the limits of accuracy of the experiment, the same amount of destruction within any area produced the same amount of retardation in learning. The number of cases in his experiment was small, however, and the average deviation of 13 per cent from equality between the groups may have been really significant. Moreover, his method of grouping cases resulted in a considerable overlap of the areas compared and this may have tended to equalize the averages of the arbitrary groupings. We have attempted to deal more adequately with this problem of the relative effects of lesions in different areas.

*The relative effects of lesions symmetrical and asymmetrical in the two hemispheres*

All of the studies dealing with quantitative effects of cerebral lesions have dealt with lesions which were made as nearly as possible symmetrical on the two hemispheres. Data on the critical areas for pattern vision (Lashley, '31) and unpublished data on maze retention after removal of one hemisphere indicate that somewhat different results are to be anticipated when the lesions are markedly asymmetrical.

<sup>2</sup> Unpublished experiments on reversal of training.

Since there is never an exact duplication of the fields involved in the two hemispheres, it is desirable to determine whether this lack of symmetry affects the correlations between extent of lesion and learning records.

*Organic dementia in relation to the complexity of the task to be performed*

Clinical studies of organic dementia frequently suggest that a nearly normal ability in the execution of simple acts may accompany a marked deterioration in the performance of more difficult tasks. Comparing the rate of learning for three mazes of 1, 3, and 8 culs de sac by partially decerebrate rats, Lashley ('29) found that whereas the ratios of difficulty of the mazes for normal animals were as 1 to 2.2 to 6.5, for animals with cerebral lesions the ratios were as 1 to 3.5 to 20.6. This seemed to conform to the clinical evidence in showing that a given amount of cerebral destruction resulted in a much greater retardation in the performance of complex than of simple tasks. There was some indication also that large lesions produced a disproportionately greater retardation in the more difficult mazes than in the simpler ones, although the evidence for this was not statistically valid (Lashley, '29, table 11).

The meaning of these data is by no means clear. If we attempt to define difficulty merely in terms of the number of trials required for learning, then certain tasks, the learning of which is unaffected by cerebral lesions, appear more difficult than maze learning. The difficulty of a task may depend upon the number of similar elements which must be integrated in order to give an efficient performance. In logical processes the ability to keep in mind a number of separate elements and at the same time manipulate them in thought is essential, and, according to Boumann and Grünbaum ('25), this capacity primarily suffers in organic dementia. In learning relatively meaningless material the difficulty, as measured by practice necessary to secure perfect reproduction, increases as the  $3/2$  power of the length of the series

(Thurstone, '30). In such situations the difficulty of the task corresponds to its objective complexity and may be stated quantitatively in terms of the number of items included in the test series.

In addition to this quantitative aspect of 'difficulty' there are unquestionably qualitative elements. If the situation is unfamiliar, if the stimuli are nearly indistinguishable, if the integrations required are foreign to the native organization of the animal, or if the relations to be recognized are obscure, the difficulty of the task is increased. The maze studies reported by Lashley ('29) did not distinguish clearly between number of similar elements and qualitative diversities. The mazes differed in number of culs de sac, but also presented fundamental differences in general plan, so that it is impossible to determine which of the possible sources of difficulty was predominant.

In the present study we have attempted to devise a series of mazes differing only in the number of similar elements which must be learned and to test their relative difficulty for normal and operated animals.

### *Problems*

We may summarize the chief problems with which the present study is concerned in the following questions:

1. Is there a significant correlation between extent of cerebral lesion and degree of retardation in maze learning when tested with an extensive series of cases? Confirmation of this leads to the further problems:
2. Is retardation a continuous function of the size of the lesion, or is there a critical amount of injury below which little effect can be observed?
3. Are the effects of equal amounts of injury in different cytoarchitectural fields the same for the learning of enclosed mazes or are there characteristic differences in the degree of retardation resulting from injury within different loci?
4. With tasks differing in complexity (number of objectively similar elements), what is the relation of the performance

of animals with cerebral lesions to the relative difficulty of the tasks for normal animals?

#### PROGRAM OF EXPERIMENTS

Since a primary object of our work was to test the influence of extent of lesion upon the rate of learning tasks of different complexity we planned to train animals with equal lesions in four mazes with 4, 8, 12, and 16 culs de sac. In such experiments on learning with animals we are confronted with two variables which cannot be controlled simultaneously; chance individual variations in capacity and transfer of training. Thus, if we wish to compare learning records on two different problems we may either train the same animals on both problems, in which case we control individual variation but ignore transfer, or we may compare the records of different groups of animals, each trained on a single problem, in which case transfer effects are eliminated but individual differences are uncontrolled except statistically. In the first case transfer effects may be controlled by subdividing the subjects and reversing the order of training with a part, but where four problems are to be compared the arrangement of tests for transfer becomes impossibly cumbersome. We decided, therefore, to use separate groups of animals, one with each of the four mazes, attempting to get comparable groups with respect to extent of lesion and to control individual differences to some extent by subsequent training of all groups on the same task (maze V). The validity of this control is somewhat lessened by the possibility of differential transfer from the different mazes and the evidence for equality of the groups is not as clear as we could wish, but the differences are small in comparison with other differences revealed by the experiments.

For comparison with the operated cases and for standardization of the mazes we have trained groups of normal animals under parallel conditions with the operated ones. The various groups and the number of animals included in each are shown in figure 1, together with the ground plans of the

different mazes used. The groups are designated Normal and Operated I, II, III, and IV, corresponding to the number of the maze in which the animals of the group were first trained.

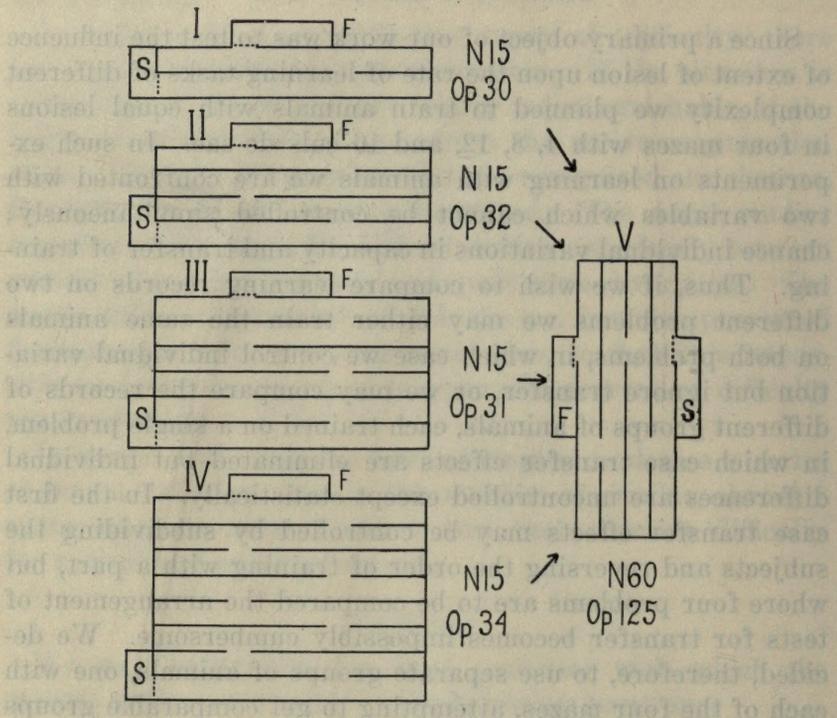


Fig. 1 General plan of the experiments. The four comparison mazes (actually constructed by subdividing the largest) are shown at the left, the control maze at the right. The number of animals trained in each maze is indicated. *F*, food compartment; *S*, starting compartment; *N*, normal group; *Op.*, operated group.

## METHODS

### *Mazes*

For comparative tests, mazes of the general plan of Lashley's maze III ('29) were used. This plan has the advantage that the number of blind alleys can be increased indefinitely without introducing any fundamental change in the general plan. It has the possible disadvantage that the correct path is one of simple alternation, capable of easy

generalization. If the animals made such generalizations, mazes of this plan should all be of about equal difficulty, whether they have few or many culs de sac. We believe, however, that this objection is not serious since the formation of the alternation habit seems to require many more trials than are required for complete mastery of our longest maze (Carr, '17; Hunter, '20; Loucks, '31).

A maze with 16 culs de sac was so constructed that segments could be closed off to give mazes with 4, 8, or 12 culs de sac. The ground plans of the resultant mazes are shown in figure 1. The maze was constructed of  $\frac{3}{8}$ -inch pine with 4-inch partitions between the alleys. The top was covered with fine wire mesh. Electrical contacts on counterbalanced sections in the floor permitted automatic recording of errors. Four separate starting boxes gave access to the segments of the maze and were closed off by one-way doors of sheet metal. In later discussion the four mazes which can be arranged by blocking or opening the doorways between the culs de sac will be referred to as mazes I, II, III, and IV, as shown in figure 1.

As a control of the training records with these four mazes, a fifth having 8 culs de sac was used. The ground plan of this maze is also shown in figure 1. Its construction was similar to that of the other, except that the order of alternation of turns was reversed and the relative position of food box and starting box altered. Its orientation in the room during use was also different from that of the other mazes, as indicated in the figure. In later discussion this will be called maze V.

### *Training methods*

Before controlled training in the mazes was begun, the animals were given preliminary training in traversing a straight runway, 10 feet in length, until they came through promptly and were not disturbed by handling. They were then fed for 3 days in succession only in the food box of the maze, without access to the alleys. In training a 'trial' was

counted as a complete trip from starting box to food box.<sup>3</sup> One trial was given on the first day and five trials per day thereafter. After each trial the animal was allowed to eat a bite of food. At the termination of each day's training it was fed to repletion.

A rigid control of the incentive cannot be employed with operated animals, since those with larger lesions require constant care and special feeding to keep them in good condition. We must therefore recognize the possibility of unequal motivation in different animals. The only test of the existence of such differences that we have is the apparent eagerness of the animals for food, and on this basis the operated animals must be judged more strongly motivated than normals. There is significant evidence from other sources that the differences between operated and normal animals in learning cannot be ascribed to differences in motivation. In two types of experiment (light-darkness discrimination, Lashley, '29, and delayed alternation, Loucks, '31) the inferiority of operated animals has appeared only in post-operative relearning, although the motivation used was always the same. In studies employing the double platform (Lashley, '20) the same incentive (hunger) was used as in the maze studies with no evidence of inferiority on the part of the operated animals.

#### *Criteria of learning*

With mazes I to IV training, after one trial on the first day, was continued with 5 trials per day until the animal made a record of 10 consecutive errorless runs, or for 150 trials (100 trials with maze V), in case the criterion of 10 errorless trials was not reached earlier. Time and errors per trial and total trials to reach the criterion were recorded. Of these, errors are probably the most reliable criterion of progress in maze

<sup>3</sup> It is not always possible to obtain a complete trial in one day because of limitations of the experimenter's time. In such cases the animals were removed from the maze, fed a limited amount, and returned to the maze on the following day, the accumulated time and errors being counted as a single trial.

learning, and constants computed from the error records are emphasized in our treatment of the data. Constants computed from time and trials are also included as contributory, but less reliable evidence.

The early trials, especially the first, are scarcely comparable with later trials in the maze after the preliminary exploratory period is passed. Lashley ('17) has shown that in early trials there is a strong tendency to explore all parts of the maze. Various means have been suggested to correct the learning records for exploratory errors in early trials (Thurstone, '33). We have not yet sufficient data for evaluation of the methods or to judge which of the errors are significant for learning. In the first trial the animal has had no opportunity to associate the maze with food and hence the motivation in this trial differs from that in all later trials. For this reason we have omitted the records of the first trial in the computation of all constants reported.

Among the animals with cerebral lesions many failed to reach the criterion within 150 trials. This affects the error scores very little; inspection of the records reveals that 80 per cent of the total errors are made in the first 50 trials, and that by 150 trials the animals have settled down to rather stereotyped runs with at most 2 or 3 errors per trial. In computing correlations based on trials for learning, we have used total trials minus the number of errorless runs made before the termination of training, to avoid throwing all of these cases into one rank.

None of the conclusions which we have drawn is dependent upon any of these special methods of grouping the data. That is, the group differences and correlations are essentially the same, within the limits of reliability which we have required, whether we use total trials, trials less errorless runs, total errors, or total errors less errors in the first trial, total time, or total time less time for the first trial.

In view of the large number of animals which failed to reach the criterion of learning within the training given, some method of extrapolating the learning curves and predicting

ultimate achievement might give more valid results than the mere averaging of the crude data. Uncertainty as to the legitimacy of the available methods, however, together with the enormous labor involved in such computations has led us to employ the crude scores.

*Animals which failed to run*

A few of the operated cases failed to reach the food compartment after 12 to 18 hours in the maze and became inactive in the maze situation. Since such records of failure cannot be interpreted in terms of capacity to learn, these cases were simply discarded. Such behavior appeared most frequently

TABLE 2

*Comparison of the four groups of operated animals with respect to extent of lesion as a test of the selective effect of excluding records of animals which failed to get through the maze*

GROUP	AVERAGE PERCENTAGE OF LESION	$\sigma$	UPPER LIMIT OF RANGE	NUMBER WITH MORE THAN 40 PER CENT DESTRUCTION
I	20.7	13.4	65.3	2
II	25.1	13.8	56.4	4
III	25.7	14.1	60.2	6
IV	24.6	12.4	49.7	5

among animals with extensive lesions and more often in the longer than in the shorter mazes, so that there was probably some selection exerted in this way. Table 2 summarizes the distribution of lesions in the four groups compared. The average destruction in the groups is essentially the same, the variation within the groups is not greatly different. The upper range indicates possible selection only for maze IV. It is doubtful, therefore, whether the failure of cases to get through the maze has had any appreciable effect upon the results. We have attempted to control this possible selection by computing constants for animals with smaller lesions only and comparing these with similar constants for the entire group.

*Surgical and anatomical methods*

The methods of destroying the cerebral cortex were those previously described by Lashley ('29), with operation in two stages for the more extensive lesions. Since we wished to obtain four groups of animals with similar lesions, we operated on not less than four animals at one time, attempting to duplicate the lesions in all and distributing them later to the four groups. At the time of operation a sketch was made of the type of lesion, and in case a member of the group died, it was replaced by another with duplicate operation.

The method of reconstruction of the lesions was that described earlier: graphic reconstruction of serial sections (Lashley, '29).<sup>4</sup> We have paid especial attention to the subcortical lesions, which cannot be avoided with larger destructions of the cortex. Analyses of the data to determine the influence of subcortical injuries upon the results are reported on page 32.

*Graphic and statistical analysis*

The diagrams prepared in reconstruction of the serial sections represent approximately the surface distribution of the

<sup>4</sup>Loucks ('32) has recently advocated a method which differs from the above in three particulars: the use of a fiber stain, the arbitrary limitation of the boundary of the lesion at the point where total destruction of tissue cuts the pyramidal layer of the cortex, and the measurement of the lesion along the perimeter of each section, instead of surface area of the reconstructed diagram. These differences in method do not seem to us advantageous. Although in old lesions there is usually a clean-cut destruction of tissue, the cortex bounding the area of completed destruction often shows pathological changes which certainly render it non-functional. Fiber stains do not reveal this and the arbitrary criterion of complete destruction disregards it. In determining the extent of lesion we have drawn the boundaries at the point where the cortex assumes a normal appearance, providing against personal bias by having the observer in ignorance of the animal's training record when the reconstruction of the lesion is made.

The method of measuring the lesion along the perimeter of the section is doubtless somewhat more accurate than the determination from the graphic reconstruction. Some years ago, the senior author made a number of determinations by both methods. The difference in results by the two methods was about 5 per cent, which was within the limits of accuracy in remeasurement by either method.

lesions, as determined in relation to internal landmarks. The distribution of cytoarchitectural fields is variable and in a large series of cases it is quite impossible to study the cytoarchitecture of the cortex in sufficient detail to determine the limits of the remaining fields in each case. At best we can only compare the diagrams of the lesions with the somewhat conventionalized diagram of cytoarchitectural fields adapted from Fortuyn's studies ('14). The classification of cases by areas destroyed has been made by superimposing a transparent diagram of the cytoarchitectural fields upon the diagrams of lesions and measuring the area of the lesion within each field with a planimeter. These measurements were then expressed as percentage of the total neocortex and used as a basis for estimation of the effects of injury to different fields.

The question of the relative effectiveness of lesions restricted to one hemisphere and of lesions of equal magnitude distributed symmetrically on both hemispheres in reducing learning ability has arisen continually in experimental work of this sort. It has been difficult to test the question by direct experiment because of the impossibility of distinguishing with certainty in maze studies between genuine reduction of learning capacity and possible disturbances of orientation produced by asymmetrical motor defects which frequently result from unilateral lesions. The recent study by Loucks ('32) of habits involving alternation of turns to right and left, where the turns were recorded separately has shown that even a strong motor tendency to rotation does not affect the rate of formation of the alternation habit, so that with a series of bilateral lesions like the present one, we may proceed with assurance upon the assumption that bilateral asymmetries have not influenced the training records through their motor effects.

We have therefore attempted to determine whether or not the bilateral destruction of corresponding areas is more effective in retarding learning than unilateral destruction. To this end the lesion in one hemisphere was traced on transparent paper which was then inverted and superimposed upon

the other hemisphere. The area of overlap between the two lesions was then outlined, measured, and expressed as percentage of the total area of the neocortex. These percentages have been used as a basis for computing the constants for comparison with those obtained by consideration of the total extent of lesion. In later discussions the measurements obtained in this way are referred to as 'lesions common to both hemispheres.'

In computing correlations we have used the method of rank order,<sup>5</sup> in preference to the Pearson  $r$ , since our data do not follow a random distribution. All methods of measuring the degree of association between variables are based upon assumptions concerning random sampling which are not fulfilled by our data on brain lesions, and the use of correlation methods in such cases can be justified only as a crude method of expressing the presence or absence of a significant association. The relation between extent of lesion and retardation is probably not rectilinear, so that the correlation ratio would be a more suitable measure of the association. In most cases it would give a somewhat higher figure than the correlation coefficients reported, but in the present state of our knowledge slight differences in the magnitude of the coefficients have no significance. We are dealing with differences between individuals and groups which are many times greater than the range of normal variation, so that refinement of statistical treatment is of relatively less importance than if we were trying to measure smaller differences.

#### *Special controls*

There is little doubt that the experimenter may influence the maze records of his animals by slight differences in procedure of which he is scarcely aware. Somewhat more gentle handling of one than of another animal, deviations in the allowance of food, and personal variations in the criteria of errors may influence the data and are likely to do so in a

$$^5 p = 1 - \frac{6\sum dx^2}{n(n^2-1)}; \text{ P.E.}_p = \frac{1-p^2}{\sqrt{n}} 0.7063.$$

constant direction where the experimenter has definite pre-conceptions. We have tried to control such influences of the personal equation as follows. The recording of errors was automatic, leaving no room for personal judgment. The training was done, as far as possible, in ignorance of the character of the operation to which the animal had been subjected, although such ignorance can be only partial, since it is impossible to mistake an animal with extensive cerebral lesion. In all cases the lesions were reconstructed and the reconstructions checked without knowledge of the experimental records of the animals.

TABLE 3  
*Correlations between the scores of the same animals in learning two mazes*

MAZES	NUMBER CASES	TOTAL ERRORS LESS FIRST TRIAL	TOTAL TRIALS	TOTAL TRIALS LESS CORRECT RUNS	TOTAL TIME LESS FIRST RUN
Normal animals					
I with V	15	$-0.18 \pm 0.18$	$0.09 \pm 0.18$	$0.18 \pm 0.18$	$0.50 \pm 0.14$
II with V	15	$0.20 \pm 0.18$	$0.18 \pm 0.18$	$-0.30 \pm 0.17$	$0.46 \pm 0.14$
III with V	15	$0.22 \pm 0.17$	$0.29 \pm 0.17$	$0.44 \pm 0.15$	$0.38 \pm 0.16$
IV with V	15	$0.49 \pm 0.14$	$0.06 \pm 0.18$	$0.17 \pm 0.18$	$0.42 \pm 0.15$
Operated animals					
I with V	30	$0.84 \pm 0.04$	$0.78 \pm 0.05$	$0.89 \pm 0.03$	$0.72 \pm 0.06$
II with V	31	$0.91 \pm 0.02$	$0.78 \pm 0.05$	$0.84 \pm 0.04$	$0.63 \pm 0.08$
III with V	30	$0.92 \pm 0.02$	$0.84 \pm 0.04$	$0.90 \pm 0.03$	$0.54 \pm 0.09$
IV with V	34	$0.88 \pm 0.02$	$0.80 \pm 0.04$	$0.84 \pm 0.04$	$0.69 \pm 0.06$

#### VALIDITY OF THE MAZE TECHNIQUE

The consistency of performance of the animals and the validity of the methods can best be tested by a comparison of the scores made in different mazes. The scores by the various criteria for the same animals on the two mazes in which each was trained have been correlated and the results are shown in table 3. Except for time, the correlations obtained from normal animals are low. There is a tendency for them to increase in magnitude with increase in the number of culs de sac in the mazes, which suggests that we increase the reliability of maze studies by increasing the complexity

of the mazes, but in general they indicate that our mazes do not provide a trustworthy index of individual differences among normal animals.

In contrast to this, the correlations for the operated cases are uniformly high and significantly greater than their probable errors. The most consistent results are obtained from errors and from total trials less errorless runs, with correlation coefficients ranging from 0.84 to 0.91. They indicate that there is some common factor involved in the learning of all five mazes by operated animals which is reliably measured by the criteria which we have adopted.

The nature of this factor is not clearly indicated. The lower correlations for time show that it is not mere activity or speed of running. Whether it is motivation, ekphorie, or some sort of insight into the problem is not revealed by these figures. The previous results of Lashley ('29) for learning in the double platform box and in the maze show that there is no correlation in the learning of these two problems, although the same incentives are used, so differences in motivation seem improbable as a cause of the correlations.

This throws us back upon some mechanism directly involved in the learning process itself as the function measured in our study. The divergent results with the double platform box (Lashley, '20), the learning of which was unaffected by any cortical lesion, indicate that mere fixation in memory or ekphorie is not the factor involved. Our knowledge of the actual factors responsible for maze learning, such as the influence of thwarting in blind alleys, the formation of associations with specific cues in the maze, maintenance of the sense of direction, symbolization of the maze pattern and the like, is too slight to justify any further conclusions concerning the nature of the function which is being measured. Lashley ('29) has attempted to relate it to general intelligence, but such speculation can be justified only by showing a high correlation of maze learning with tests known to involve some general capacity which can be termed intelligence by common consent. All that we can justifiably conclude is that our mazes

reliably measure some function which is common to the learning of different mazes.

#### ANALYSIS OF EXPERIMENTAL DATA

##### *Deterioration after cerebral lesion*

The training records of all operated cases are summarized in tables 4, 5, 6, and 7 and the details of the lesions are shown in plates 1 to 5. The numbers and arrangement of the figures in the plates correspond to the experimental numbers of the animals in the tables, for ready reference. Similar data for normal animals trained under parallel conditions are given in tables 8, 9, 10, and 11.

The mean scores for the various criteria, with probable errors of the means and standard deviations of the distributions, are summarized in table 12. With average destructions of 20 to 25 per cent, the operated animals require from 2 to 17 times as much practice to reach the criterion of learning as do normals. The greatest differences are in the numbers of errors, the least in the numbers of trials. Since training was discontinued after 150 trials, the average of trials for the operated cases does not express the actual retardation. Fifty-one of the operated animals failed to reach the criterion in mazes I to IV, and 47 in maze V, and the scores for trials would have been very much higher, if these animals had been trained to errorless running. The error scores, therefore, probably represent most truly the difference between the normal and operated groups. Judged by this criterion, the latter require from 7 to 17 times as much practice as the former.

A comparison of table 12 with table 15 reveals that there is retardation, even for the smallest amounts of injury. Animals with lesions of less than 10 per cent of the neocortex required 153 per cent as much practice, measured in terms of errors, as did normals.

All these differences between normal and operated groups are statistically reliable. They demonstrate that cerebral lesions produce a significant reduction in the capacity for maze performance, even when the lesions are quite small.

*The relation between extent of lesion and rate of learning*

The coefficients of correlation between extent of lesion and maze learning for four criteria with each of the mazes are given in tables 13 and 14. For all mazes by each criterion

TABLE 4

*Learning scores for operated group I on mazes I and V. The score for time omits the record of the first trial. Scores for all errors and for all except those of the first trial are given*

NO.	PER CENT DESTRUC- TION	MAZE I				MAZE V			
		Total time minus time first trial (seconds)	Total errors	Total errors minus errors first trial	Trials	Total time minus time first trial (seconds)	Total errors	Total errors minus errors first trial	Trials
1	3.0	561	21	18	11	471	61	27	16
2	3.0	681	42	35	46	223	33	25	36
3	3.3	229	34	23	31	956	141	106	41
4	4.9	981	36	35	11	2,284	109	97	16
5	5.7	682	51	46	61	524	80	50	31
6	9.8	850	69	60	31	1,594	220	165	61
7	9.9	168	17	13	11	754	104	61	91
8	10.9	246	51	31	26	216	39	33	26
9	13.4	793	44	37	21	1,335	146	129	36
10	14.1	394	41	40	46	351	108	71	36
11	14.3	683	48	44	66	4,351	368	275	69
12	15.2	3,599	579	561	150	1,810	787	549	100
13	17.9	3,447	142	130	66	1,991	258	253	100
14	18.4	2,974	116	100	66	1,176	156	108	56
15	19.1	498	109	88	56	314	95	64	41
16	19.3	532	37	33	41	256	82	72	31
17	19.6	8,884	62	57	63	479	72	30	31
18	20.7	1,122	282	280	150	2,195	689	648	100
19	21.0	1,431	85	61	54	534	75	70	41
20	21.9	12,373	263	249	150	2,343	420	324	100
21	24.6	2,598	396	367	150	1,307	362	353	100
22	25.2	3,077	41	38	31	10,417	295	272	100
23	28.3	3,901	591	566	150	5,387	899	831	100
24	31.4	11,104	272	258	84	5,735	533	478	100
25	31.9	2,580	153	147	107	2,055	247	220	86
26	34.7	2,894	602	563	121	2,758	554	451	100
27	36.6	28,043	616	609	150	24,932	1160	1044	100
28	36.8	3,130	269	226	81	1,082	319	300	100
29	41.7	14,356	1341	1330	150	9,596	1496	1448	100
30	65.3	14,147	1362	1356	150	10,247	1581	1462	100

the correlations are quite high and significantly greater than their probable errors. For the most significant of the criteria, errors exclusive of the first trial, the coefficients range from  $0.57 \pm 0.08$  for group 4 on maze V to  $0.80 \pm 0.05$ , with an

TABLE 5

*Learning scores for operated group II in mazes II and V. Arranged as table 4*

NO.	PER CENT DESTRUCTION	MAZE II				MAZE V			
		Total time minus time first trial (seconds)	Total errors	Total errors minus errors first trial	Trials	Total time minus time first trial (seconds)	Total errors	Total errors minus errors first trial	Trials
31	2.1	23,984	100	93	35	505	49	44	36
32	2.1	7,882	166	119	47	934	92	70	61
33	5.3	6,247	115	87	31	808	69	54	81
34	6.2	1,636	112	60	31	131	37	8	11
35	12.4	6,293	91	88	71	319	27	20	21
36	13.5	7,680	222	182	61	772	74	56	46
37	14.7	3,254	387	308	136	562	202	143	41
38	15.6	1,313	102	57	150	465	90	62	100
39	16.4	14,959	387	367	150	3,421	170	135	76
40	18.6	1,440	107	93	36	79	25	5	11
41	19.2	22,365	318	314	77	538	107	96	31
42	20.6	6,697	139	133	50	323	49	38	26
43	20.7	2,040	188	149	71	817	168	153	81
44	21.3	3,926	338	285	150	2,054	294	201	85
45	21.3	88,476	442	432	30	1,685	313	226	46
46	21.5	2,276	183	160	136	557	178	104	71
47	22.5	19,579	858	838	150	13,060	998	695	100
48	22.9	528	85	77	56	428	167	89	30
49	24.9	17,295	307	283	98	1,508	129	125	100
50	26.1	5,235	917	900	150	3,232	2714	1187	100
51	31.9	9,671	1,708	1695	150	4,573	1087	1046	100
52	32.1	2,466	409	374	146	506	125	120	66
53	32.5	1,747	267	241	106	367	148	93	31
54	33.9	35,515	837	751	150	2,143	285	225	100
55	36.4	3,814	475	439	120	456	146	112	36
56	37.2	10,612	2,230	1509	66	1,678	498	437	36
57	39.8	15,500	1,973	1819	150	1,569	340	314	100
58	39.8	9,621	2,594	2120	150	8,646	1716	1712	100
59	41.2	6,352	717	703	150	2,237	802	469	100
60	46.3	92,267	2,485	2391	150	22,636	3770	3700	100
61	49.1	70,006	3,196	2971	150	21,437	2257	2253	100
62 <sup>1</sup>	56.4	34,722	10,204	9437	119 <sup>1</sup>				

<sup>1</sup> Died after 119 trials.

average of 0.72. These figures demonstrate a significant relationship between extent of lesion and degree of retardation in maze learning, and substantiate the earlier results of Lashley ('29) with similar mazes.

TABLE 6

*Learning scores for group III in mazes III and V. Arranged as table 4*

NO.	PER CENT DESTRUCTION	MAZE III				MAZE V			
		Total time minus time first trial (seconds)	Total errors	Total errors minus errors first trial	Trials	Total time minus time first trial (seconds)	Total errors	Total errors minus errors first trial	Trials
63	2.1	1,337	125	103	31	216	28	14	21
64	2.8	17,141	166	98	24	263	28	16	16
65	3.8	9,335	189	143	94	397	56	40	26
66	10.0	4,084	388	371	126	8093	530	437	48
67	10.5	3,176	216	145	61	185	29	21	21
68	13.5	981	115	80	41	153	40	32	11
69	15.6	4,645	188	137	77	365	47	36	31
70	16.9	8,934	127	85	51	210	197	26	16
71	18.4	9,460	1106	1095	150	756	256	143	46
72	19.0	13,445	1766	1735	150	2117	845	427	100
73	19.1	6,178	749	642	150	2896	438	437	100
74	19.4	1,119	144	99	58	124	21	14	26
75	19.9	1,949	118	96	66	384	72	63	30
76	20.6	56,316	723	655	150	1136	172	151	66
77	23.2	3,795	335	295	91				
78	24.1	11,270	402	302	85	2922	408	223	61
79	24.5	10,614	605	528	146	666	94	88	41
80	25.2	14,857	410	348	150	466	127	78	26
81	26.8	12,435	4323	2411	150	4495	453	420	100
82	28.3	23,014	1043	995	124	3047	767	237	51
83	29.8	2,046	174	154	101	303	100	75	21
84	29.8	8,250	764	662	127	1904	615	471	100
85	30.4	9,074	453	440	150	1301	283	275	100
86	36.0	45,877	6947	6480	150	2085	441	394	100
87	40.0	7,594	671	634	150	1650	299	262	100
88	40.2	17,427	2133	1946	150	7170	1021	927	100
89	41.9	31,690	3898	3866	150	4028	1376	1144	100
90	44.2	11,713	666	619	150	1318	169	99	66
91	47.6	18,649	3488	3321	150	1767	535	511	100
92	52.3	5,891	1205	1102	150	1226	445	380	100
93 <sup>1</sup>	60.2	12,405	2791	2624	150	3754	1094	1008	92 <sup>1</sup>

<sup>1</sup> Refused to run after 92 trials.

So far as the magnitude of the correlation is concerned, no definite conclusions are justified. Lashley reported a correlation of 0.72 between errors and extent of lesion—a figure

TABLE 7

*Learning scores for operated group IV in mazes IV and V. Arranged as table 4*

NO.	PER CENT DESTRUCTION	MAZE IV				MAZE V			
		Total time minus time first trial (seconds)	Total errors	Total errors minus errors first trial	Trials	Total time minus time first trial (seconds)	Total errors	Total errors minus errors first trial	Trials
94	1.3	15,440	448	379	105	568	73	49	21
95	1.5	4,035	189	112	16	75	14	5	6
96	3.4	7,476	180	149	60	1,013	37	32	11
97	6.6	4,321	183	173	51	1,902	89	83	33
98	11.5	6,136	145	98	31	446	34	21	21
99	12.3	5,869	810	737	150	1,664	389	347	100
100	13.4	1,600	306	232	46	391	87	65	16
101	15.6	4,747	313	285	76	184	57	21	16
102	18.4	3,521	257	194	89	812	80	75	41
103	18.8	3,492	645	530	71	738	163	115	71
104	19.9	33,967	6,050	6,011	150	1,525	478	468	100
105	21.3	8,920	418	389	98	151	19	9	11
106	21.5	1,596	197	157	71	195	48	42	31
107	21.5	5,898	644	607	57	386	136	63	51
108	22.8	7,384	1,276	954	150	1,043	618	362	100
109	23.5	2,248	161	127	61	175	30	23	11
110	23.5	6,536	561	523	150	244	40	35	21
111	24.9	93,181	2,063	1,855	150	12,651	623	479	100
112	25.3	2,157	188	131	66	236	29	17	21
113	25.4	5,954	1,233	1,221	150	997	419	407	100
114	25.4	7,661	1,495	1,386	150	3,116	934	874	100
115	25.6	1,731	360	230	51	423	138	96	41
116	26.8	4,665	461	428	136	597	156	119	46
117	29.4	4,038	714	501	106	367	61	38	16
118	29.4	79,522	12,844	7,163	150	6,939	1217	1192	100
119	32.6	16,241	1,774	801	150	3,983	505	378	100
120	34.7	19,262	497	445	119	828	159	76	43
121	37.7	29,654	2,659	1,888	150	5,565	738	544	100
122	37.7	27,971	2,748	2,734	150	1,730	320	312	100
123	42.1	13,177	924	812	150	940	203	143	61
124	43.2	5,858	1,725	1,660	150	1,922	764	691	100
125	44.2	6,253	667	547	150	1,095	423	216	51
126	46.5	12,920	1,013	741	150	4,073	272	266	100
127	49.7	137,964	12,978	12,818	150	19,320	1504	1364	90

TABLE 8

*Training records of normal group I in mazes I and V. Arranged as table 4*

NO.	MAZE I					MAZE V				
	Time (seconds)	Time minus time first trial	Errors	Errors minus errors first trial	Trials	Time (seconds)	Time minus time first trial	Errors	Errors minus errors first trial	Trials
1	2833	1756	34	31	21	1243	488	45	21	21
2	187	164	17	14	16	494	363	69	54	41
3	206	179	12	10	6	1049	581	88	52	26
4	285	114	23	11	11	413	360	51	40	36
5	4138	3998	34	31	15	5873	5428	79	68	17
6	4331	4201	55	49	71	865	534	38	29	21
7	2084	1824	50	44	41	615	385	36	26	31
8	567	481	21	17	31	466	381	36	29	26
9	414	387	13	11	16	629	359	47	32	51
10	764	668	29	22	41	284	230	22	17	26
11	1565	1510	33	31	20	61	48	2	2	11
12	264	222	5	4	11	844	677	47	29	21
13	713	404	13	8	21	3319	2604	172	147	46
14	171	103	4	2	6	428	243	25	16	21
15	2436	2389	25	24	35	4715	2870	167	104	36

TABLE 9

*Training records of normal group II in mazes II and V. Arranged as table 4*

NO.	MAZE II					MAZE V				
	Time (seconds)	Time minus time first trial	Errors	Errors minus errors first trial	Trials	Time (seconds)	Time minus time first trial	Errors	Errors minus errors first trial	Trials
16	513	426	48	40	21	241	101	29	11	11
17	10781	2475	112	52	26	1333	654	72	46	32
18	6586	5654	38	49	26	1069	326	39	21	26
19	4389	3129	100	69	41	5998	898	123	18	36
20	18885	17513	127	104	45	9337	1002	182	20	31
21	2618	1981	90	73	26	536	300	44	24	31
22	12927	11336	199	156	18	405	287	45	37	26
23	3012	2647	116	98	54	8324	4628	172	88	40
24	4748	1838	124	77	37	346	304	20	13	26
25	2846	2616	80	73	50	344	189	21	14	11
26	2536	2391	90	82	30	235	100	14	8	21
27	5570	4293	113	95	35	896	329	33	17	16
28	2235	1071	81	58	46	198	149	13	10	16
29	1595	1222	58	46	21	1301	1166	64	58	21
30	829	707	33	27	31	432	205	22	11	31

TABLE 10

*Training records of normal group III in mazes III and V. Arranged as table 4*

NO.	MAZE III					MAZE V				
	Time (seconds)	Time minus time first trial	Errors	Errors minus errors first trial	Trials	Time (seconds)	Time minus time first trial	Errors	Errors minus errors first trial	Trials
31	750	656	31	25	21	312	124	21	8	16
32	2163	2030	89	82	41	215	63	13	4	11
33	2064	1504	82	65	29	441	299	56	47	43
34	3287	2816	175	142	56	853	349	35	13	21
35	7210	5145	189	171	46	591	372	46	35	26
36	2071	1814	90	76	31	879	720	46	35	40
37	4849	1669	191	125	31	627	262	48	26	16
38	9620	2420	169	98	52	414	147	31	13	16
39	2518	1258	92	61	51	406	355	23	20	41
40	9555	8790	153	132	43	457	155	24	3	8
41	1462	527	89	43	26	219	141	36	25	11
42	5802	4537	146	114	57	1508	793	98	82	100
43	7241	2155	267	64	101	1158	602	40	21	41
44	1360	1114	50	39	11	341	182	19	7	31
45	2442	1542	82	49	36	143	82	7	4	16

TABLE 11

*Training records of normal group IV in mazes IV and V. Arranged as table 4*

NO.	MAZE IV					MAZE V				
	Time (seconds)	Time minus time first trial	Errors	Errors minus errors first trial	Trials	Time (seconds)	Time minus time first trial	Errors	Errors minus errors first trial	Trials
46	10382	3481	245	101	76	938	698	46	38	61
47	3558	2633	113	73	101	625	461	27	17	31
48	2327	1718	180	130	101	364	355	50	49	56
49	3113	2941	465	448	36	2398	1612	417	349	51
50	9681	6391	113	87	51	2928	499	71	12	11
51	11100	2858	209	106	51	876	30	33	1	6
52	10562	7861	97	82	41	407	210	17	12	6
53	2291	1041	72	41	81	817	149	22	12	16
54	3761	2736	149	82	41	144	80	11	5	11
55	6389	2461	221	91	26	305	224	22	16	26
56	5376	2428	186	60	31	451	159	21	5	21
57	3979	1279	219	47	51	229	147	16	11	26
58	11539	11013	253	230	126	405	310	17	13	11
59	15035	4235	64	50	27	854	458	27	14	23
60	30241	22141	220	194	79	1956	407	72	28	16

TABLE 12  
Averages for each of the eight groups of animals used in the experiment

GROUP	MAZE	NUMBER OF ANIMALS	AVERAGE PER CENT LESION	ERRORS MINUS FIRST TRIAL	$\sigma_{dis.}$	P.E.av.	TRIALS MINUS CORRECT RUNS	$\sigma_{dis.}$	P.E.av.	TOTAL TRIALS	$\sigma_{dis.}$	P.E.av.	TIME MINUS FIRST TRIAL, 100 SEC.	$\sigma_{dis.}$	P.E.av.
Norm. 1	I	15	0	22.0	13.75	2.40	11.40	5.80	1.01	24.27	16.73	2.91	12.27	13.31	2.35
Norm. 2	II	15	0	66.73	43.18	7.52	21.00	7.09	1.24	33.80	10.33	1.90	39.53	44.52	7.75
Norm. 3	III	15	0	85.73	41.43	7.22	21.93	8.40	1.46	42.13	20.35	3.55	25.32	20.81	3.62
Norm. 4	IV	15	0	121.47	100.93	17.58	29.20	10.94	1.91	61.27	29.86	5.20	50.14	52.66	9.15
Norm. 1	V	15	0	44.40	36.45	6.34	13.87	7.41	1.29	28.73	10.87	1.89	10.37	14.25	2.47
Norm. 2	V	15	0	26.40	21.51	3.75	11.13	4.39	0.77	25.00	8.53	1.49	7.09	10.98	1.91
Norm. 3	V	15	0	22.87	20.28	3.53	11.67	8.01	1.40	29.00	22.47	3.91	3.10	2.22	0.39
Norm. 4	V	15	0	38.80 <sup>1</sup>	83.92	14.62	12.13	10.43	1.82	24.80	17.25	3.00	3.87	3.71	0.64
Op. 1	I	30	20.7 ± 1.7	246.70	345.36	42.53	55.63	49.28	6.07	77.70	50.24	6.19	42.57	60.74	7.48
Op. 2	II	32	25.1 ± 1.6	921.09	1706.61	203.49	79.39	48.86	5.92	103.84	46.75	5.57	167.38	234.89	23.01
Op. 3	III	31	25.7 ± 1.7	1039.06	1396.96	169.23	91.68	46.32	5.61	114.61	42.78	5.18	124.19	122.83	14.88
Op. 4	IV	34	24.6 ± 1.4	1382.88	5089.95	588.79	88.44	49.13	5.68	109.12	44.36	5.13	174.03	289.19	33.45
Op. 1	V	30	20.7 ± 1.7	333.87	387.74	47.75	50.93	32.65	4.02	68.17	32.09	3.95	32.63	49.48	6.09
Op. 2	V	31	24.1 ± 1.5	451.36	786.56	95.29	48.77	32.40	3.93	65.26	31.72	3.84	31.77	56.10	6.80
Op. 3	V	30	25.7 ± 1.7	281.63	297.78	36.67	44.72	33.23	4.16	60.53	34.41	4.24	18.57	19.83	2.44
Op. 4	V	34	24.6 ± 1.4	265.50	331.59	38.36	44.36	36.89	4.33	56.76	36.31	4.20	22.44	38.65	4.47

<sup>1</sup> One case in this group which exceeds  $4 \times \sigma_{dis.}$  of the group has been omitted in computing this mean.

which corresponds exactly to our average, but this can scarcely be considered more than a chance correspondence. If all conditions of random sampling are fulfilled, the coefficient of correlation gives a measure of the relative effectiveness of a common factor and other causes of deviation in determining the distribution of two series of variables. Our data, however, do not fulfill the necessary conditions, and the magnitude of the correlations only justifies the conclusion

TABLE 13

*Correlations between total extent of lesion and the various criteria of maze learning for the five mazes studied*

Maze	I	II	III	IV	V
Number of cases	30	32	31	34	125
Errors less first trial	0.80 ± 0.05	0.80 ± 0.05	0.70 ± 0.07	0.60 ± 0.08	0.64 ± 0.04
Time less first trial	0.75 ± 0.06	0.32 ± 0.11	0.43 ± 0.10	0.39 ± 0.10	
Total trials	0.75 ± 0.06	0.69 ± 0.07	0.71 ± 0.06	0.66 ± 0.07	
Trials less correct runs	0.79 ± 0.05	0.79 ± 0.05	0.74 ± 0.06	0.56 ± 0.08	

TABLE 14

*Correlations between total extent of lesions and the various criteria of maze learning for the comparison maze (maze V)*

	ERRORS LESS FIRST TRIAL	TIME LESS FIRST TRIAL	TOTAL TRIALS	TRIALS MINUS CORRECT RUNS
Group I	0.74 ± 0.06	0.60 ± 0.08	0.74 ± 0.06	0.79 ± 0.05
Group II	0.75 ± 0.06	0.51 ± 0.09	0.63 ± 0.08	0.71 ± 0.06
Group III	0.66 ± 0.07	0.52 ± 0.09	0.70 ± 0.07	0.73 ± 0.06
Group IV	0.57 ± 0.08	0.46 ± 0.10	0.54 ± 0.09	0.52 ± 0.09
All combined	0.64 ± 0.04			

that there is a significant relationship between the extent of cerebral lesion and the amount of retardation in maze learning.

In fact, the use of the correlation coefficient with our data is justified only as a rough test of the existence of a relationship. Data presented below show that the relationship is not rectilinear and that the correlation ratio would more accurately express it, as Lashley ('26) found for the effect of lesions upon brightness discrimination. Since the distribu-

tion of cases is not normal either with respect to extent of lesion or learning scores, and since the slight increase in the magnitude of the measure of association given by the correlation ratio would be meaningless at present, we have not computed these constants.

Comparison of table 3 with tables 13 and 14 shows that for operated animals the intermaze correlations are significantly higher than the correlations between extent of lesion and the criteria of learning. Since the intermaze correlations for normal animals are very low, the intermaze correlations for operated animals must be ascribed to some effect of the lesion, and it appears that the effective agent in cerebral lesion is more accurately measured by maze performance than by measurement of the surface area of the lesion. This may be due either to the failure of our methods of measurement of the lesions to express all of the significant characteristics of the lesion or to the fact that the correlation coefficient is a better expression of the relationship in one case than in the other. The latter possibility has certainly played some part in the matter, for the intermaze relationship is rectilinear, whereas the cortex-learning relationship is not. However, since the distribution of lesions is not normal, no correlation method can be depended upon to give a certain picture of the relationship and there is no present method of finding the causes of the discrepancy.

#### *Continuity of the mass relationship*

As a further test of the validity of our conclusions based on correlations, and to determine whether the correlations represent a continuous relationship or are due to the destruction of some critical amount of tissue, we have divided the cases by class intervals of 10 per cent destruction and computed the average practice for learning required by the animals in each class interval. The results of this analysis are presented in table 15. The last two intervals (40 to 49, and 50 per cent) are based upon too few cases to have significance. In the remaining four intervals there are only six inversions

of order in the fifteen series of constants. This is conclusive evidence of a continuous relationship between the extent of injury and the degree of retardation.

Previous data (Lashley, '29; Maier, '32) have suggested that there may be a limit of size below which lesions are relatively ineffective and above which there is marked defect. In these more adequate data there is no indication of a consistent, marked flexion point between any two of the class

TABLE 15  
*Average practice for learning required by animals with various amounts of cerebral destruction grouped in class intervals of 10 per cent*

MAZE		NORMAL	0-9	10-19	20-29	30-39	40-49	50+
Errors	I	22.0	32.9	112.1	260.2	360.6	1330.0	1356.0
minus	II	66.7	89.8	201.3	361.9	1118.5	2015.0	9437.0
errors	III	85.7	114.7	448.5	705.6	3460.0	2075.2	1863.0
first trial	IV	121.5	203.3	1155.3	1119.4	1467.0	3323.6	.....
	V	33.2	52.6	142.6	292.9	449.0	963.8	950.0
	I	24.3	28.9	60.1	114.2	103.6	150.0	150.0
Total	II	33.8	36.0	97.3	99.0	129.8	150.0	119.0 <sup>1</sup>
trials	III	42.1	49.7	93.0	124.9	150.0	150.0	150.0
	IV	61.2	58.0	87.6	110.4	142.3	150.0	.....
	V	26.8	34.2	48.4	64.7	84.1	90.6	97.3
Time	I	1227	593	2205	4084	9550	14356	14147
minus	II	3953	9937	8186	16228	11118	56208	34722
time	III	2532	9284	5397	15844	27476	17415	9148
of first	IV	5014	7818	8476	16535	23282	35234	.....
trial	V	610	757	1161	2387	3789	7085	5076

<sup>1</sup> died after 119 trials.

intervals. It is very probable that where such a condition has appeared in earlier data, it has been due to chance variation in inadequate samples.

With the limited data heretofore available, it has not been possible to define the form of the relationship between extent of lesion and degree of retardation. Lashley's data ('26, '29) indicate that extensive lesions produce a disproportionately great effect. Thurstone ('33) has concluded from an analysis of the original data that the formula  $k = aC^b$ , where

$k$  is the efficiency in learning,  $a$  a constant, and  $C$  the amount of cortex intact, best expresses the form of the relation.

With the 125 cases on maze V we have a more reliable basis for analyzing this relationship. Figure 2 shows the data for extent of lesions, grouped by class intervals of 5 per cent

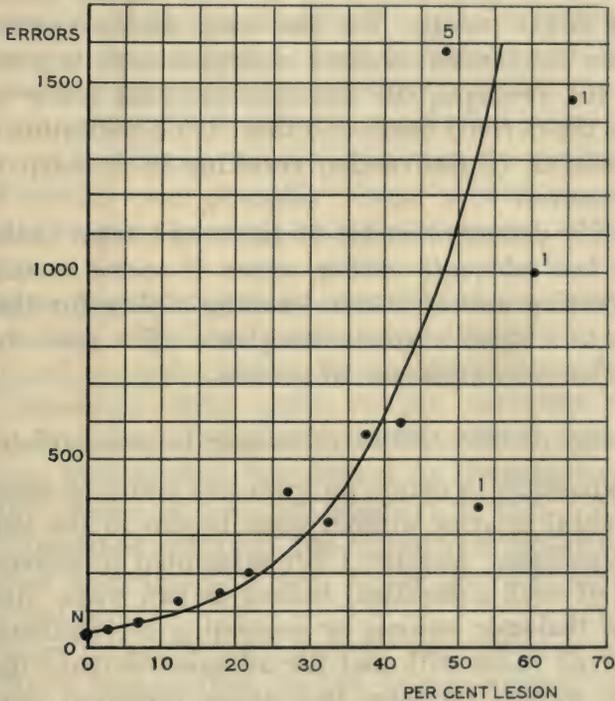


Fig. 2 The relation between extent of lesion and errors made in learning in maze V (8 culs de sac). The data on 60 normal and 127 operated animals are averaged by class intervals of 5 per cent destruction. The smooth curve is the best fitting one of logarithmic form. The numerals in the figure indicate the number of cases on which the more unreliable points are based.

destruction and plotted against average errors for each class. The first point in the curve, zero destruction, is the average of 60 normal animals. The other points are determined by smaller numbers of cases. The continuous curve has been derived from the data by the method of least squares and is the best fitting logarithmic form. It is given by the equation

$E = (38.39)_e^{(0.0698)L}$ , in which  $E$  is the error score,  $L$  the percentage lesion, and  $e$  the Naperian base.

Where there is an adequate number of cases, the data conform quite closely to the regular curve and suggest that the retardation from cortical destruction follows some definite law by which learning ability for the maze rapidly approaches zero with larger lesions. For the lower amounts of destruction, where the number of cases is large enough to give reliability to the averages, the experimental data conform quite closely to the derived curve and there is no indication of any critical amount of destruction resulting in a sharp rise in the error scores.

From this extensive series of cases and from indications given by less adequate earlier series, it seems certain that with increasing size of lesion, learning ability for the maze decreases at a steadily accelerating rate. The exact form of the curve has no significance at present.

*The influence of subcortical lesions upon the mass relationship*

It is impossible to obtain an extensive series of cases with large cerebral injuries without some lesions in the thalamus and archipallium. Lashley ('29) attempted to estimate the influence of such subcortical lesions in two ways: first, for control of thalamic lesions, by computing correlations separately for all cases with and for all cases without thalamic lesions, as well as for these two classes combined; secondly, by assigning an arbitrary value to each type of subcortical lesion and correcting the ranking for correlation obtained from cortical lesions by these arbitrary values. These analyses gave the following results. Correlations between errors and cortical destruction:

For all cases,	$p = 0.86$
For cases without thalamic lesion,	$p = 0.83$
For cases with thalamic lesion,	$p = 0.86$

With arbitrary correction for all types of subcortical lesions the correlations were the following between total destruction and the learning constants:

	<i>Uncorrected</i>	<i>Corrected</i>
Time,	0.62	0.67
Errors,	0.86	0.87
Trials,	0.77	0.80

The results of this analysis indicated that the inclusion in the series of animals with lesions in the archipallium or thalamus did not significantly alter the results from those which would have been obtained had only cases with lesions in the neocortex been included in all computation of constants. Correction for subcortical injury slightly raised the correlations with extent of destruction and indicated that "if lesions to internal structures could be accurately evaluated, the method would most probably reveal a still closer correspondence between learning ability and amount of functional tissue."

We have instituted similar controls for the present data. The possibly significant subcortical structures, septum, caudate and lenticular nuclei, fornix, hippocampal lobes, colliculi, habenulae, optic paths to the thalamus, anterior, median, and lateral thalamic nuclei, lateral and median geniculate bodies were represented on diagrams and three arbitrary grades of severity assigned to each. All brains were reexamined carefully for subcortical injuries and the amount of destruction in each of the above structures was graded and listed.

All cases in which there was no subcortical injury or at most slight degeneration in the dorsal convexities of the hippocampal lobes were selected. With these the correlations between extent of cortical destruction and errors in learning were computed. These constants are listed in table 16, in comparison with the constants computed from all cases. The elimination of the animals with significant subcortical lesions reduces the correlations very slightly, but since the cases with most extensive subcortical destruction have also the more severe cortical injuries, this procedure reduces the range of variation and the reduction in correlation is no more than would be expected from the reduction in range alone.

We have attempted to analyze the data on the basis of specific subcortical injuries, but have not been able to discover any significant relations. In our data it is usually possible to match any case with a subcortical injury with another having only a similar cortical destruction. It seems entirely a matter of chance as to which member of such pairs has the worse training record. We have not been able to find indications of specific effects upon maze learning of any lesions in the corpora striata or thalamus within our series of cases.<sup>6</sup> It seems quite certain that the correlation between extent of cortical injury and the degree of retardation is not due to the inclusion in the series of animals with subcortical lesions.

TABLE 16

*Comparison of the correlations between extent of lesion and errors in learning for all cases and for cases without significant subcortical lesions. Maze V*

GROUP	ALL CASES P	NO SUBCORTICAL LESIONS P
I	0.80 ± 0.05	0.76 ± 0.06
II	0.80 ± 0.05	0.72 ± 0.07
III	0.70 ± 0.07	0.69 ± 0.08
IV	0.60 ± 0.08	0.66 ± 0.08
Average	0.72	0.71

*The relative effects of symmetrical and asymmetrical lesions in producing retardation of learning*

In order to test the influence of corresponding areas of the two hemispheres, we have determined the areas common to the destruction in both hemispheres, as described on page 16, and from these have computed the correlation between extent of symmetrical destruction and errors made during training, using only the data on mazes I and IV, as a sample. The results of this analysis were given in table 17. The correlation for the symmetrical portions alone is the same as for the total extent of lesion. That for the asymmetrical

<sup>6</sup>The subcortical lesions are for the most part slight and unilateral. In no case is there an extensive bilateral injury to any thalamic center.

portions with error scores is less (0.49 and 0.35), but is as great as is to be expected, considering the reduction in range of lesion when computed on this basis alone. There is a still smaller correlation between the extent of symmetrical and asymmetrical lesion. This may be responsible for the apparent correlation between the latter and error scores. We have attempted to partial out its influence, with the results shown in the last column of the table. Not much weight can be ascribed to such statistical analyses, however, since the relationships are not rectilinear and the data have not normal distribution. The partial correlations (asymmetrical lesion with errors, with the influence of symmetrical lesions held constant) seem still significantly large and suggest that the asymmetrical portions of the lesions contribute to the deterioration as do the symmetrical portions.

TABLE 17

*Comparison of the effects of lesions common to the two hemispheres and of the asymmetrical portions of the lesions. The correlations are between extent of lesion and scores for errors less first trial in learning maze V*

GROUP	TOTAL LESION	PART COMMON TO BOTH HEMISPHERES	ASYMMETRICAL PART	SYMMETRICAL WITH ASYMMETRICAL	PARTIAL CORRELATION
I	0.74	0.79	0.49	0.39	0.32
IV	0.57	0.60	0.35	0.24	0.39

*The relative influence of lesions within different cytoarchitectural fields*

Lashley ('29) attempted to estimate the effectiveness of injuries within each of the chief functional fields by grouping his cases according to the field most seriously involved and computing constants for each group. The small number of animals which he had available made it necessary to include ambiguous cases, and inspection of his figures shows that there was an extensive overlap between the groups. This considerably reduces the validity of the evidence presented for equal effects of equal lesions in different areas.

We have sought a more conclusive test of the matter by selecting nearly unequivocal cases from our data. Since there

is a good bit of individual variation in the positions of the boundaries of the fields and probably even more distortion resultant from our methods of plotting the lesions and, finally, a good bit of uncertainty in determining the exact boundaries of the fields by direct histological methods,<sup>7</sup> there is no accurate means available for determining the amount of destruction in the different functional areas. As an approximate measure, a transparent diagram of Lashley's modification of Fortuyn's diagram of cytoarchitectural fields was superimposed upon the diagram of the lesions for each case, and the percentage of the total cortex destroyed within each field was measured with a planimeter.

On the basis of these percentage measurements, we selected cases in which the extent of lesion in one field was three or more times as great as in any other field and in which the total extent of lesion in any except the primary field did not exceed 5 per cent of the total cortex. The cases assigned to the four principal areas were the following:

*ff'n* (motor): 17, 18, 32, 41, 64, 65, 70, 75, 76, 80, 111.

*j* (somesthetic): 2, 4, 7, 9, 20, 34, 47, 73, 74, 78, 85, 94, 102, 106.

*p* (auditory): 1, 3, 6, 11, 19, 39, 40, 52, 79, 98, 100, 103, 105.

*w* (visual): 10, 14, 35, 36, 44, 66, 69, 71, 72, 97, 99, 104, 113.

For each of these groups the average extent of lesion and the average of errors (less first trial) in learning maze V were computed. These constants are given in table 18 and graphically in figure 3. The average destruction for the four groups is so nearly the same that the differences may be disregarded. In comparison with the average for normal animals in maze V ( $33.1 \pm 4.2$  errors) all of the groups were markedly retarded. For the motor, visual, and somesthetic fields the training records are essentially equal, the differences between the

<sup>7</sup> A survey of the literature on the cytoarchitecture of the brains of rodents by the senior author reveals that the disagreements among investigators in this field are so great as to cast doubt upon the significance of most of the areas differentiated. No two investigators have used the same criteria for distinguishing the areas and such criteria as have been defined are purely relative. The confusion is particularly striking within the areas called auditory and somesthetic.

groups being less than their probable errors, whereas all make approximately five times as many errors as do normals. The record for cases with lesions in field *p* is not consistent with the others. The score of 92.3 errors is markedly less than that of the next lowest group, but is also significantly

TABLE 18

*Analysis of effects of lesions largely confined to single cytoarchitectural fields*

FIELD	NUMBER OF CASES	AVERAGE LESION	$\sigma$	AVERAGE ERRORS	$\sigma$	STANDARD COEFFICIENT OF VARIATION
ff'n (motor)	11	15.9 $\pm$ 1.7	8.5	154.2 $\pm$ 40.5	199.5	1.29
j (somesthetic)	14	15.4 $\pm$ 1.3	7.4	173.1 $\pm$ 32.3	185.4	1.07
p (auditory)	13	16.0 $\pm$ 1.5	7.8	92.3 $\pm$ 21.1	112.7	1.21
w (visual)	13	15.9 $\pm$ 0.9	5.0	156.9 $\pm$ 41.5	222.3	1.42
Normals	60	0		33.1 $\pm$ 4.2	48.8	1.45

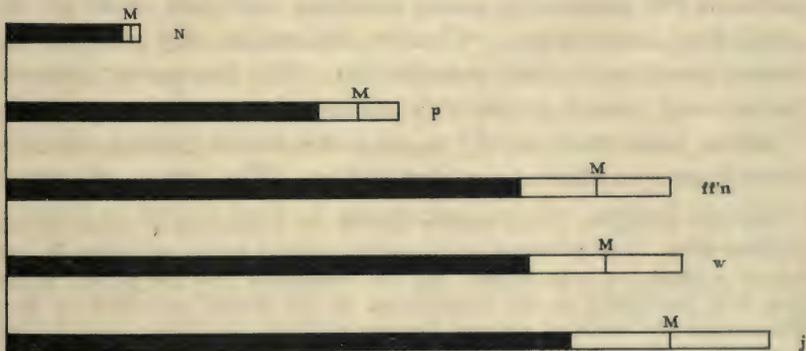


Fig. 3 A comparison of the effects of equal amounts of destruction in different cortical fields upon maze learning of maze V. The lines represent the relative magnitudes of the mean scores (*M*) with their probable errors for normal animals and for four groups of operated cases trained in maze V. *N*, normal; *p*, auditory; *w*, visual; *ff'n*, motor; *j*, somesthetic.

higher than the score of normals. The reliability of the differences in both cases is low (*ff'n* — *p* = 80.8  $\pm$  36.6; *p* — normal = 59.2  $\pm$  21.5). The indications from this analysis of the data are that equal lesions within the motor, somesthetic, and visual areas produce approximately equal effects upon the capacity to learn the maze and that lesions within the

auditory area are relatively less effective, although they also produce a significant deterioration of the function.<sup>8</sup>

We have computed the correlation between extent of lesion and error scores for each of the above four groups with lesions largely restricted to single architectural fields. The constants obtained were the following: Motor,  $p = 0.66 \pm 0.12$ ; somesthetic,  $p = 0.58 \pm 0.12$ ; auditory,  $p = 0.07 \pm 0.19$ ; visual,  $p = 0.36 \pm 0.17$ .

This selection of cases greatly reduces the range of variation and consequently affects the magnitude of the correlations. The mass relation seems to hold within the motor, somesthetic, and visual areas. As in the comparison of averages, the auditory area does not conform to the trend of the others.

For the question of the exact equivalence of the various parts of the cortex for maze learning our data must be regarded as inconclusive. Clearly, lesions in any part of the cortex produce marked retardation. For the motor, somesthetic, and visual areas this retardation is approximately equal, at least more nearly so than the demonstrated retardations from peripheral sensory defect. The lesser effects of

<sup>8</sup> We have attempted to use another method for comparison of lesions in the different fields. The percentage of the cortex included within the lesion in each field was listed for each animal. On the assumption that the injury within each field contributes to the deterioration of the animal according to some determinant ( $D$ ) which is constant for that field, the total error score may be expressed as the sum of the products of the percentage destruction within each field by the determinant for that field. This gives, for example, for animals nos. 16 and 17 in maze I the equations—

$$13.6 D_{r/n} + 7.1 D_j = 57$$

$$10.9 D_{r/n} + 7.8 D_j = 33.$$

We thus obtained 127 equations for maze V. These were solved by the method of Doolittle to give an average value for  $D$  for each of the four chief fields. The results were  $D_{r/n} = 7$ ;  $D_p = 8$ ;  $D_j = 18$ ;  $D_w = 27$ . Using these values, the expected errors were computed for each animal and plotted against the experimental errors. This comparison gave theoretical values consistently too high for the lesser lesions and too low for the greater, showing that what had seemed a promising method is inapplicable to our data, because of the non-linear relation between extent of lesion and performance. We are indebted to Prof. L. L. Thurstone for the test of this method.

injuries in the auditory area are probably significant, but even with so extensive a series of cases as are included in our study, the statistical reliability of our data is low.

#### INFLUENCE OF THE COMPLEXITY OF THE PROBLEM

To test the question whether an increasing number of culs de sac in the maze offers a progressively greater difficulty for animals with cerebral lesions than for normals, we have made a comparative analysis of the learning records of normal and operated animals trained on mazes I, II, III, and IV. The individual records of the normal animals have been presented in tables 4 to 11.

#### *Equation of groups*

Since, as was pointed out in our discussion of methods, it is necessary to use a separate group of animals with each of the mazes in the tests of complexity, some method of equating the groups is desirable. Members of the groups were taken at random from the stock colony and were presumably a random sample of the colony. An attempt was made to obtain four identical series of animals with destructions of from 5 to 60 per cent of the cortex, by the method described on page 15. Comparative data on the extent of lesion have been presented in table 2. Group I shows the smallest average percentage destruction, but also the greatest range. The averages for the other three groups are practically identical, with a somewhat smaller range in group IV than in the others. In the loci of the lesions the groups seem sufficiently alike to permit of direct comparison.

As a test of the equality of the groups, all were trained on maze V after completion of training on the comparison mazes. The average scores for the eight groups in maze V are presented in table 19. Measurement of the equality of the groups by the use of a second maze is complicated by the possibility of differential transfer from the different mazes used in the initial training. The table gives some indication that such a transfer has taken place. Converting all the constants of

the table into percentages of the scores of group I and averaging these, we obtain the following figures: group I, 100; group II, 89.9; group III, 72.9; group IV, 77.1 per cent. These figures indicate that the animals previously trained on the

TABLE 19

*Constants for normal and operated animals on maze V: to test the equality of the groups previously trained on mazes I to IV*

GROUP	ERRORS LESS FIRST RUN	$\sigma$	TRIALS	$\sigma$	TRIALS LES CORRECT RUN	$\sigma$	TIME LESS FIRST TRIAL (SECONDS)	$\sigma$
Normals								
I	44.4 $\pm$ 6.3	36.4	28.7 $\pm$ 1.9	10.8	13.9 $\pm$ 1.3	7.4	1037 $\pm$ 254	1425
II	26.4 $\pm$ 3.7	21.5	25.0 $\pm$ 1.5	8.5	11.1 $\pm$ 0.8	4.4	709 $\pm$ 197	1098
III	22.9 $\pm$ 3.5	20.3	29.0 $\pm$ 3.9	22.4	11.7 $\pm$ 1.4	8.0	310 $\pm$ 39	222
IV	38.8 $\pm$ 14.6	83.9	24.8 $\pm$ 3.0	17.2	12.1 $\pm$ 1.8	10.4	387 $\pm$ 66	371
Operated								
I	333.9 $\pm$ 47.7	387.7	68.2 $\pm$ 4.0	32.0	50.9 $\pm$ 4.0	32.6	3263 $\pm$ 609	4948
II	451.4 $\pm$ 95.3	786.6	65.3 $\pm$ 3.8	31.7	48.7 $\pm$ 3.9	32.4	3177 $\pm$ 680	5610
III	281.6 $\pm$ 36.7	297.8	60.5 $\pm$ 4.2	34.4	44.7 $\pm$ 4.2	33.2	1857 $\pm$ 244	1983
IV	265.5 $\pm$ 38.4	331.6	56.8 $\pm$ 4.2	36.3	44.4 $\pm$ 4.3	36.8	2244 $\pm$ 447	3865

TABLE 20

*Reliability of the differences for error scores among the normal and among the operated groups in maze V*

	DIFFERENCE	D/P.E. <sub>D</sub>
Normal groups		
I- II	18.0 $\pm$ 7.3	2.4
I-III	21.5 $\pm$ 7.2	2.9
I- IV	5.6 $\pm$ 16.3	0.3
Operated groups		
II- I	117.5 $\pm$ 106.0	1.1
II-III	169.8 $\pm$ 60.0	2.8
II- IV	185.9 $\pm$ 61.0	3.0

mazes with few culs de sac learned maze V less rapidly than did those previously trained on mazes III and IV. The reliability of the differences between the constants in table 19 is not great. Table 20 gives the ratios of the greatest differences between the error scores to their probable errors. Al-

lowing for the differential transfer indicated above, these differences will be still further reduced. Groups III and IV tend to be somewhat better than groups I and II in both the normal and operated series, so the inequalities may be expected to influence the results for both series in the same direction. Although absolutely large, the differences with maze V are relatively small in comparison with those obtaining between the records of the groups in the comparison

TABLE 21

*Average learning records of normal animals on the four mazes used for test of the influence of complexity*

MAZE	ERRORS LESS FIRST TRIAL	$\sigma$	TRIALS	$\sigma$	TRIALS LESS CORRECT RUNS	$\sigma$	TIME LESS FIRST TRIAL	$\sigma$
I	22.0 $\pm$ 2.4	13.8	24.3 $\pm$ 2.9	16.7	11.4 $\pm$ 1.0	5.8	1227 $\pm$ 214	1331
II	66.7 $\pm$ 7.5	43.2	33.8 $\pm$ 1.9	10.9	21.0 $\pm$ 1.2	7.1	3953 $\pm$ 691	4452
III	85.7 $\pm$ 7.2	41.4	42.1 $\pm$ 3.5	20.4	21.9 $\pm$ 1.5	8.4	2532 $\pm$ 449	2081
IV	121.5 $\pm$ 17.6	100.9	61.3 $\pm$ 5.2	29.8	29.2 $\pm$ 1.9	10.9	5014 $\pm$ 874	5266

TABLE 22

*Average learning records of operated animals on the four mazes used for test of the influence of complexity*

MAZE	ERRORS LESS FIRST TRIAL	$\sigma$	TRIALS	$\sigma$	TRIALS LESS CORRECT RUNS	$\sigma$	TIME LESS FIRST TRIAL	$\sigma$
I	246.7 $\pm$ 42.5	345.4	77.7 $\pm$ 6.2	50.2	55.6 $\pm$ 6.1	49.3	4257 $\pm$ 784	6074
II	921.1 $\pm$ 203.5	1706.6	103.8 $\pm$ 5.6	46.7	79.4 $\pm$ 5.9	48.9	16738 $\pm$ 2801	23489
III	1039.1 $\pm$ 169.2	1396.9	114.6 $\pm$ 5.2	42.7	91.7 $\pm$ 5.6	46.3	12419 $\pm$ 1488	12283
IV	1382.9 $\pm$ 588.8	5089.9	109.0 $\pm$ 5.1	44.4	88.4 $\pm$ 5.7	49.1	17403 $\pm$ 3345	28919

mazes. They seem unlikely to have produced a constant error in relation to the complexity of the mazes. They do restrict us to a consideration only of large differences as evidence of any genuine influence of the complexity of the problems upon rate of learning.

*Comparison of normal and operated animals in learning mazes of different complexities*

Tables 21 and 22 summarize the learning scores for normal and operated animals in mazes I to IV. For error scores there is a regular progression in practice required for learn-

ing from the simplest to the most complex. The scores by other criteria are less consistent, but show in general the same trend. The averages of all scores, expressed as percentages of the scores on maze I are given at the right.

The ratios of practice required for learning by all criteria for mazes II, III, and IV on maze I are given in table 23. The objective complexities of the mazes, expressed in numbers of culs de sac are in the proportions of 1:2:3:4. The relative difficulty for normal animals, in terms of the most consistent criterion, error scores, is as 1:3:4:5.5. The ratio of difficulty for the operated cases (1:3.7:4.2:5.6) is not significantly different from that of the normal animals. The indica-

TABLE 23

*Comparison of scores in learning tests for normal and operated animals in the four comparison mazes, expressed as ratios on scores in maze I. N = normal group, Op. = operated group, Op.<40% = cases with lesions of less than 40 per cent, included as a control of the effects of exclusion of cases which failed to get through the maze on the first trial*

MAZE	RATIO OF CULS DE SAC	ERRORS			TRIALS		TIME		AVERAGE	
		N.	Op.	Op.<40%	N.	Op.	N.	Op.	N.	Op.
I	1	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
II	2	3.03	3.70	2.97	1.39	1.33	3.24	3.92	2.55	2.98
III	3	3.90	4.20	4.59	1.73	1.47	2.11	2.92	2.58	3.81
IV	4	5.50	5.60	6.23	2.52	1.40	4.12	4.07	4.06	4.19

tions from time and trials are essentially the same. There is no evidence that the longer mazes are disproportionately more difficult for the operated animals than for normals.

The indication in table 19 that operated groups III and IV are superior to the others, even allowing for differential transfer, suggests that the selection of cases brought about by failure of some cases to get through a single trial may have favored those groups. We have therefore computed the average errors for the operated animals exclusive of cases having more than 40 per cent destruction, thus eliminating the most badly deteriorated cases from groups I and II. The ratios of these averages on the scores for maze I for the four groups were the following:

	<i>Maze I</i>	<i>Maze II</i>	<i>Maze III</i>	<i>Maze IV</i>
Ratio,	1.00	2.97	4.59	6.23

With this correction the operated animals do somewhat worse proportionately on the longer mazes than do normals, but the differences are still too small to be regarded as significant.

As a further test we have computed the ratios for errors for groups taken by increments of 10 per cent lesion as given in table 15. These ratios are given in table 24. The figures are more variable, owing to the smaller number of cases, but there is no indication that any extent of lesion produces disproportionately poorer records in the more complex mazes.

TABLE 24

*The ratio of errors made during training for mazes II, III, and IV to maze I, for animals classed according to extent of lesion*

MAZE	I	II	III	IV
0	1.00	3.03	3.90	5.50
1-10	1.00	2.72	3.46	6.10
10-20	1.00	1.79	4.00	10.30
20-30	1.00	1.39	2.72	4.31
30-40	1.00	3.12	9.63	4.07
40-50	1.00	1.52	1.56	2.50

As by other methods of treating the data, there is no evidence of any influence of cerebral lesion upon the proportionate difficulty of the different mazes.

## DISCUSSION

Our primary object in these experiments was to test the influence of various amounts of cerebral destruction upon the capacity to form habits involving different numbers of similar tasks. We have failed to confirm Lashley's finding ('29) that increasing the number of culs de sac disproportionately increases the difficulty of the mazes for animals with brain lesions. His experiments differed from the present ones in the following respects:

1) All animals were trained in all mazes, thus permitting of transfer or interference effects. 2) His animals were first

trained on the most complex maze, thus including in the records of this maze any greater difficulty which the operated animals may have had in adapting to the general training situation. 3) His mazes differed markedly in the relations of the culs de sac to the true path and so presented qualitatively different situations. 4) His cases included a greater proportion of animals with extensive lesions and a few cases with lesions greater than our present maximum. 5) Our mazes all present the same general plan of simple right-left alternation and so admit the possibility of learning by a simple generalization.

The first of these differences in the experimental situations does not seem likely to have produced the differences in results, unless we assume that operated animals differ in capacity for transfer of training from normals. Such an assumption has been made by Melton ('31) concerning retroactive inhibition, but the evidence available indicates that perseveration and consequently interference of habits is most likely in the operated animals, and this would have tended to make the simplest of Lashley's mazes (second in the series) relatively more difficult for the operated animals.

The common plan of our mazes might permit of learning by simple generalization. But to account for our results on this basis, we should have to assume that the capacity to generalize is less affected by extensive lesions than the capacity to form associations between unrelated elements—an assumption not in accord with Maier's experimental results ('31, '32).

The available data are not adequate to decide among the remaining three possibilities. A large part of the inferiority of the operated animals on Lashley's most complex maze was evidently due to the inclusion of cases with lesions greater than 50 per cent. We have few comparable cases and it may be that the disproportionate retardation holds only for the most severely deteriorated animals. However, the influence of qualitative differences in the tasks cannot be disregarded.

The evidence from studies of the normal growth of intelligence in man and from clinical studies of dementia goes far toward proving that differences in capacity are in some measure qualitative. Tasks where a mere reduplication of elements is involved, as in span of attention or memorizing of nonsense syllables, do not reveal differences in intelligence brought out by batteries of qualitatively different tasks. Clinical studies, such as those of Head ('26) on semantic aphasia (interpreted as dementia by Henschen, '27), indicate that limitations to performance are set by qualitative characters of the tasks. We should therefore expect to find that a battery of qualitatively different tasks, graded in difficulty and making up an apparently continuous series for normal individuals, would show disproportionate difficulty at the higher levels for demented individuals in accord with the severity of the dementia. Comparison of the results with the qualitatively different tasks used by Cameron, Lashley, and Maier suggests that this is actually the case for animals with cerebral lesions. Extensive experiments will be necessary, however, to establish the point and to reveal the nature of the effective qualitative differences, if they exist.

For tasks where difficulty is determined chiefly by the reduplication of similar elements our data seem conclusive. The difficulty of such tasks for animals with cerebral lesions increases at an accelerating rate with the extent of lesion, but the relative difficulty of the several tasks remains the same.

*Difficulties of interpretation arising from individual variations*

One of the most difficult problems in the study of cerebral functions is that of accounting for the widely divergent symptoms following similar lesions in different individuals. In our present series, as in other similar studies, we find a number of animals with extensive lesions and yet with training records which are far better than the averages of other animals with equal amounts of destruction, and which may approach the records of normal animals. Such cases are nos.

15, 16, 17, 19, 22, 48, 53, 73, 74, 82, 90, 112, 117, and 120. Many of these show markedly asymmetrical lesions in the two hemispheres, but it is possible to match almost every case with another from our series having practically identical lesions and a poor training record. Neither locus nor depth of lesion nor injuries to subcortical structures provides any apparent basis for the differences in scores. The more obvious explanations possible for the differences are:

1. Anatomical variation. Studies of variation in locus of cytoarchitectural areas by the senior author now in progress do not reveal such individual differences as would be required to account for the data on behavior.

2. Chance success in solving of the problems. Maze-learning scores are certainly influenced to a large extent by chance factors which would seriously influence the number of errors or trials in the final score, but the very high intermaze correlations and the fact that many of the above animals showed superior ability in two different mazes means that, if chance determined the low scores, it was a chance discovery of some general principle of maze running, and this, although possible, is difficult to fit into our present conceptions of maze learning.

3. Different animals employ differently localized cerebral mechanisms in learning the maze. One animal might be primarily dependent upon visual, another upon kinesthetic cues and the like, and a lesion in the striate area might in consequence markedly affect the former and leave the latter unaffected. Such an hypothesis is contradicted by the relatively slight effect of sensory privation on maze learning in comparison with the effects of lesions in cortical sensory fields (Lashley, '31 a). An alternative would be the assumption that the different sensory fields contribute differently to maze learning in different animals in other ways than by direct mediation of peripheral impulses. This hypothesis approaches the doctrine of image types and studies of the latter have given no conclusive evidence that the image type in any way correlates with the mode of learning. It is doubtful

that even the most extreme visual type, the eidetic, employs visual mechanisms in routine learning to a greater extent than do noneidetics. The assumption that differently localized functions predominate in maze learning by different animals, although possible, is not supported by any direct evidence.

The difficulty of the problem is increased by the clinical evidence, especially in the field of aphasia. The conflicting evidence on localization bespeaks a condition in man like that which we find in our series of animals. Except for the primary projection areas, negative cases have been reported for practically every cortical region. (Compare Monakow, '14, p. 768, for a summary of the situation on motor aphasia and Broca's area.) Negative cases in motor aphasia and similar non-sensory functions cannot be explained plausibly in terms of individual differences in the imagery used in speech.

A significant point for the problem, perhaps, comes from the repeated observation that the severity and duration of symptoms from brain lesions are less in young than in old and less in intelligent than in low-grade individuals. If true, this can only mean that the severity of symptoms is dependent not only upon the locus and extent of lesion, but also upon the general level of dynamic functioning of the organism.

In spite of the marked individual variation, the consistency of the results on various functions presented in table 1 and the uniform trend of the data summarized in figure 2 suggest that there must be some constant causal factor in consistency of maze performance dependent upon the mere quantity of cerebral tissue and not an artifact arising from the limitation of this or that special function.

#### SUMMARY

One hundred twenty-seven rats with cerebral lesions and 60 normal controls were trained in a maze of 8 culs de sac. The learning scores in this task have been analyzed with the following results:

1. The relation between extent of lesion and retardation in maze learning is curvilinear and the error scores appear to be a logarithmic function of the extent of lesion.

2. It has been impossible to detect any influence of small injuries in the archipallium or thalamus (when combined with extensive cortical destruction) upon the maze scores.

3. The portions of the lesions, asymmetrical with respect to the two hemispheres seem to contribute to the retardation, as well as do the symmetrical portions.

4. Lesions in all parts of the neocortex produce a marked deterioration. Our data are inconclusive with respect to the exact equipotentiality of the areas.

The cases were divided into four equal groups, each of which was trained on one of four mazes with 4, 8, 12, and 16 culs de sac. The normal and operated groups were compared with respect to the relative difficulty of the four mazes. The operated animals were markedly retarded in all mazes, but the relative difficulty of the simple and complex mazes was the same for them as for the normal controls. This conclusion applies to mazes in which difficulty is increased by duplicating identical elements. The problem of relative difficulty where qualitative differences are introduced remains unsettled.

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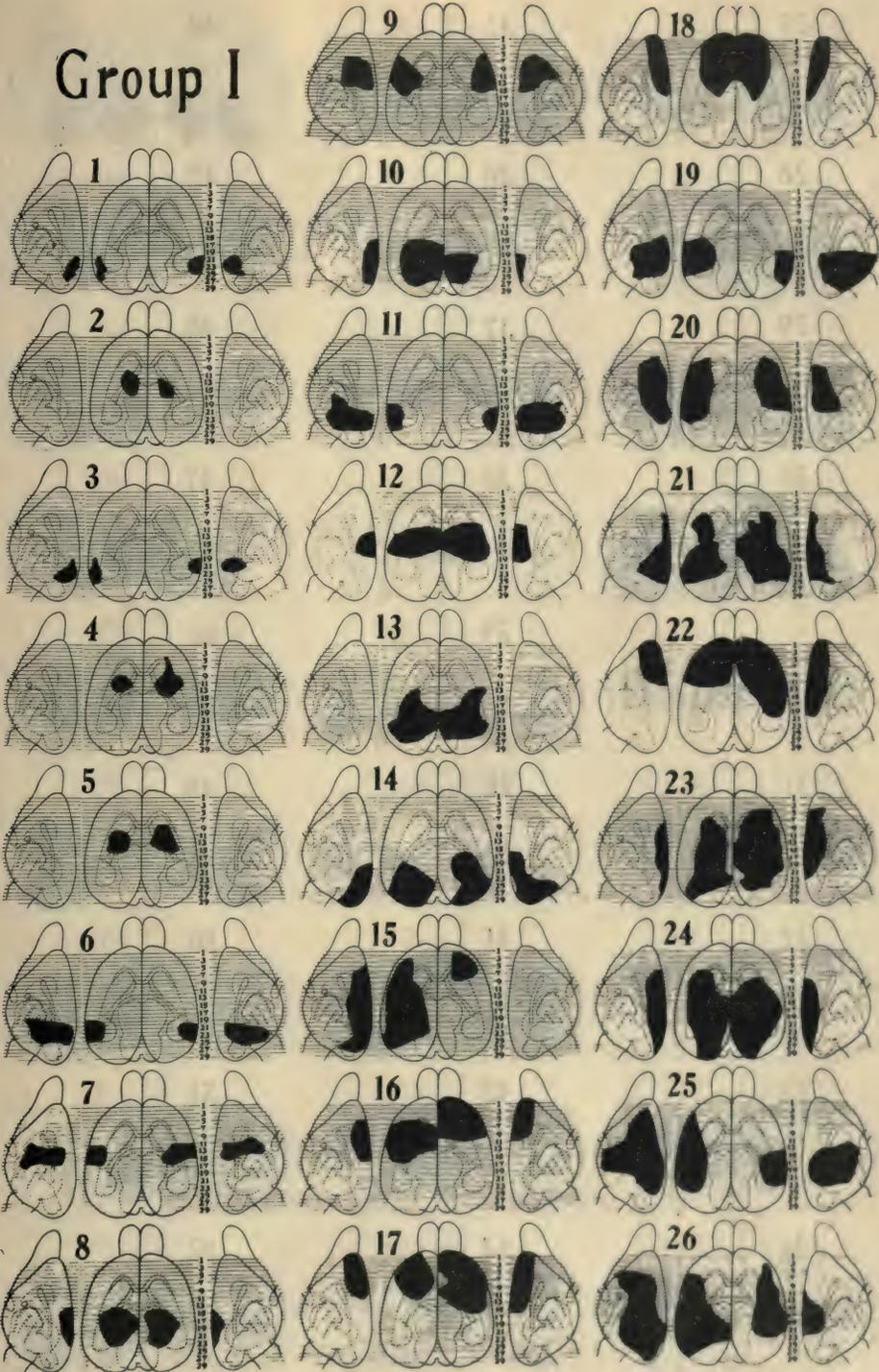
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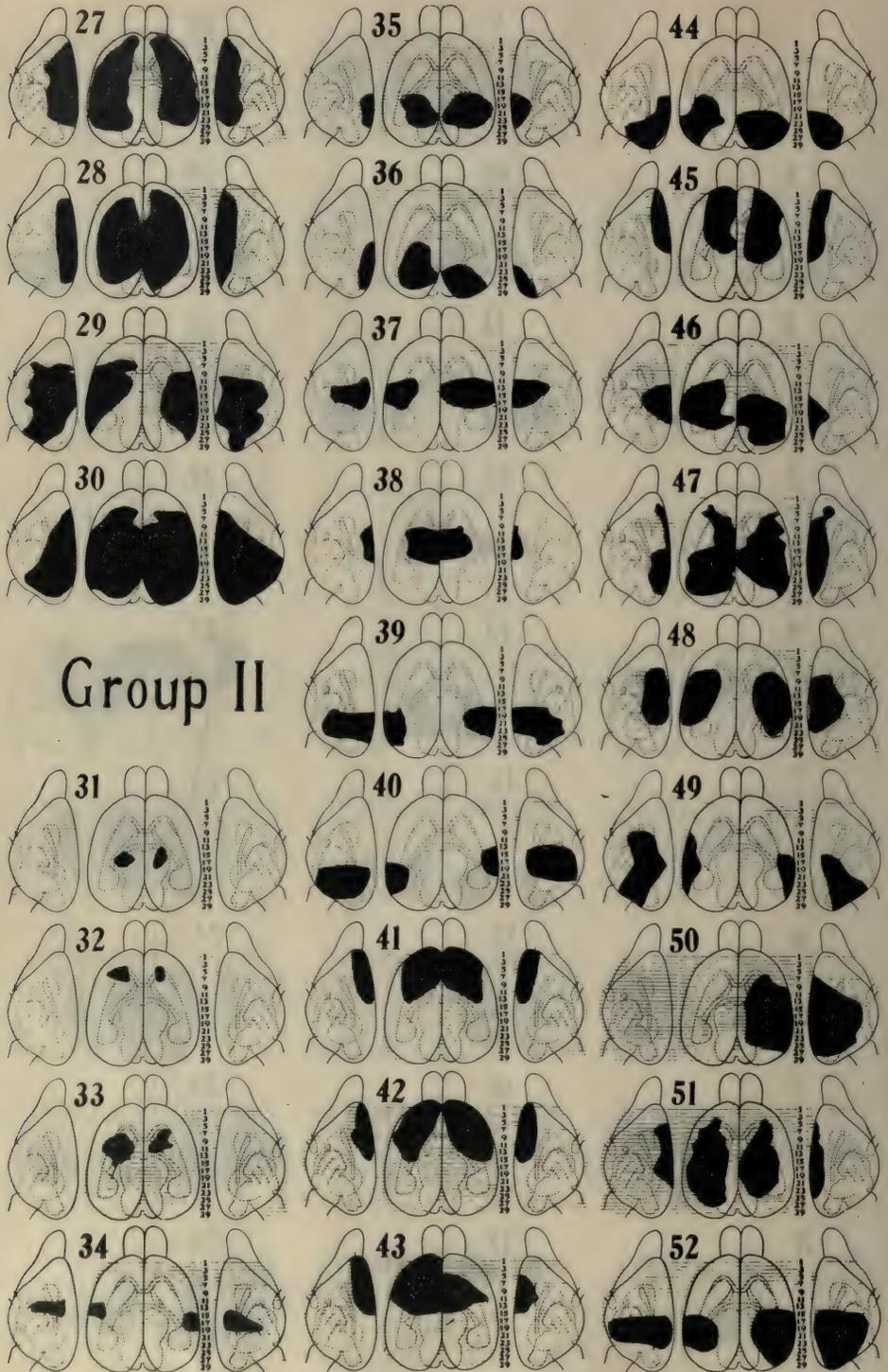
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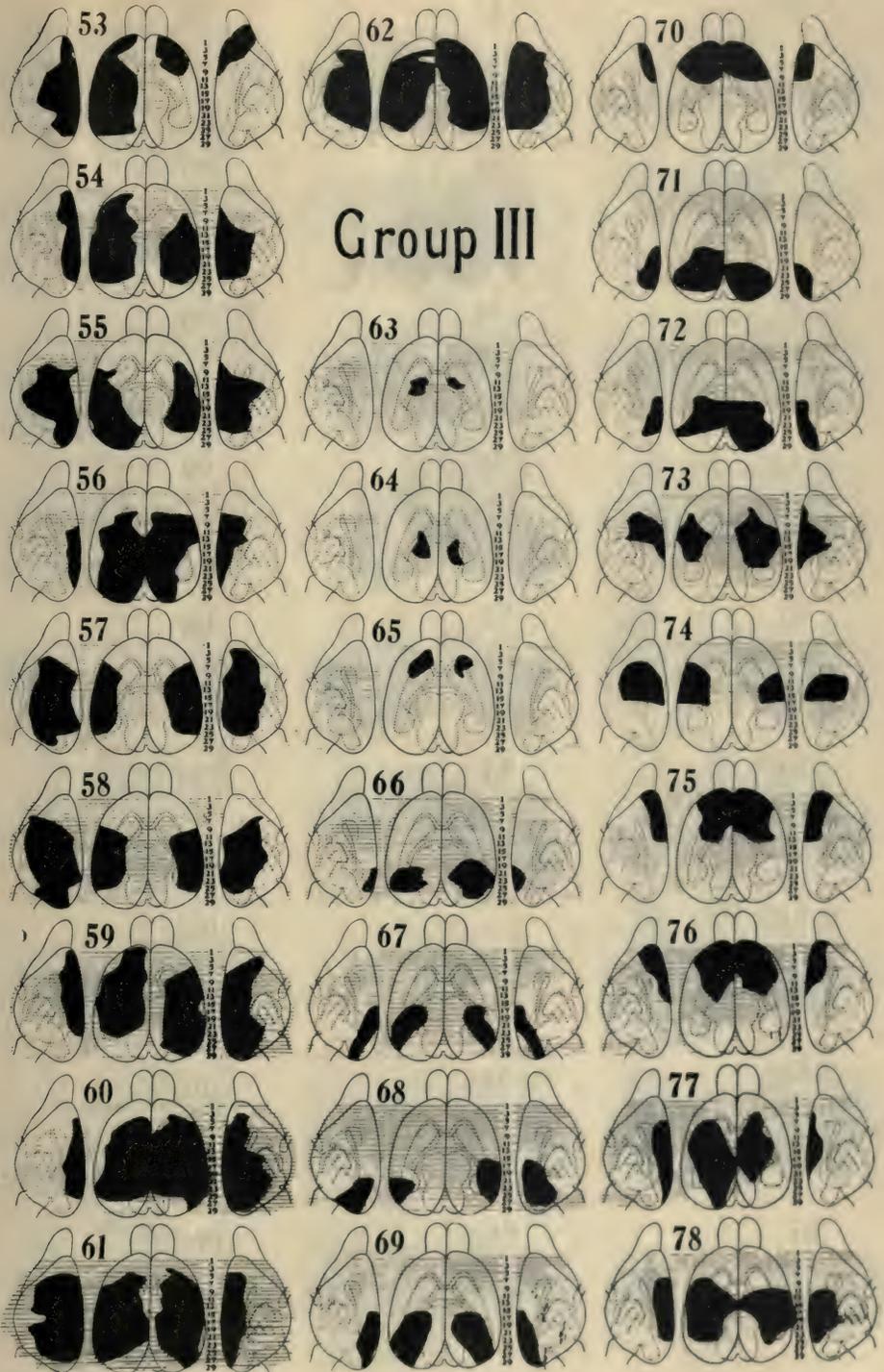
PLATES 1 TO 5

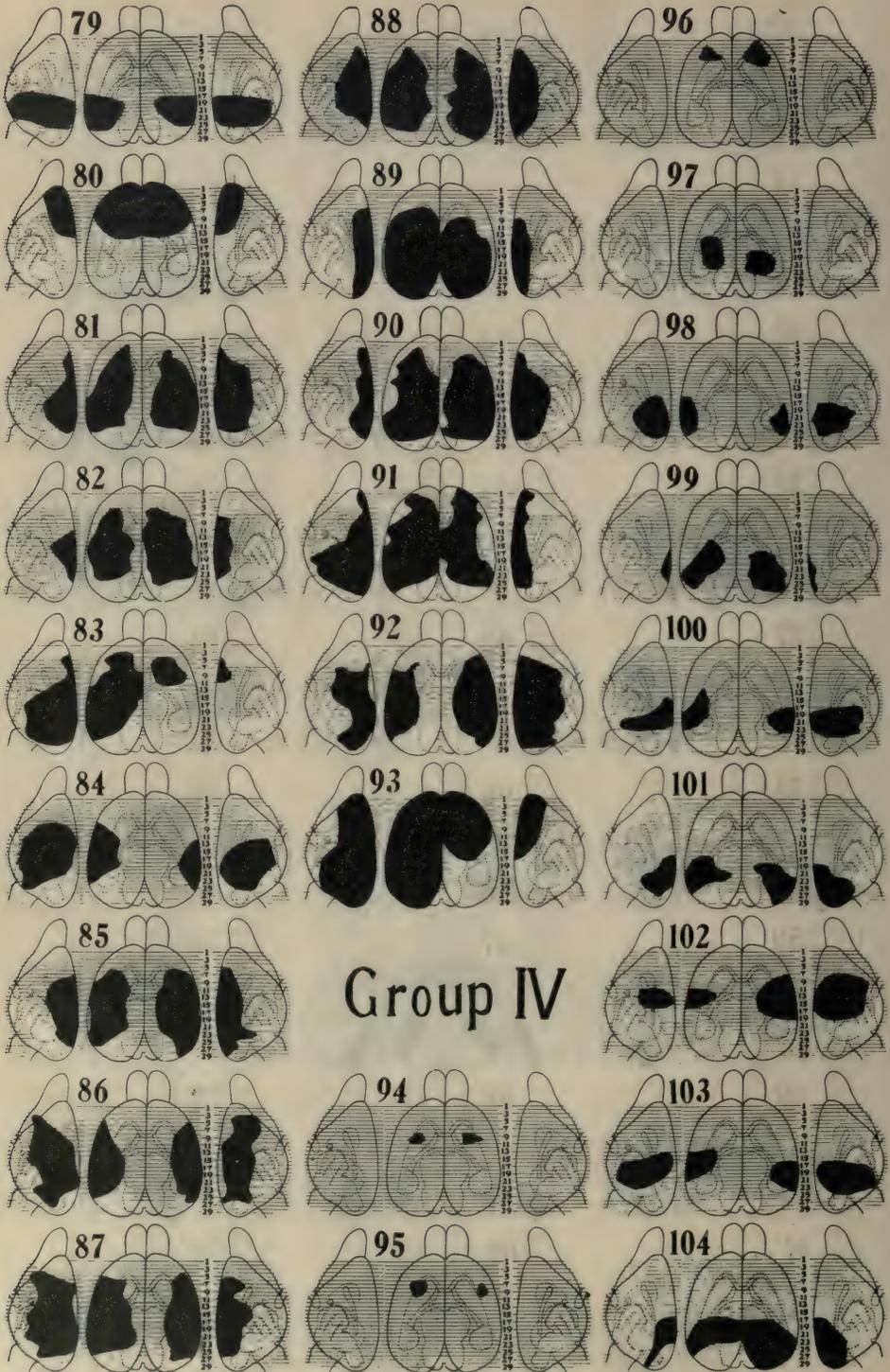
Diagrams of the extent and locus of lesion in each of the animals reported in the present study. The diagrams are numbered to correspond to the numbering of the records in tables 4 to 7. Groups I to IV were trained, respectively, in mazes I to IV, then all cases on maze V.

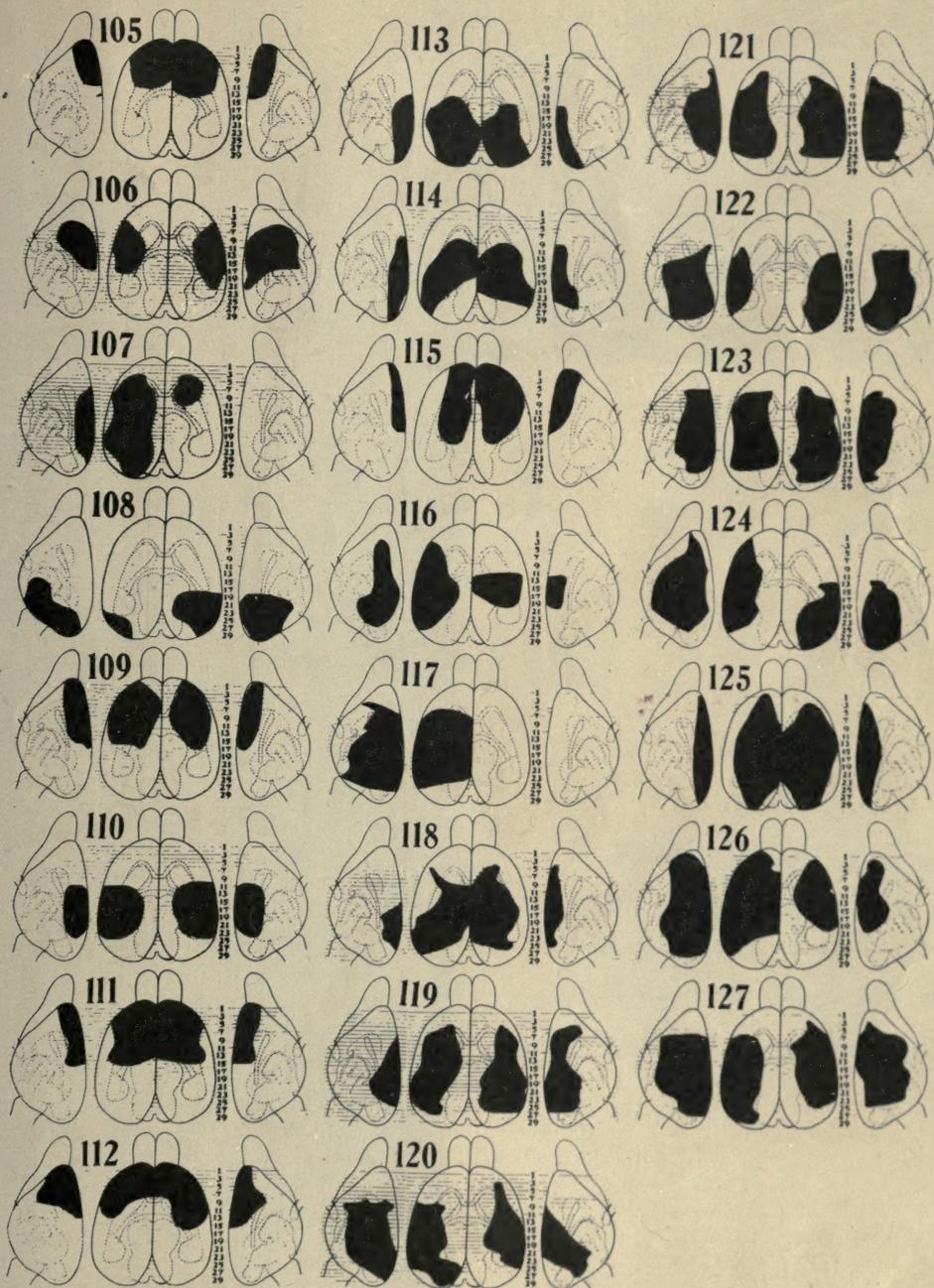
# Group I



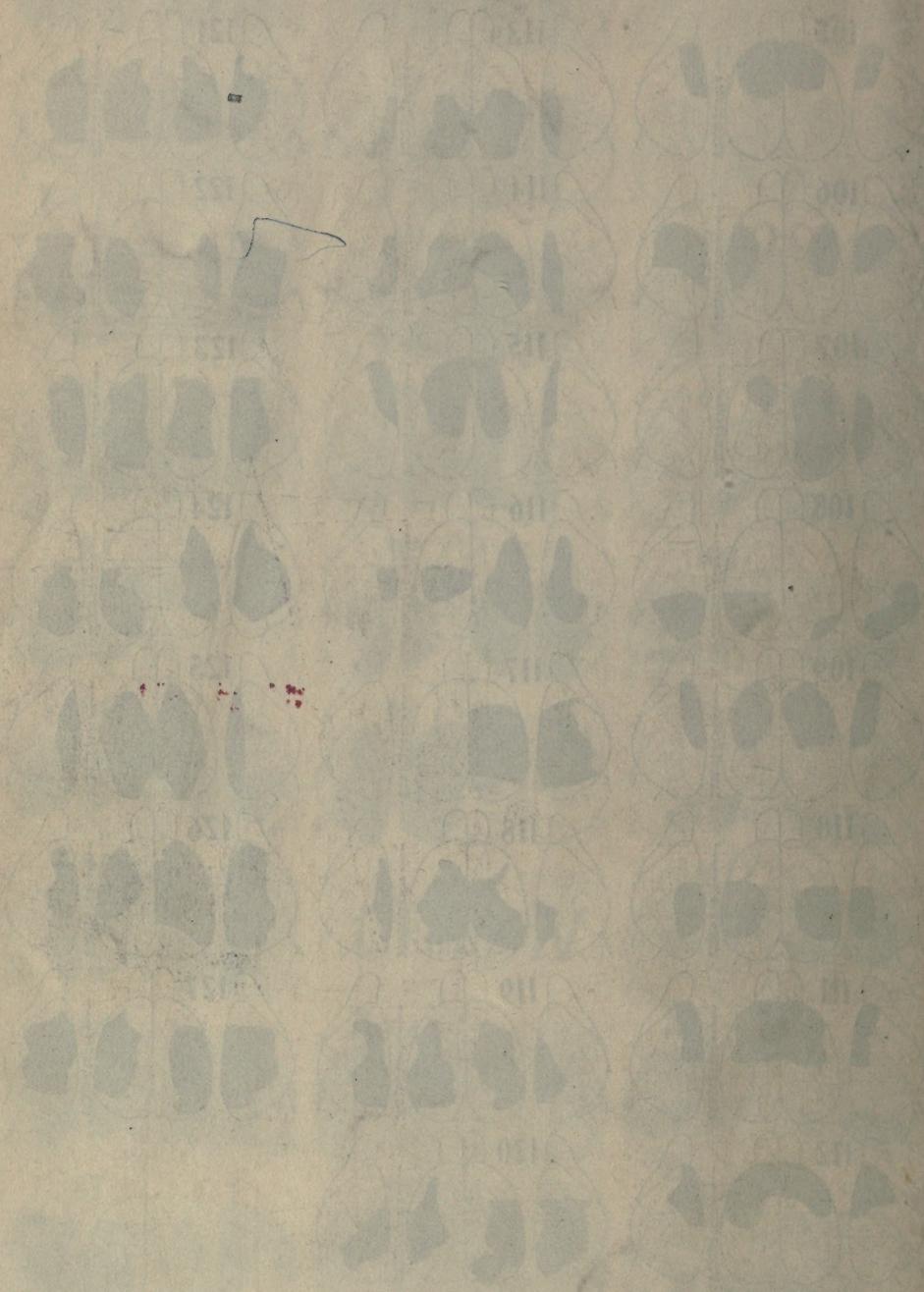








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